APPENDIX Q.2

AMBIENT AIR MODELLING

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MEMORANDUM

TO: April Gowing

REF. NO.: 3698

FROM: Tara Bailey/ca/25 TB

DATE: February 3, 2005

RE: Air Emissions and Dispersion Modeling During Excavation
Former Lagoon Site, Hamptonburgh, New York

1.0 INTRODUCTION

The following memo outlines the air emissions and dispersion modeling conducted for potential on-site, above ground exposure to emissions from contaminated soils and exposed groundwater at the Former Lagoon Site (Site) located in Hamptonburgh, New York.

The air emissions and dispersion modeling was conducted to estimate the maximum 8-hour average ground level concentrations on-Site during excavation activities. An excavation activity will result in the maximum ambient air exposure point concentrations to which construction workers may be exposed. The emissions modeled are potential volatile organic compounds (VOCs), and respirable particulate matter (PM) emissions of semi-volatile organic compounds (SVOCs), PCBs/Pesticides and inorganics released from excavated soil and exposed groundwater.

Air emissions and dispersion modeling was conducted for three scenarios:

a) the emissions from excavation activities of the Site soil (excluding Lagoon 6 soils);

b) the emissions from excavation activities of Lagoon 6 soils; and

c) the emissions from exposed groundwater during excavation activities.

The air emissions and dispersion modeling was based on the characteristic concentrations for each scenario which were chosen as the 95 percent upper confidence level (UCL) of the mean or maximum detected soil concentrations (whichever was lower). The concentrations used in the modeling for the Site soils (excluding Lagoon 6), the Lagoon 6 soils, and the Site groundwater are summarized in Tables 1A, 1B, and 1C, respectively.

2.0 AIR EMISSIONS MODELING

The modeling assessed the potential VOC, particulate matter, PCB/Pesticides and inorganic emissions, which could occur at the Site if excavation of contaminated materials was conducted.

2.1 VOC Emissions During Excavation

The principal source of atmospheric contaminants, if any, from the excavation of the Site soils, is expected to be the volatilization of VOCs from the soil. An appropriate mathematical emission model was selected for the potential VOC emissions during excavation activities from a review of available models. The model selected for use was the average emission rate detailed model as presented in "Air/Superfund National Technical Guidance Study Series Estimation of Air Impacts for the Excavation of Contaminated Soil", USEPA, March 1992. This model provides an estimate of the average emission rate of compounds from diffusion processes and exposure of soil pore spaces based on soil conditions (density, porosity, and moisture content), soil concentrations, an estimated excavation rate and the physical and chemical properties of the soil contaminants.

The basis for this model are the following equations:

$$ER = ER_{PS} + ER_{DIFF} \qquad \text{(Eq. 1)}$$

$$ER_{PS} = \frac{(P)\,(MW)\,(10^6)\,(Ea)\,(Q)\,(ExC)}{(R)\,(T)} \qquad \text{(Eq. 2)}$$

$$ER_{DIFF} = \frac{(C)\ (10{,}000)\ (SA)}{\left(\frac{Ea}{(Keq)\,Kg)}\right) + \left(\frac{\pi t}{(De)\,(Keq)}\right)^{1/2}} \qquad \text{(Eq. 3)}$$

where:

ER	=	total emission rate (g/sec);
ER_{PS}	=	pore space emission rate (g/sec);
ER_{DIFF}	=	diffusion emission rate (g/sec);
P	=	vapor pressure of compound (35 mm Hg);
MW	=	molecular weight (100 g/g-mol);
R	=	gas constant (62,361 mm Hg-cm^3/g-mol K);
T	=	temperature (298 degrees Kelvin);
Ea	=	air-filled porosity (0.44 dimensionless);
Sv	=	volume of soil moved (150 m^3);
Q	=	excavation rate (0.042 m^3/sec);
ExC	=	soil gas to atmosphere exchange constant (0.33 dimensionless);
C	=	concentration in soil (1.35E-04 g/cm^3);
SA	=	emitting surface area (290 m^2);
Keq	=	equilibrium coefficient (0.613 dimensionless);

Kg	=	gas-phase mass transfer coefficient (0.15 cm/sec);
t	=	time (60 sec); and
De	=	effective diffusivity in air (0.0269 cm^2/sec).

Note that the default values, in parentheses, suggested by the USEPA, are to be used when the Site-specific parameter is unknown.

The pore space emission rate equation (Eq. 2) is based on the assumption that the soil pore gas is saturated with the compound of interest. If this is not the case, equation 2 may over-predict the emission rate. Therefore, if the value of equation 2 exceeds one third the total mass of contaminant in a given volume of soil, the pore space emission rate should be calculated using:

$ER_{PS} = M^* (0.33)/tsv$ (Eq. 4)

where:

M	=	total mass of contaminants in a given volume, Sv, of soil (g); and
tsv	=	time to excavate a given volume, Sv, of soil (sec).

The following assumptions were used in the emissions modeling:

- a contaminated soil excavation rate of 0.042 m^3/sec for a period of 8 hours/day was chosen (this is equivalent to 1,200 m^3/day); and
- emitting surface area equal to 290 m^2.

It is noted that the pore space emission rate for a compound is constant during the excavation period while the diffusion emission rates decay exponentially with time, therefore, average diffusion rates have been calculated for the 8-hour period and are summarized in Tables 2A and 2B.

Using the 8-hour average diffusion rates, the time at which the average emissions occur was calculated to be at approximately 7,130 seconds (i.e., 2 hours). The 8-hour average pore space and diffusion emissions of VOCs during excavation were calculated. The total compound emissions from a source area during excavation, as summarized in Tables 3A and 3B, are equal to the sum of the pore space and diffusion emissions.

2.2 SVOC, PCB/Pesticides, Inorganics, and Particulate Emissions during Excavation

SVOCs, PCBs/pesticides and inorganics have only been considered to be present in particulate matter potentially emitted from the Site during excavation activities. Particulate Matter (PM10) emissions from the excavation of contaminated soils from the area sources were calculated using the 1993 USEPA document "Estimation of Air Impacts from Area Sources of Particulate Matter Emissions at Superfund Sites".

The following equation was used:

$$EF = \frac{k\,(0.0016)\,(M)\,(\frac{U}{2.2})^{1.3}}{(\frac{X_{H2O}}{2})^{1.4}}$$

Where:

EF	=	PM emissions (g);
k	=	particle size multiplier (0.35);
0.0016	=	empirical constant (g/kg);
M	=	mass of waste handled (kg);
U	=	mean wind speed (4.4 m/s);
2.2	=	empirical constant (m/s); and
X_{H2O}	=	percent moisture content (10 percent).

The particulate matter excavation emission model and the PM emission rates from the source areas are detailed in Tables 5A and 5B for the excavation of Site soils (excluding Lagoon 6) and Lagoon 6 soils, respectively.

The emission rates of SVOCs, PCBs/pesticides and inorganics were calculated using the source area characteristic concentrations in the bulk soil with applicable enrichment factors for PM10. Tables 5A and 5B summarize the SVOCs, PCBs/pesticides and inorganics for the Site soils and Lagoon 6 soils, respectively.

2.3 Emissions from Exposed Groundwater

The principal source of atmospheric contaminants from the exposed groundwater area source is expected to be the volatilization of VOCs. An appropriate mathematical emission model was selected from a review of available models. The model selected for use was the RTI Lagoon Model.

The RTI model is a simple volatile constituent mass transfer model and is based on the following equation:

$$ER_i = K_i \bullet A \bullet C_i$$

Where:

ER_i	=	emission rate of compound (grams/sec);
K_i	=	overall mass transfer coefficient of compound i;
C_i	=	liquid-phase concentration of compound i (g/cm^3);
A	=	exposed surface area (cm^2);

and

$$\frac{1}{K_i} = \frac{1}{k_{iL}} + \frac{RT}{H_i k_{iG}}$$

Where:

k_{iL} = Liquid-phase mass transfer coefficient, cm/s;

R = Ideal gas constant, 8.2×10^{-5} atm-m³/mole-°K;

T = Absolute temperature, °K;

H_i = Henry's Law constant of component I, atm-m³/mole; and

k_{iG} = Gas-phase mass transfer coefficient, cm/s.

Estimation of liquid-phase mass transfer coefficient (k_{iL}):

$$k_{iL} = \left(\frac{MW_{O_2}}{MW_i}\right)^{0.5} \left(\frac{T}{298}\right)(k_L, O_2)$$

Where:

k_{iL} = Liquid-phase mass transfer coefficient, cm/s;

$MWO_2; MW_i$ = Molecular weights of oxygen (32.0) and component i, respectively, g/mole;

T = Absolute temperature, °K; and

k_L, O_2 = Liquid-phase mass transfer coefficient for oxygen at 25°C, cm/s (default = 0.002cm/s).

Estimation of gas-phase mass transfer coefficient (k_{iG}):

$$k_{iG} = \left(\frac{MW_{H_2O}}{MW_i}\right)^{0.335} \left(\frac{T}{298}\right)^{1.005} (k_{iG}, H_2O)$$

Where:

k_{iG} = Gas-phase mass transfer coefficient, cm/s;

$MW_{H_2O}; MW_i$ = Molecular weights of water (18.0) and component i, respectively, g/mole;

T = Absolute temperature, °K; and

k_{iG}, H_2O = Gas-phase mass transfer coefficient of water vapor at 25°C, cm/s (default = 0.833cm/s).

The RTI model is applicable to assessing gaseous emissions from non-aerated surface impoundment and contaminants (in solution) pooled at soil surfaces. Further information regarding the RTI model is provided in the USEPA document "Air/Superfund National Technical Guidance Study Series, Guideline for Predictive Baseline Emissions Estimation Procedures for Superfund Sites" and "Evaluation and Selection of Models for Estimating Air Emissions From Hazardous Waste Treatment, Storage, and Disposal Facilities, Section 2, Office of Air Quality Planning and Standards, Research Triangle Park, NC".

The air emissions from impacted groundwater are conservatively assumed to be released from a potential area source of 10 meters by 10 meters [100 square meter (m^2)]. Table 6A provides a summary of the estimated maximum VOC emissions rates from the groundwater area source using the RTI model.

3.0 SCREENING AIR DISPERSION MODELING

Air dispersion modeling was completed to estimate the maximum ground level concentrations of the contaminants potentially emitted from the source areas under excavation and with exposed soil and groundwater.

A conservative air dispersion model, SCREEN3, recommended by USEPA for screening purposes was used. SCREEN3 uses Gaussian dispersion equations and worst-case meteorological conditions to estimate maximum downwind concentrations due to emissions from a source. The SCREEN3 model will provide more conservative modeling results than a detailed dispersion model such as the Industrial Source Complex (ISC3) model.

The SCREEN3 model utilizes rectangular source areas and only one source may be modeled at a time. A 290 m^2 area, 17 m x 17 m was used for the excavated soil surface area while a 100 m^2 area, 10 m x 10 m, was used for the exposed groundwater area.

The SCREEN3 model was used to determine the worst-case atmospheric conditions that would produce the maximum ground level concentrations. The modeling showed that maximum concentrations occurred for stability Class 6 (stable) and a wind speed of 1 meter per second (m/s). The maximum concentration occurs at the edge of the area source. Source and receptor heights were both set at 0 m and urban dispersion coefficients were used. The SCREEN3 model output for a normalized 1 gram per second emission rate over each surface area is provided in Attachment A.

It is noted that the SCREEN3 model calculates 1-hour concentrations. The 1-hour concentrations were multiplied by a time averaging conversion factor of 0.7 to obtain 8-hour concentrations for use in construction worker exposure assessment. The time averaging is recommended in the EPA publication "Air/Superfund National Technical Guidance Study Series", Document Number EPA-451/R-93-005.

Maximum 8-hour ground level concentrations due to VOC emissions during soils excavation were calculated and are detailed in Tables 4A and 4B for the Site soils and Lagoon 6 soils, respectively while the particulate/SVOC/PCB/metals emissions are summarized in Tables 5A and 5B. Table 6B summarizes the estimated maximum 8-hour ground level concentrations for VOC emissions from exposed groundwater.

Also summarized on the above tables are the American Conference of Industrial Hygienists (ACGIH) Threshold Limit Values (TLVs). The TLVs are the time-weighted average concentrations for a conventional 8-hour workday and a 40-hour workweek, to which it is believed that nearly all workers may be repeatedly

exposed, day after day, without adverse effects. All estimated maximum 8-hour concentrations are below their respective TLVs, except for benzene, whose estimated concentration for excavation of Site soils (excluding Lagoon 6) is 7,780 $\mu g/m^3$ and is above its TLV of 1,600 $\mu g/m^3$.

Should you have any questions please do not hesitate to contact us.

TABLE 1A

EXPOSURE POINT CONCENTRATION (EPC) SUMMARY FOR CHEMICALS OF POTENTIAL CONCERN IN SOIL
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Soil
Exposure Point: Ingestion, Dermal and Inhalation

Chemical of Potential Concern	*Units*	*Arithmetic Mean*	*95% UCL of Normal Data*	*Maximum Detected Concentration*	*Maximum Qualifier*	*EPC Units*	*Reasonable Maximum Exposure*			*Central Tendency*		
							Medium EPC Value	*Medium EPC Statistic*	*Medium EPC Rationale*	*Medium EPC Value*	*Medium EPC Statistic*	*Medium EPC Rationale*
VOCs												
Acetone	ug/kg	9.43E+01	(1)	2.80E+03		ug/kg	5.13E+02	95% UCL-NP	W-Test (5)	5.13E+02	95% UCL-NP	W-Test (5)
Carbon Disulfide	ug/kg	3.57E+01	(1)	2.70E+01	J	ug/kg	2.70E+01	Max.	(4)	2.70E+01	Max.	(4)
1,2-Dichloroethene (total)	ug/kg	3.50E+01	(1)	4.00E+00	J	ug/kg	4.00E+00	Max.	(4)	4.00E+00	Max.	(4)
1,2-Dichloroethane	ug/kg	3.52E+01	(1)	7.00E+00		ug/kg	7.00E+00	Max.	(4)	7.00E+00	Max.	(4)
2-Butanone	ug/kg	1.31E+01	(1)	2.40E+02		ug/kg	5.03E+01	95% UCL-NP	W-Test (5)	5.03E+01	95% UCL-NP	W-Test (5)
1,2-Dichloropropane	ug/kg	5.14E+00	(1)	2.00E+00	J	ug/kg	2.00E+00	Max.	(4)	2.00E+00	Max.	(4)
Trichloroethene	ug/kg	3.51E+01	(1)	1.30E+01	J	ug/kg	1.30E+01	Max.	(4)	1.30E+01	Max.	(4)
Benzene	ug/kg	2.86E+02	(1)	1.30E+04		ug/kg	4.44E+03	95% UCL-NP	W-Test (5)	4.44E+03	95% UCL-NP	W-Test (5)
4-Methyl-2-Pentanone	ug/kg	2.61E+01	(1)	8.40E+02		ug/kg	2.24E+02	95% UCL-NP	W-Test (5)	2.24E+02	95% UCL-NP	W-Test (5)
Tetrachloroethene	ug/kg	6.43E+01	(1)	5.10E+01	J	ug/kg	5.10E+01	Max.	(4)	5.10E+01	Max.	(4)
Toluene	ug/kg	1.84E+03	(1)	5.20E+04		ug/kg	1.01E+04	95% UCL-NP	W-Test (5)	1.01E+04	95% UCL-NP	W-Test (5)
Chlorobenzene	ug/kg	4.72E+02	(1)	1.20E+04		ug/kg	1.01E+03	95% UCL-NP	W-Test (5)	1.01E+03	95% UCL-NP	W-Test (5)
Ethylbenzene	ug/kg	1.11E+03	(1)	3.20E+04		ug/kg	2.29E+03	95% UCL-NP	W-Test (5)	2.29E+03	95% UCL-NP	W-Test (5)
Styrene	ug/kg	5.24E+01	(1)	2.60E+03	J	ug/kg	1.37E+03	95% UCL-NP	W-Test (5)	1.37E+03	95% UCL-NP	W-Test (5)
Xylenes	ug/kg	8.28E+03	(1)	3.00E+05		ug/kg	6.96E+04	95% UCL-NP	W-Test (5)	6.96E+04	95% UCL-NP	W-Test (5)
TPH	mg/kg	3.70E+02	(1)	4.98E+03		mg/kg	2.87E+03	95% UCL-NP	W-Test (5)	2.87E+03	95% UCL-NP	W-Test (5)
VOC TICs												
Cyclohexane	ug/kg	3.17E+01	1.02E+02	8.00E+01		ug/kg	8.00E+01	Max.	(4)	8.00E+01	Max.	(4)
Methyl-cyclohexane Isomer	ug/kg	1.50E+01	(2)	1.90E+01		ug/kg	1.90E+01	Max.	(2)	1.90E+01	Max.	(2)
2-Butoxy-ethanol	ug/kg	2.90E+01	(2)	2.90E+01		ug/kg	2.90E+01	Max.	(2)	2.90E+01	Max.	(2)
Trimethylpentane Isomer	ug/kg	1.50E+01	(2)	1.50E+01		ug/kg	1.50E+01	Max.	(2)	1.50E+01	Max.	(2)
Propylbenzene	ug/kg	2.45E+03	(3)	1.20E+04		ug/kg	1.20E+04	Max.	(4)	1.20E+04	Max.	(4)
Methyl-pentene Isomer	ug/kg	1.20E+01	(2)	1.20E+01		ug/kg	1.20E+01	Max.	(2)	1.20E+01	Max.	(2)
Ethyl-cyclopentane	ug/kg	1.00E+01	(2)	1.00E+01		ug/kg	1.00E+01	Max.	(2)	1.00E+01	Max.	(2)
SVOCs												
4-Chloro-3-Methylphenol	ug/kg	2.34E+02	(1)	4.20E+01	J	ug/kg	4.20E+01	Max.	(4)	4.20E+01	Max.	(4)
Bis(2-ethylhexyl)phthalate	ug/kg	1.39E+03	(1)	6.00E+04	E	ug/kg	1.66E+04	95% UCL-NP	W-Test (5)	1.66E+04	95% UCL-NP	W-Test (5)
Benzo(a)pyrene	ug/kg	2.45E+02	(1)	2.00E+02		ug/kg	2.00E+02	Max.	(4)	2.00E+02	Max.	(4)
Alpha-picoline	ug/kg	3.65E+02	(1)	7.60E+03	J	ug/kg	2.53E+03	95% UCL-NP	W-Test (5)	2.53E+03	95% UCL-NP	W-Test (5)
2-Aminopyridine	ug/kg	5.33E+03	(1)	9.90E+04		ug/kg	2.34E+04	95% UCL-NP	W-Test (5)	2.34E+04	95% UCL-NP	W-Test (5)
SVOC TICs												
1,4-Benzenediamine	ug/kg	4.00E+02	(2)	4.00E+02		ug/kg	4.00E+02	Max.	(2)	4.00E+02	Max.	(2)
1H-indazole, 4,5,6,7-tetrahydro	ug/kg	2.00E+02	(2)	2.00E+02		ug/kg	2.00E+02	Max.	(2)	2.00E+02	Max.	(2)
1-Nitroethyl-benzene	ug/kg	3.00E+01	(2)	3.00E+01		ug/kg	3.00E+01	Max.	(2)	3.00E+01	Max.	(2)
2-Chloroethyl benzene	ug/kg	1.00E+04	(2)	1.00E+04		ug/kg	1.00E+04	Max.	(2)	1.00E+04	Max.	(2)
2-Chloro-5-(trifluoro)-benzeneamine	ug/kg	1.10E+04	(2)	1.10E+04		ug/kg	1.10E+04	Max.	(2)	1.10E+04	Max.	(2)
2,2'-Methylenedithiophene	ug/kg	1.50E+04	(2)	1.50E+04		ug/kg	1.50E+04	Max.	(2)	1.50E+04	Max.	(2)
2,4-Bipyridyl	ug/kg	2.00E+02	(2)	2.00E+02		ug/kg	2.00E+02	Max.	(2)	2.00E+02	Max.	(2)
3-Methyl-2-pyridinamine	ug/kg	3.00E+02	(2)	3.00E+02		ug/kg	3.00E+02	Max.	(2)	3.00E+02	Max.	(2)
3,5-Dimethyl-1-phenypyrazole	ug/kg	5.00E+02	(2)	5.00E+02		ug/kg	5.00E+02	Max.	(2)	5.00E+02	Max.	(2)
5-Ethyl-2-methyl-Pyridine	ug/kg	8.93E+02	(3)	3.00E+03		ug/kg	2.01E+03	95% UCL-T	W-Test (5)	2.01E+03	95% UCL-T	W-Test (5)
5-Methyl-2-pyridinamine	ug/kg	3.00E+02	(2)	3.00E+02		ug/kg	3.00E+02	Max.	(2)	3.00E+02	Max.	(2)
6-Methyl-2-pyridinamine	ug/kg	2.00E+02	(2)	2.00E+02		ug/kg	2.00E+02	Max.	(2)	2.00E+02	Max.	(2)
Acetophenone	ug/kg	3.99E+03	(2)	7.60E+03		ug/kg	7.60E+03	Max.	(2)	7.60E+03	Max.	(2)
Benzaldehyde	ug/kg	4.02E+02	5.80E+02	6.70E+02		ug/kg	5.80E+02	95% UCL-N	W-Test (5)	5.80E+02	95% UCL-N	W-Test (5)
Benzamide	ug/kg	4.38E+03	(2)	8.20E+03		ug/kg	8.20E+03	Max.	(2)	8.20E+03	Max.	(2)
Benzene, 1,1'-oxybis-	ug/kg	3.00E+02	(2)	3.00E+02		ug/kg	3.00E+02	Max.	(2)	3.00E+02	Max.	(2)
Benzene, 1-chloro-4(1-methyl	ug/kg	8.00E+02	(2)	8.00E+02		ug/kg	8.00E+02	Max.	(2)	8.00E+02	Max.	(2)
Bicyclo(3.1.1)hept-2-ene, 3,	ug/kg	2.00E+02	(2)	2.00E+02		ug/kg	2.00E+02	Max.	(2)	2.00E+02	Max.	(2)
Bipyridine Isomer	ug/kg	3.54E+03	(3)	1.60E+04		ug/kg	5.79E+03	95% UCL-T	W-Test (5)	5.79E+03	95% UCL-T	W-Test (5)
Bromohexane isomer	ug/kg	4.00E+02	(2)	4.00E+02		ug/kg	4.00E+02	Max.	(2)	4.00E+02	Max.	(2)
Chlorothioxanthenone isomer 1	ug/kg	2.00E+02	(2)	2.00E+02		ug/kg	2.00E+02	Max.	(2)	2.00E+02	Max.	(2)
Chlorothioxanthenone isomer 2	ug/kg	1.00E+02	(2)	1.00E+02		ug/kg	1.00E+02	Max.	(2)	1.00E+02	Max.	(2)
Diisopropylether	ug/kg	4.00E+01	(2)	4.00E+01		ug/kg	4.00E+01	Max.	(2)	4.00E+01	Max.	(2)
Diethylbenzeneamine isomer	ug/kg	2.00E+03	(2)	2.00E+03		ug/kg	2.00E+03	Max.	(2)	2.00E+03	Max.	(2)
Dimethylbenzeneamine isomer	ug/kg	3.00E+02	(2)	3.00E+02		ug/kg	3.00E+02	Max.	(2)	3.00E+02	Max.	(2)
Diphenyl ether	ug/kg	1.00E+03	(2)	1.00E+03		ug/kg	1.00E+03	Max.	(2)	1.00E+03	Max.	(2)
Ethyl methyl benzene isomer	ug/kg	1.67E+01	(1)	2.00E+01		ug/kg	2.00E+01	Max.	(2)	2.00E+01	Max.	(2)
Ethylmethylpyridine isomer	ug/kg	1.48E+03	(3)	5.90E+03		ug/kg	2.88E+03	95% UCL-T	W-Test (5)	2.88E+03	95% UCL-T	W-Test (5)
Ethenyl pyridine isomer	ug/kg	3.10E+03	(2)	6.00E+03		ug/kg	6.00E+03	Max.	(2)	6.00E+03	Max.	(2)
Ethyl pyridine isomer	ug/kg	4.50E+02	(2)	7.00E+02		ug/kg	7.00E+02	Max.	(2)	7.00E+02	Max.	(2)

TABLE 1A

EXPOSURE POINT CONCENTRATION (EPC) SUMMARY FOR CHEMICALS OF POTENTIAL CONCERN IN SOIL
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

Scenario Timeframe: Future
Medium: Soil
Exposure Medium: Soil
Exposure Point: Ingestion, Dermal and Inhalation

Chemical of Potential Concern	*Units*	*Arithmetic Mean*	*95% UCL of Normal Data*	*Maximum Detected Concentration*	*Maximum Qualifier*	*EPC Units*	*Reasonable Maximum Exposure*			*Central Tendency*		
							Medium EPC Value	*Medium EPC Statistic*	*Medium EPC Rationale*	*Medium EPC Value*	*Medium EPC Statistic*	*Medium EPC Rationale*
SVOC TICs (cont.'d)												
Methyl-pyridinamine isomer	ug/kg	2.00E+02	(2)	2.00E+02		ug/kg	2.00E+02	Max.	(2)	2.00E+02	Max.	(2)
Methyl pyridine isomer	ug/kg	6.50E+01	(2)	1.00E+02		ug/kg	1.00E+02	Max.	(2)	1.00E+02	Max.	(2)
Naphthalene, 1,2,3,4-tetrahydro	ug/kg	1.07E+04	(3)	4.40E+04		ug/kg	4.40E+04	Max.	(4)	4.40E+04	Max.	(4)
Nonane	ug/kg	1.00E+01	(2)	1.00E+01		ug/kg	1.00E+01	Max.	(2)	1.00E+01	Max.	(2)
Phenothiazine	ug/kg	1.09E+03	(2)	1.30E+03		ug/kg	1.30E+03	Max.	(2)	1.30E+03	Max.	(2)
Sulfur, mol (S8)	ug/kg	1.50E+03	(2)	2.00E+03		ug/kg	2.00E+03	Max.	(2)	2.00E+03	Max.	(2)
Pesticides/PCBs												
Aldrin	ug/kg	3.62E+00	(1)	7.70E+01	J	ug/kg	1.63E+01	95% UCL-NP	W-Test (5)	1.63E+01	95% UCL-NP	W-Test (5)
Dieldrin	ug/kg	7.72E+00	(1)	1.70E+02	J	ug/kg	3.41E+01	95% UCL-NP	W-Test (5)	3.41E+01	95% UCL-NP	W-Test (5)
Aroclor-1254	ug/kg	4.10E+02	(1)	1.50E+04		ug/kg	2.59E+03	95% UCL-NP	W-Test (5)	2.59E+03	95% UCL-NP	W-Test (5)
Aroclor-1260	ug/kg	1.39E+02	(1)	4.30E+03	J	ug/kg	7.33E+02	95% UCL-NP	W-Test (5)	7.33E+02	95% UCL-NP	W-Test (5)
INORGANICS												
Aluminum	mg/kg	1.58E+04	(1)	2.94E+05		mg/kg	3.14E+04	95% UCL-NP	W-Test (5)	3.14E+04	95% UCL-NP	W-Test (5)
Antimony	mg/kg	1.58E+00	(3)	6.75E+01		mg/kg	1.80E+00	95% UCL-T	W-Test (5)	1.80E+00	95% UCL-T	W-Test (5)
Arsenic	mg/kg	6.91E+00	(1)	8.75E+01		mg/kg	1.13E+01	95% UCL-NP	W-Test (5)	1.13E+01	95% UCL-NP	W-Test (5)
Cadmium	mg/kg	7.04E-01	(1)	1.36E+01		mg/kg	9.13E-01	95% UCL-NP	W-Test (5)	9.13E-01	95% UCL-NP	W-Test (5)
Chromium	mg/kg	2.10E+01	(1)	1.84E+02		mg/kg	3.04E+01	95% UCL-NP	W-Test (5)	3.04E+01	95% UCL-NP	W-Test (5)
Copper	mg/kg	7.31E+01	(1)	2.89E+03		mg/kg	2.18E+02	95% UCL-NP	W-Test (5)	2.18E+02	95% UCL-NP	W-Test (5)
Iron	mg/kg	2.73E+04	(1)	4.24E+04		mg/kg	2.80E+04	95% UCL-NP	W-Test (5)	2.80E+04	95% UCL-NP	W-Test (5)
Manganese	mg/kg	8.83E+02	9.3E+02	2.47E+03		mg/kg	9.30E+02	95% UCL-N	W-Test (5)	9.30E+02	95% UCL-N	W-Test (5)
Mercury	mg/kg	2.34E+00	(1)	7.99E+01		mg/kg	4.67E+00	95% UCL-NP	W-Test (5)	4.67E+00	95% UCL-NP	W-Test (5)
Thallium	mg/kg	6.85E-01	(1)	2.00E+00	J	mg/kg	7.58E-01	95% UCL-NP	W-Test (5)	7.58E-01	95% UCL-NP	W-Test (5)
Vanadium	mg/kg	2.17E+01	(1)	2.04E+02		mg/kg	3.17E+01	95% UCL-NP	W-Test (5)	3.17E+01	95% UCL-NP	W-Test (5)

Notes:

For non-detects, 1/2 laboratory detection limit was used as a proxy concentration.
W-Test : Developed by Shapiro and Wilks for data sets with under 50 samples.
W-Test : Developed by Shapiro and Francia for data sets with greater than 50 samples.
Refer to USEPA Supplemental Guidance to RAGS: Calculating the Concentration Term (RAGS, 1992), OSWER Directive 9285.7-081, May 1992.
Statistics: Maximum Detected Value (Max); 95% UCL of Normal Data (95% UCL-N); 95% UCL of Log-transformed Data (95% UCL-T);
95% UCL of Non-Parametrically Distributed Data (95% UCL-NP); Mean of Log-transformed Data (Mean-T); Mean of Normal Data (Mean-N).
J = Associated value is estimated.
E = Associated value is outside the calibration range.

(1) Data set is neither normally or lognormally distributed.
(2) Due to a limited number of samples, a 95% UCL can't be calculated and therefore the maximum detected concentration is used for the CT and RME EPC.
(3) Data set is log-normally distributed.
(4) The 95% UCL calculated is greater than maximum detected concentration; therefore maximum detected concentration is the CT and RME EPC.
(5) Shapiro-Wilks W Test was used for data sets where n<=50 and the Shapiro-Francia W Test was used for data sets where : 50<n<100.

TABLE 1B

EXPOSURE POINT CONCENTRATION (EPC) SUMMARY FOR CHEMICALS OF POTENTIAL CONCERN IN LAGOON 6 SOIL
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

Scenario Timeframe: Future
Medium: Lagoon 6 Soil
Exposure Medium: Lagoon 6 Soil
Exposure Point: Ingestion, Dermal and Inhalation

Chemical of Potential Concern	*Units*	*Arithmetic Mean*	*95% UCL of Normal Data*	*Maximum Detected Concentration*	*Maximum Qualifier*	*EPC Units*	*Reasonable Maximum Exposure*			*Central Tendency*		
							Medium EPC Value	*Medium EPC Statistic*	*Medium EPC Rationale*	*Medium EPC Value*	*Medium EPC Statistic*	*Medium EPC Rationale*
VOCs												
Acetone	ug/kg	6.33E+00	(1)	8.00E+00		ug/kg	8.00E+00	Max.	(4)	8.00E+00	Max.	(4)
Tetrachloroethene	ug/kg	4.17E+00	1.82E+01	4.00E+00		ug/kg	4.00E+00	Max.	(4)	4.00E+00	Max.	(4)
Toluene	ug/kg	6.04E+02	(1)	1.80E+03		ug/kg	1.80E+03	Max.	(4)	1.80E+03	Max.	(4)
TPH	mg/kg	3.00E+02	(2)	3.00E+02		mg/kg	3.00E+02	Max.	(2)	3.00E+02	Max.	(2)
SVOC TICs												
1,2-Propanedione, 1-phenyl	ug/kg	2.00E+03	(2)	2.00E+03		ug/kg	2.00E+03	Max.	(2)	2.00E+03	Max.	(2)
1-Propanone, 1-(3-pyridinyl)	ug/kg	2.00E+04	(2)	2.00E+04		ug/kg	2.00E+04	Max.	(2)	2.00E+04	Max.	(2)
2-Butyl pyridine	ug/kg	1.00E+03	(2)	1.00E+03		ug/kg	1.00E+03	Max.	(2)	1.00E+03	Max.	(2)
2-Ethenyl pyridine	ug/kg	7.00E+03	(2)	7.00E+03		ug/kg	7.00E+03	Max.	(2)	7.00E+03	Max.	(2)
Dichlorobiphenyl Isomer	ug/kg	1.04E+04	(2)	2.00E+04		ug/kg	2.00E+04	Max.	(2)	2.00E+04	Max.	(2)
Methyl ester benzoic acid	ug/kg	5.00E+03	(2)	5.00E+03		ug/kg	5.00E+03	Max.	(2)	5.00E+03	Max.	(2)
Methyl phenanthrene Isomer	ug/kg	5.00E+02	(2)	5.00E+02		ug/kg	5.00E+02	Max.	(2)	5.00E+02	Max.	(2)
Pesticides/PCBs												
Aldrin	ug/kg	3.88E+01	(1)	1.90E+02	J	ug/kg	1.90E+02	Max.	(4)	1.90E+02	Max.	(4)
Aroclor-1254	ug/kg	5.39E+03	1.33E+04	9.20E+03	J	ug/kg	9.20E+03	Max.	(4)	9.20E+03	Max.	(4)
INORGANICS												
Aluminum	mg/kg	1.53E+04	(1)	1.96E+04		mg/kg	1.65E+04	95% UCL-NP	W-Test (5)	1.65E+04	95% UCL-NP	W-Test (5)
Arsenic	mg/kg	1.16E+01	(1)	4.76E+01		mg/kg	4.63E+01	95% UCL-NP	W-Test (5)	4.63E+01	95% UCL-NP	W-Test (5)
Cadmium	mg/kg	4.08E-01	(1)	4.40E+00		mg/kg	3.94E+00	95% UCL-NP	W-Test (5)	3.94E+00	95% UCL-NP	W-Test (5)
Chromium	mg/kg	2.59E+01	(3)	6.07E+01		mg/kg	3.40E+01	95% UCL-T	W-Test (5)	3.40E+01	95% UCL-T	W-Test (5)
Iron	mg/kg	3.38E+04	3.74E+04	4.92E+04		mg/kg	3.74E+04	95% UCL-N	W-Test (5)	3.74E+04	95% UCL-N	W-Test (5)
Manganese	mg/kg	5.46E+02	6.99E+02	1.09E+03		mg/kg	6.99E+02	95% UCL-N	W-Test (5)	6.99E+02	95% UCL-N	W-Test (5)
Mercury	mg/kg	2.11E+00	(1)	2.34E+01		mg/kg	2.34E+01	Max.	(4)	2.34E+01	Max.	(4)
Thallium	mg/kg	1.26E+00	1.61E+00	2.90E+00		mg/kg	1.61E+00	95% UCL-N	W-Test (5)	1.61E+00	95% UCL-N	W-Test (5)
Vanadium	mg/kg	2.27E+01	2.68E+01	4.12E+01		mg/kg	2.68E+01	95% UCL-N	W-Test (5)	2.68E+01	95% UCL-N	W-Test (5)

Notes:

For non-detects, 1/2 laboratory detection limit was used as a proxy concentration.
W-Test : Developed by Shapiro and Wilks for data sets with under 50 samples.
Refer to USEPA Supplemental Guidance to RAGS: Calculating the Concentration Term (RAGS, 1992), OSWER Directive 9285.7-081, May 1992.
Statistics: Maximum Detected Value (Max); 95% UCL of Normal Data (95% UCL-N); 95% UCL of Log-transformed Data (95% UCL-T);
95% UCL of Non-Parametrically Distributed Data (95% UCL-NP); Mean of Log-transformed Data (Mean-T); Mean of Normal Data (Mean-N).
J = Associated value is estimated.

(1) Data set is neither normally or lognormally distributed.
(2) Due to a limited number of samples, a 95% UCL can't be calculated and therefore the maximum detected concentration is used for the CT and RME EPC.
(3) Data set is lognormally distributed.
(4) The 95% UCL calculated is greater than maximum detected concentration; therefore maximum detected concentration is the CT and RME EPC.
(5) Shapiro-Wilks W Test was used since n<=50.

TABLE 1C

EXPOSURE POINT CONCENTRATION (EPC) SUMMARY FOR CHEMICALS OF POTENTIAL CONCERN IN ON-SITE GROUNDWATER

FORMER LAGOON SITE

HAMPTONBURGH, NEW YORK

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Ingestion, Dermal and Inhalation

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
VOCs (6)												
1,1-Dichloroethane	µg/L	5.13E-01	(1)	9.00E-01	J	µg/L	9.00E-01	Max	(4)	9.00E-01	Max	(4)
2-Butanone	µg/L	2.10E+01	3.73E+01	2.80E+01	J	µg/L	2.80E+01	Max	(4)	2.80E+01	Max	(4)
4-Methyl-2-pentanone	µg/L	2.91E+00	(1)	7.00E+00	J	µg/L	6.40E+00	95% UCL - NP	W-Test (5, 7)	6.40E+00	95% UCL - NP	W-Test (5, 7)
Acetone	µg/L	7.55E+01	(3)	2.90E+02	J	µg/L	2.90E+02	Max	(4)	2.90E+02	Max	(4)
Benzene	µg/L	5.88E+01	(1)	1.10E+03		µg/L	3.32E+02	95% UCL - NP	W-Test (5, 7)	3.32E+02	95% UCL - NP	W-Test (5, 7)
Carbon disulfide	µg/L	7.00E-01	(1)	6.90E+00	J	µg/L	1.18E+00	95% UCL - NP	W-Test (5, 7)	1.18E+00	95% UCL - NP	W-Test (5, 7)
Chlorobenzene	µg/L	5.97E+00	(1)	8.40E+01		µg/L	2.67E+01	95% UCL - NP	W-Test (5, 7)	2.67E+01	95% UCL - NP	W-Test (5, 7)
Cis-1,2-Dichloroethene	µg/L	5.13E-01	(1)	8.00E-01	J	µg/L	8.00E-01	Max	(4)	8.00E-01	Max	(4)
Ethylbenzene	µg/L	1.11E+01	(1)	1.50E+02		µg/L	1.23E+02	95% UCL - NP	W-Test (5, 7)	1.23E+02	95% UCL - NP	W-Test (5, 7)
Methyl Tert Butyl Ether	µg/L	8.03E-01	(1)	9.00E+00		µg/L	6.16E+00	95% UCL - NP	W-Test (5, 7)	6.16E+00	95% UCL - NP	W-Test (5, 7)
Toluene	µg/L	1.98E+00	(1)	2.30E+01		µg/L	8.93E+00	95% UCL - NP	W-Test (5, 7)	8.93E+00	95% UCL - NP	W-Test (5, 7)
Vinyl chloride	µg/L	5.22E-01	(1)	1.20E+00		µg/L	1.01E+00	95% UCL - NP	W-Test (5, 7)	1.01E+00	95% UCL - NP	W-Test (5, 7)
Xylene (total)	µg/L	2.19E+01	(1)	5.20E+02		µg/L	2.73E+02	95% UCL - NP	W-Test (5, 7)	2.73E+02	95% UCL - NP	W-Test (5, 7)
VOC TICs												
Diisopropyl ether	µg/L	5.25E+00	7.07E+00	9.00E+00	PJ	µg/L	7.07E+00	95% UCL - N	W-Test (5)	7.07E+00	95% UCL - N	W-Test (5)
n-Butyl ether	µg/L	9.00E+00	(1)	1.90E+01	PJ	µg/L	9.00E+00	95% UCL - NP	W-Test (5, 7)	9.00E+00	95% UCL - NP	W-Test (5, 7)
2-Ethyl-1-hexanol	µg/L	4.50E+00	(2)	5.00E+00	PJ	µg/L	5.00E+00	Max.	(2)	5.00E+00	Max.	(2)
Tetrahydrofuran	µg/L	4.25E+00	(2)	6.00E+00	PJ	µg/L	6.00E+00	Max.	(2)	6.00E+00	Max.	(2)
Dimethyl disulfide	µg/L	2.90E+01	(2)	3.00E+01	PJ	µg/L	3.00E+01	Max.	(2)	3.00E+01	Max.	(2)
Methanethiol	µg/L	1.00E+01	(2)	1.00E+01	PJ	µg/L	1.00E+01	Max.	(2)	1.00E+01	Max.	(2)
1,2,3,4-Tetrahydronaphthalene	µg/L	1.45E+01	(2)	1.50E+01	PJ	µg/L	1.50E+01	Max.	(2)	1.50E+01	Max.	(2)
SVOCs												
4-Chloroaniline	µg/L	5.63E+00	(1)	2.50E+01		µg/L	1.05E+01	95% UCL - NP	W-Test (5, 7)	1.05E+01	95% UCL - NP	W-Test (5, 7)
Bis(2-Ethylhexyl)phthalate	µg/L	5.06E+00	(1)	8.00E+00	J	µg/L	8.00E+00	Max	(4)	8.00E+00	Max	(4)
Pyridine	µg/L	5.03E+00	(1)	8.00E+00	J	µg/L	8.00E+00	Max	(4)	8.00E+00	Max	(4)
2-Aminopyridine	µg/L	2.64E+01	(1)	5.20E+02		µg/L	1.89E+02	95% UCL - NP	W-Test (5, 7)	1.89E+02	95% UCL - NP	W-Test (5, 7)
alpha-Picoline	µg/L	5.44E+00	(1)	1.40E+01		µg/L	1.03E+01	95% UCL - NP	W-Test (5, 7)	1.03E+01	95% UCL - NP	W-Test (5, 7)
SVOC TICs												
1(2H)-Naphthalenone, 3,4-dihydro-	µg/L	7.00E+00	(2)	8.00E+00	PJ	µg/L	8.00E+00	Max.	(2)	8.00E+00	Max.	(2)
1-Hexanol, 2-ethyl-	µg/L	6.30E+01	(2)	6.30E+01	PJ	µg/L	6.30E+01	Max.	(2)	6.30E+01	Max.	(2)
1-Propanone, 1-phenyl-	µg/L	1.61E+01	(3)	4.70E+01	PJD	µg/L	4.47E+01	95% UCL - T	W-Test (5)	4.47E+01	95% UCL - T	W-Test (5)
2(1h)-Pyridinone, 1-methyl-	µg/L	2.00E+01	(2)	2.00E+01	PJ	µg/L	2.00E+01	Max.	(2)	2.00E+01	Max.	(2)
2(3H)-Benzothiazolone	µg/L	6.00E+00	(2)	6.00E+00	PJ	µg/L	6.00E+00	Max.	(2)	6.00E+00	Max.	(2)
2,4-Bipyridyl	µg/L	7.67E+00	1.56E+01	1.50E+01	PJD	µg/L	1.50E+01	Max	(4)	1.50E+01	Max	(4)
2-Isopropyl-6-methylaniline	µg/L	2.07E+01	4.15E+01	3.10E+01	PJ	µg/L	3.10E+01	Max	(4)	3.10E+01	Max	(4)
2-Methyl-5-butylpyridine	µg/L	6.00E+00	(2)	6.00E+00	PJ	µg/L	6.00E+00	Max.	(2)	6.00E+00	Max.	(2)
2-Pyridinamide,6-methyl	µg/L	1.90E+01	(2)	1.90E+01	PJ	µg/L	1.90E+01	Max.	(2)	1.90E+01	Max.	(2)
3-Hexene, 3-ethyl-2,5-dimethyl-	µg/L	2.00E+00	(2)	2.00E+00	PJ	µg/L	2.00E+00	Max.	(2)	2.00E+00	Max.	(2)
4,4´-Difluorobiphenyl	µg/L	3.00E+00	(2)	3.00E+00	PJ	µg/L	3.00E+00	Max.	(2)	3.00E+00	Max.	(2)
Acetamide, N-(_alpha_-methylpheneth	µg/L	4.00E+00	(2)	6.00E+00	PJ	µg/L	6.00E+00	Max.	(2)	6.00E+00	Max.	(2)
Aniline	µg/L	1.23E+01	(2)	1.60E+01	PJ	µg/L	1.60E+01	Max.	(2)	1.60E+01	Max.	(2)
Benzaldehyde, 3-hydroxy-4-methoxy-	µg/L	4.00E+00	(2)	4.00E+00	PJ	µg/L	4.00E+00	Max.	(2)	4.00E+00	Max.	(2)
Benzaldehyde, 4-hydroxy-	µg/L	6.00E+00	(2)	6.00E+00	PJ	µg/L	6.00E+00	Max.	(2)	6.00E+00	Max.	(2)
Benzenamine, 2-chloro-5-(trifluorm	µg/L	9.00E+00	(3)	2.80E+01	PJD	µg/L	2.60E+01	95% UCL - T	W-Test (5)	2.60E+01	95% UCL - T	W-Test (5)
Benzenamine, 2,6-bis(1-methylethyl)	µg/L	2.88E+01	4.30E+01	4.10E+01	PJ	µg/L	4.10E+01	Max	(4)	4.10E+01	Max	(4)
Benzenamine, 3-(trifluoromethyl)-	µg/L	4.83E+01	(3)	1.40E+02	PJD	µg/L	1.40E+02	Max	(4)	1.40E+02	Max	(4)
Benzenamine, 4-methoxy-	µg/L	5.20E+01	(2)	5.20E+01	PJ	µg/L	5.20E+01	Max.	(2)	5.20E+01	Max.	(2)
Benzenamine, 4-methoxy-2-methyl	µg/L	2.60E+01	(2)	2.60E+01	PJ	µg/L	2.60E+01	Max.	(2)	2.60E+01	Max.	(2)
Benzenamine, 4-methoxy-N-methyl-	µg/L	1.80E+02	(2)	1.80E+02	PJ	µg/L	1.80E+02	Max.	(2)	1.80E+02	Max.	(2)
Benzeneacetic acid, _alpha_-methoxy	µg/L	5.00E+00	(2)	5.00E+00	PJ	µg/L	5.00E+00	Max.	(2)	5.00E+00	Max.	(2)
Benzenemethanol, 4-methyl-	µg/L	3.00E+00	(2)	3.00E+00	PJ	µg/L	3.00E+00	Max.	(2)	3.00E+00	Max.	(2)
Benzenethanol, 4-chloro-_alpha_,_al	µg/L	1.15E+01	(2)	1.20E+01	PJ	µg/L	1.20E+01	Max.	(2)	1.20E+01	Max.	(2)
Benzoic Acid	µg/L	7.00E+01	(2)	7.00E+01	PJ	µg/L	7.00E+01	Max.	(2)	7.00E+01	Max.	(2)
Bicyclo(3_2_2)non-2-ene, 2-phenyl-	µg/L	3.00E+00	(2)	3.00E+00	J	µg/L	3.00E+00	Max.	(2)	3.00E+00	Max.	(2)
Butanoic acid	µg/L	1.20E+01	(2)	1.20E+01	PJ	µg/L	1.20E+01	Max.	(2)	1.20E+01	Max.	(2)
Butanoic acid, 3-methyl	µg/L	1.60E+01	(2)	1.60E+01	PJ	µg/L	1.60E+01	Max.	(2)	1.60E+01	Max.	(2)
Cyclopentanone, 2,5-bis(phenylmethy	µg/L	5.00E+00	(2)	5.00E+00	PJ	µg/L	5.00E+00	Max.	(2)	5.00E+00	Max.	(2)
Diethyltoluamide	µg/L	1.56E+01	(1)	7.50E+01	PJ	µg/L	7.50E+01	Max	(4)	7.50E+01	Max	(4)
difluorobiphenyl isomer+unknown	µg/L	2.00E+00	(2)	2.00E+00	J	µg/L	2.00E+00	Max.	(2)	2.00E+00	Max.	(2)
Dimethyl pyridine isomer	µg/L	3.95E+01	(2)	4.00E+01	J	µg/L	4.00E+01	Max.	(2)	4.00E+01	Max.	(2)
Ethanol, 2-(2-butoxyethoxy)-	µg/L	2.50E+00	(2)	3.00E+00	PJ	µg/L	3.00E+00	Max.	(2)	3.00E+00	Max.	(2)
Ethanol, 2-butoxy-	µg/L	1.20E+01	(2)	1.20E+01	PJ	µg/L	1.20E+01	Max.	(2)	1.20E+01	Max.	(2)
Fluoronitrophenol isomer	µg/L	2.00E+00	(2)	2.00E+00	J	µg/L	2.00E+00	Max.	(2)	2.00E+00	Max.	(2)
Formamide, N,N-dimethyl-	µg/L	4.00E+00	(2)	4.00E+00	PJ	µg/L	4.00E+00	Max.	(2)	4.00E+00	Max.	(2)
Methanone, phenyl-2-pyridinyl-	µg/L	4.00E+00	(2)	4.00E+00	PJ	µg/L	4.00E+00	Max.	(2)	4.00E+00	Max.	(2)
Nonanoic acid	µg/L	8.00E+00	(2)	8.00E+00	PJ	µg/L	8.00E+00	Max.	(2)	8.00E+00	Max.	(2)

TABLE 1C

EXPOSURE POINT CONCENTRATION (EPC) SUMMARY FOR CHEMICALS OF POTENTIAL CONCERN IN ON-SITE GROUNDWATER

FORMER LAGOON SITE

HAMPTONBURGH, NEW YORK

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Ingestion, Dermal and Inhalation

Chemical of Potential Concern	Units	Arithmetic Mean	95% UCL of Normal Data	Maximum Detected Concentration	Maximum Qualifier	EPC Units	Reasonable Maximum Exposure			Central Tendency		
							Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale	Medium EPC Value	Medium EPC Statistic	Medium EPC Rationale
SVOC TICs (cont.'d)												
Octadecanoic acid, 1,2-ethanediyl e	µg/L	6.40E+01	(2)	6.40E+01	PJ	µg/L	6.40E+01	Max.	(2)	6.40E+01	Max.	(2)
Octadecanoic acid, 2-((1-oxohexadec	µg/L	5.00E+01	(2)	7.70E+01	PJ	µg/L	7.70E+01	Max.	(2)	7.70E+01	Max.	(2)
Oxazole, 2,5-dimethyl-4-phenyl-	µg/L	6.50E+00	(2)	7.00E+00	PJ	µg/L	7.00E+00	Max.	(2)	7.00E+00	Max.	(2)
p-Diethylaminoacetophenone	µg/L	4.00E+00	(2)	4.00E+00	PJ	µg/L	4.00E+00	Max.	(2)	4.00E+00	Max.	(2)
Phenol, diethyl-	µg/L	2.70E+01	(2)	2.70E+01	PJ	µg/L	2.70E+01	Max.	(2)	2.70E+01	Max.	(2)
Pyridine, 2,4-dimethyl-	µg/L	4.40E+01	(2)	4.40E+01	PJD	µg/L	4.40E+01	Max.	(2)	4.40E+01	Max.	(2)
Pyridine, 2-chloro-	µg/L	1.18E+01	(1)	6.20E+01	PJD	µg/L	6.20E+01	Max	(4)	6.20E+01	Max	(4)
Pyridine, 2-ethyl-5-methyl-	µg/L	2.10E+01	3.71E+01	3.00E+01	PJ	µg/L	3.00E+01	Max	(4)	3.00E+01	Max	(4)
Pyridine, 3-ethyl-	µg/L	4.13E+01	(3)	1.80E+02	PJD	µg/L	1.80E+02	Max	(4)	1.80E+02	Max	(4)
Pyridine, 4-methyl-2-(2-methyl-1-pr	µg/L	1.40E+01	(2)	1.40E+01	PJ	µg/L	1.40E+01	Max.	(2)	1.40E+01	Max.	(2)
Pyridine, 5-ethyl-2-methyl-	µg/L	5.83E+01	(3)	2.70E+02	PJ	µg/L	1.67E+02	95% UCL - T	W-Test (5)	1.67E+02	95% UCL - T	W-Test (5)
Undecanoic acid	µg/L	4.00E+00	(2)	4.00E+00	PJ	µg/L	4.00E+00	Max.	(2)	4.00E+00	Max.	(2)
INORGANICS												
Antimony	µg/L	2.58E+00	(1)	1.44E+01		µg/L	1.44E+01	Max	(4)	1.44E+01	Max	(4)
Arsenic	µg/L	3.66E+00	(1)	3.27E+01	J	µg/L	1.29E+01	95% UCL - NP	W-Test (5, 7)	1.29E+01	95% UCL - NP	W-Test (5, 7)
Barium	µg/L	7.00E+01	(3)	5.18E+02		µg/L	1.52E+02	95% UCL - T	W-Test (5)	1.52E+02	95% UCL - T	W-Test (5)
Chromium	µg/L	2.57E+00	(1)	3.43E+01		µg/L	1.06E+01	95% UCL - NP	W-Test (5, 7)	1.06E+01	95% UCL - NP	W-Test (5, 7)
Iron	µg/L	9.27E+02	(3)	6.73E+03		µg/L	2.34E+03	95% UCL - T	W-Test (5)	2.34E+03	95% UCL - T	W-Test (5)
Lead	µg/L	4.36E+00	(1)	1.09E+02	J	µg/L	7.30E+01	95% UCL - NP	W-Test (5, 7)	7.30E+01	95% UCL - NP	W-Test (5, 7)
Manganese	µg/L	1.39E+03	(1)	7.49E+03		µg/L	1.40E+03	95% UCL - NP	W-Test (5, 7)	1.40E+03	95% UCL - NP	W-Test (5, 7)
Vanadium	µg/L	1.07E+00	(1)	4.50E+00		µg/L	1.86E+00	95% UCL - NP	W-Test (5, 7)	1.86E+00	95% UCL - NP	W-Test (5, 7)
Cyanide (total)	µg/L	2.54E+01	(1)	1.66E+02		µg/L	4.21E+01	95% UCL - NP	W-Test (5, 7)	4.21E+01	95% UCL - NP	W-Test (5, 7)

Notes:

For non-detects, 1/2 laboratory detection limit was used as a proxy concentration.

W-Test : Developed by Shapiro and Wilks for data sets with under 50 samples.
Refer to USEPA Supplemental Guidance to RAGS: Calculating the Concentration Term (RAGS, 1992), OSWER Directive 9285.7-081, May 1992.

Statistics: Maximum Detected Value (Max); 95% UCL of Normal Data (95% UCL-N); 95% UCL of Log-transformed Data (95% UCL-T);
95% UCL of Non-Parametrically Distributed Data (95% UCL-NP); Mean of Log-transformed Data (Mean-T); Mean of Normal Data (Mean-N).

J = Associated value is estimated.

P = Presumptively identified.

D = Result from sample dilution.

(1) Data set is neither normally or lognormally distributed.

(2) Due to a limited number of samples, a 95% UCL can't be calculated and therefore the maximum detected concentration is used for the CT and RME EPC.

(3) Data set is log-normally distributed.

(4) The 95% UCL calculated is greater than maximum detected concentration; therefore maximum detected concentration is the CT and RME EPC.

(5) Shapiro-Wilk W Test was used for data sets where n<=50.

(6) All detected VOCs were modeled for the potential ambient air exposure pathway; with the exception of benzene, chlorobenzene, ethylbenzene, MTBE, vinyl chloride and xylenes, they are not COPCs for the ingestion and dermal exposure pathways.

(7) EPC derived using Halls Bootstrap method.

TABLE 2A

AVERAGE VOC EMISSIONS FROM EXPOSED SOIL DUE TO EXCAVATION OF SITE SOILS (EXCLUDING LAGOON 6)
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

AVERAGE DIFFUSION EMISSIONS

ERDIFF = (2xA) [(t^.5)/C - (B/C^2) x log(B + C(t^.5) + (B/C^2) x log(B + C(t^.5)]/t (grams/sec)

WHERE:
A= Ci x 10000 x SA
B = Ea / (Keq * Kg)
C = (3.14159 / (De * Keq)) ^ (1/2)
SA = Emitting Surface Area= 290 (m2)
t = Time of emission= 28800 (sec)
Ea = air - filled porosity= 0.55
De = effective diffusivity = Da x Ea^(4/3) (cm2/sec)

Keq = equilibrium coefficient = H/(R*T)
H = Henry's law constant (mmHg.cm3/mol)
R = gas constant (mmHg.cm3/mol.K)
T = temperature (K)

Kg = gas phase mass transfer coefficient
Kg = (.482) x (U)^.78 x (Sgc)^-.67 x (de)^-.12
U = wind speed = 4 (m/s)
Sgc = viscosity of air/(density of air * Da)
de = effective diameter = 1.27*SA (m2)

	Bulk Conc. (1) (g/g)	*Conc. Ci (2) (g/cm^3)*	*Molecular Weight (3) (g/g-mole)*	*Vapor Pressure (4) (mm Hg)*	*Henry's Law Constant (5) ($atm.m^3/mol$)*	*Equilibrium Coefficient Keq (6)*	*Diffusivity in Air Da (7) (cm^2/s)*	*Effective Diffusivity De (cm^2/s)*	*Kg*	*A*	*B*	*C*	*ERDIFF 8-hour Average (g/s)*
Volatile Organic Compounds													
Acetone	5.13E-07	7.70E-07	58.0	230.7	3.66E-05	1.50E-03	0.124	0.0434	0.6507	2.23	564.71	220.02	1.195E-04
Carbon disulfide	2.70E-08	4.05E-08	76.1	366.0	1.92E-02	7.83E-01	0.104	0.0364	0.5784	0.12	1.21	10.50	1.318E-04
1,2-Dichloroethene (total)	4.00E-09	6.00E-09	97.0	200.0	1.55E-02	6.34E-01	0.0736	0.0257	0.4588	0.02	1.89	13.88	1.478E-05
1,2-Dichloroethane	7.00E-09	1.05E-08	99.0	80.0	1.18E-03	4.81E-02	0.104	0.0364	0.5784	0.03	19.76	42.37	8.470E-06
2-Butanone	5.03E-08	7.55E-08	72.1	100.0	1.30E-04	5.31E-03	0.0808	0.0283	0.4884	0.22	211.90	144.64	1.783E-05
1,2-Dichloropropane	2.00E-09	3.00E-09	113.0	40.0	2.86E-03	1.17E-01	0.0782	0.0273	0.4778	0.01	9.84	31.33	3.273E-06
Trichloroethene	1.30E-08	1.95E-08	131.4	75.0	1.02E-02	4.17E-01	0.079	0.0276	0.4810	0.06	2.74	16.51	4.036E-05
Benzene	4.44E-06	6.65E-06	78.1	95.2	5.55E-03	2.27E-01	0.088	0.0308	0.5171	19.30	4.69	21.21	1.072E-02
4-Methyl-2-pentanone	2.24E-07	3.36E-07	100.2	15.7	3.90E-04	1.60E-02	0.075	0.0262	0.4646	0.97	74.21	86.66	1.325E-04
Tetrachloroethene	5.10E-08	7.65E-08	165.8	19.0	1.77E-02	7.24E-01	0.072	0.0252	0.4521	0.22	1.68	13.13	1.991E-04
Toluene	1.01E-05	1.52E-05	92.4	30.0	6.42E-03	2.63E-01	0.087	0.0304	0.5132	44.09	4.08	19.83	2.620E-02
Chlorobenzene	1.01E-06	1.51E-06	112.6	11.8	3.76E-03	1.54E-01	0.073	0.0255	0.4563	4.38	7.84	28.28	1.823E-03
Ethylbenzene	2.29E-06	3.44E-06	106.2	10.0	7.88E-03	3.22E-01	0.075	0.0262	0.4646	9.96	3.67	19.28	6.089E-03
Styrene	1.37E-06	2.05E-06	104.2	7.3	2.60E-03	1.07E-01	0.071	0.0248	0.4478	5.94	11.53	34.47	2.032E-03
Xylenes (total)	6.96E-05	1.04E-04	106.2	8.5	5.25E-03	2.15E-01	0.0714	0.0250	0.4495	302.61	5.70	24.21	1.473E-01
TPH (mg/kg)	2.87E-03	4.31E-03	168.0	6.5	3.80E-04	1.55E-02	0.078	0.0273	0.4770	12493.20	74.20	86.09	1.710E+00
TIC Volatile Organic Compounds													
Cyclohexane	8.00E-08	1.20E-07	84.2	98.8	1.95E-01	1.00E+00	0.084	0.0293	0.5008	0.35	1.10	10.35	3.963E-04
Methyl-cyclohexane isomer	1.90E-08	2.85E-08	92.2	43.0	9.79E-01	1.00E+00	0.099	0.0345	0.5581	0.08	0.99	9.55	1.020E-04
2-Butoxy-ethanol	2.90E-08	4.35E-08	118.2	0.3	5.26E-07	2.15E-05	0.065	0.0228	0.4225	0.13	60566.43	2534.29	5.866E-07
Trimethylpentane isomer	1.50E-08	2.25E-08	114.2	40.6	3.34E+00	1.00E+00	0.073	0.0256	0.4575	0.07	1.20	11.07	6.946E-05
Propylbenzene	1.20E-05	1.80E-05	120.2	2.5	6.59E-03	2.70E-01	0.065	0.0227	0.4221	52.20	4.83	22.65	2.716E-02
Methyl-pentene isomer	1.20E-08	1.80E-08	100.0	35.0	1.00E-04	4.09E-03	0.100	0.0350	0.5634	0.05	238.73	148.22	4.150E-06
Ethyl-cyclopentane	1.00E-08	1.50E-08	100.0	35.0	1.00E-04	4.09E-03	0.100	0.0350	0.5634	0.04	238.73	148.22	3.459E-06

Notes: (1) Based on average concentrations.
(2) Based on soil bulk density of 1.5 g/cm3.
(3) Default value for molecular weight is 100.0 g/gmol.
(4) Default value for vapor pressure is 35.0 mmHg.
(5) Default value for Henry's Law constant is 1.00E-04 atm m3/mol.
(6) Equilibrium coefficient must be less than or equal to one.
(7) Default value for diffusivity in air is 0.10 cm2/sec.

TABLE 2A

AVERAGE VOC EMISSIONS FROM EXPOSED SOIL DUE TO EXCAVATION OF SITE SOILS (EXCLUDING LAGOON 6)
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

INPUT VARIABLES	DEFINITION	UNITS	DEFAULT VALUE
P	vapor pressure of compound	mm Hg	35
MW	molecular weight	g/g-mol	100
R	gas constant	mm Hg-cm3/g-mol K	62361
T	temperature	Kelvin	298
Ea	air-filled porosity	dimensionless	0.55
Sv	volume of soil moved	m3	150
Q	excavation rate	m3/sec	0.042
1E06	conversion factor	cm2/m2	----
EXC	soil gas to atmosphere exchange constant	dimensionless	0.33
C	concentration in soil	g/cm3	1.35E-04
1E04	conversion factor	cm2/m2	----
SA	emitting surface area	m2	290
Keq	equilibrium coefficient	dimensionless	0.613
Kg	gas-phase mass transfer coefficient	cm/sec	0.15
PI	PI	dimensionless	3.14
t	time	sec	60
De	effective diffusivity in air	cm2/sec	0.0269
0.98	conversion factor	g/mm Hg-m3	----
1.22E+06	conversion factor	cm2-sec-mmHg/g	----
1.79E+09	conversion factor	sec2-cm-mmHg/g	----
M	total mass of contaminant	g	----
Cg	concentration in soil	ug/g	100
Tsv	time to excavate a given volume of soil	sec	----
B	bulk density	g/cm3	1.5
p	particle density	g/cm3	2.65
Da	diffusivity in air	cm2/sec	0.1
U	wind speed	m/s	4
ua	viscosity of air	g/cm-sec	1.81E-04
pa	density of air	g/cm3	0.0012
de	diameter of excavation	m	24

TABLE 2B

AVERAGE VOC EMISSIONS FROM EXPOSED SOIL DUE TO EXCAVATION OF LAGOON 6
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

AVERAGE DIFFUSION EMISSIONS

ERDIFF = (2xA) [(t^.5)/C - (B/C^2) x log(B + C(t^.5) + (B/C^2) x log(B + C(t^.5)]/t (grams/sec)

WHERE:
A= Ci x 10000 x SA
B = Ea / (Keq * Kg)
C = (3.14159 / (De * Keq)) ^ (1/2)
SA = Emitting Surface Area= 290 (m2)
t = Time of emission= 28800 (sec)
Ea = air - filled porosity= 0.55
De = effective diffusivity = Da x Ea^(4/3) (cm2/sec)

Keq = equilibrium coefficient = H/(R*T)
H = Henry's law constant (mmHg.cm3/mol)
R = gas constant (mmHg.cm3/mol.K)
T = temperature (K)

Kg = gas phase mass transfer coefficient
Kg = (.482) x (U)^.78 x (Sgc)^-.67 x (de)^-.12
U = wind speed = 4 (m/s)
Sgc = viscosity of air/(density of air * Da)
de = effective diameter = 1.27*SA (m2)

	Bulk Conc. (1) (g/g)	*Conc. Ci (2) (g/cm^3)*	*Molecular Weight (3) (g/g-mole)*	*Vapor Pressure (4) (mm Hg)*	*Henry's Law Constant (5) ($atm.m^3/mol$)*	*Equilibrium Coefficient Keq (6)*	*Diffusivity in Air Da (7) (cm^2/s)*	*Effective Diffusivity De (cm^2/s)*	*Kg*	*A*	*B*	*C*	*ERDIFF 8-hour Average (g/s)*
Volatile Organic Compounds													
Acetone	8.00E-09	1.20E-08	58.0	230.7	3.66E-05	1.50E-03	0.124	0.0434	0.6507	0.03	564.71	220.02	1.864E-06
Tetrachloroethene	4.00E-09	6.00E-09	165.8	19.0	1.77E-02	7.24E-01	0.072	0.0252	0.4521	0.02	1.68	13.13	1.562E-05
Toluene	1.80E-06	2.70E-06	92.4	30.0	6.42E-03	2.63E-01	0.087	0.0304	0.5132	7.83	4.08	19.83	4.653E-03
TPH (mg/kg)	3.00E-04	4.50E-04	168.0	6.5	3.80E-04	1.55E-02	0.078	0.0273	0.4770	1305.00	74.20	86.09	1.786E-01

Notes: (1) Based on average concentrations.
(2) Based on soil bulk density of 1.5 g/cm3.
(3) Default value for molecular weight is 100.0 g/gmol.
(4) Default value for vapor pressure is 35.0 mmHg.
(5) Default value for Henry's Law constant is 1.00E-04 atm m3/mol.
(6) Equilibrium coefficient must be less than or equal to one.
(7) Default value for diffusivity in air is 0.10 cm2/sec.

INPUT VARIABLES	DEFINITION	UNITS	DEFAULT VALUE
P	vapor pressure of compound	mm Hg	35
MW	molecular weight	g/g-mol	100
R	gas constant	mm Hg-cm3/g-mol K	62361
T	temperature	Kelvin	298
Ea	air-filled porosity	dimensionless	0.55
Sv	volume of soil moved	m3	150
Q	excavation rate	m3/sec	0.042
1E06	conversion factor	cm2/m2	----
EXC	soil gas to atmosphere exchange constant	dimensionless	0.33
C	concentration in soil	g/cm3	1.35E-04
1E04	conversion factor	cm2/m2	----
SA	emitting surface area	m2	290
Keq	equilibrium coefficient	dimensionless	0.613
Kg	gas-phase mass transfer coefficient	cm/sec	0.15
PI	PI	dimensionless	3.14
t	time	sec	60
De	effective diffusivity in air	cm2/sec	0.0269
0.98	conversion factor	g/mm Hg-m3	----
1.22E+06	conversion factor	cm2-sec-mmHg/g	----
1.79E+09	conversion factor	sec2-cm-mmHg/g	----
M	total mass of contaminant	g	----
Cg	concentration in soil	ug/g	100
Tsv	time to excavate a given volume of soil	sec	----
B	bulk density	g/cm3	1.5
p	particle density	g/cm3	2.65
Da	diffusivity in air	cm2/sec	0.1
U	wind speed	m/s	4
ua	viscosity of air	g/cm-sec	1.81E-04
pa	density of air	g/cm3	0.0012
de	diameter of excavation	m	24

TABLE 3A

VOLATILE COMPOUND EMISSIONS FROM SOIL
AIR/SUPERFUND NATIONAL TECHNICAL GUIDANCE STUDY SERIES
ESTIMATION OF AIR IMPACTS FOR THE EXCAVATION OF CONTAMINATED SOIL
FORMER LAGOON SITE - HAMPTONBURGH, NEW YORK

ER=EMISSION RATE FROM EXCAVATION = PORE SPACE EMISSIONS + DIFFUSION EMISSIONS

PORE SPACE EMISSIONS ERPS= (P x MW x 1E06 x Ea x Q x EXC)/(R x T) (grams/second)

DIFFUSION EMISSIONS ERDIFF= (C x 1E04 x SA)/ (Ea/(Keq x Kg) + (PI x t)/(De x Keq)**1/2) (grams/second)

COMPOUND SPECIFIC EMISSIONS

Excavation Rate= 0.042 (m3/sec)
Emitting Surface Area= 290 (m2)
Time of emission= 7130 (sec)

Volatile Organic Compounds	*Concentration* (g/cm^3)	*Molecular Weight* (g/g-mole)	*Vapor Pressure* (mm Hg)	*Equilibrium Coefficient Keq*	*Diffusivity in Air Da* (cm^2/s)	*Effective Diffusivity De* (cm^2/s)	*Kg*	*ERPS* (g/s)	*ERDIFF* (g/s)	*ER* (g/s)
Acetone	7.70E-07	58	230.7	1.50E-03	0.124	0.0434	0.6507	1.08E-02	1.17E-04	1.09E-02
Carbon disulfide	4.05E-08	76.1	366.0	7.83E-01	0.104	0.0364	0.5784	5.67E-04	1.32E-04	6.99E-04
1,2-Dichloroethene (total)	6.00E-09	96.95	200.0	6.34E-01	0.0736	0.0257	0.4588	8.40E-05	1.48E-05	9.88E-05
1,2-Dichloroethane	1.05E-08	99	80.0	4.81E-02	0.104	0.0364	0.5784	1.47E-04	8.47E-06	1.55E-04
2-Butanone	7.55E-08	72.1	100.0	5.31E-03	0.0808	0.0283	0.4884	1.06E-03	1.76E-05	1.07E-03
1,2-Dichloropropane	3.00E-09	113	40.0	1.17E-01	0.0782	0.0273	0.4778	4.20E-05	3.28E-06	4.53E-05
Trichloroethene	1.95E-08	131.4	75.0	4.17E-01	0.079	0.0276	0.4810	2.73E-04	4.05E-05	3.13E-04
Benzene	6.65E-06	78.1	95.2	2.27E-01	0.088	0.0308	0.5171	9.32E-02	1.07E-02	1.04E-01
4-Methyl-2-pentanone	3.36E-07	100.2	15.7	1.60E-02	0.075	0.0262	0.4646	4.70E-03	1.32E-04	4.84E-03
Tetrachloroethene	7.65E-08	165.83	19.0	7.24E-01	0.072	0.0252	0.4521	1.07E-03	2.00E-04	1.27E-03
Toluene	1.52E-05	92.4	30.0	2.63E-01	0.087	0.0304	0.5132	2.13E-01	2.63E-02	2.39E-01
Chlorobenzene	1.51E-06	112.6	11.8	1.54E-01	0.073	0.0255	0.4563	2.11E-02	1.83E-03	2.30E-02
Ethylbenzene	3.44E-06	106.2	10.0	3.22E-01	0.075	0.0262	0.4646	4.81E-02	6.11E-03	5.42E-02
Styrene	2.05E-06	104.2	7.3	1.07E-01	0.071	0.0248	0.4478	2.87E-02	2.03E-03	3.07E-02
Xylenes (total)	1.04E-04	106.2	8.5	2.15E-01	0.0714	0.0250	0.4495	3.70E-01	1.48E-01	5.18E-01
TPH (mg/kg)	4.31E-03	168.0	6.5	1.55E-02	0.08	0.0273	0.4770	4.48E-01	1.70E+00	2.15E+00
TIC Volatile Organics										
Cyclohexane	1.20E-07	84.2	98.8	1.00E+00	0.08	0.0293	0.5008	1.68E-03	3.98E-04	2.08E-03
Methyl-cyclohexane isomer	2.85E-08	92.2	43.0	1.00E+00	0.10	0.0345	0.5581	3.99E-04	1.02E-04	5.01E-04
2-Butoxy-ethanol	4.35E-08	118.18	0.34	2.15E-05	0.07	0.0228	0.4225	6.09E-04	4.60E-07	6.09E-04
Trimethylpentane isomer	2.25E-08	114.22	40.6	1.00E+00	0.07	0.0256	0.4575	3.15E-04	6.97E-05	3.85E-04
Propylbenzene	1.80E-05	120.2	2.5	2.70E-01	0.07	0.0227	0.4221	1.23E-01	2.72E-02	1.50E-01
Methyl-pentene isomer	1.80E-08	100	35.0	4.09E-03	0.10	0.0350	0.5634	2.52E-04	4.09E-06	2.56E-04
Ethyl-cyclopentane	1.50E-08	100	35.0	4.09E-03	0.10	0.0350	0.5634	2.10E-04	3.41E-06	2.13E-04
							Total VOC Emissions	**1.3676**	**1.92**	**3.29**

Note:

All assumptions and calculated parameters from Table 1 are valid for Table 2.

TABLE 3B

VOLATILE COMPOUND EMISSIONS FROM SOIL
AIR/SUPERFUND NATIONAL TECHNICAL GUIDANCE STUDY SERIES
ESTIMATION OF AIR IMPACTS FOR THE EXCAVATION OF CONTAMINATED SOIL FROM LAGOON 6
FORMER LAGOON SITE - HAMPTONBURGH, NEW YORK

ER=EMISSION RATE FROM EXCAVATION = PORE SPACE EMISSIONS + DIFFUSION EMISSIONS

PORE SPACE EMISSIONS ERPS= (P x MW x 1E06 x Ea x Q x EXC)/(R x T) (grams/second)

DIFFUSION EMISSIONS ERDIFF= (C x 1E04 x SA)/ (Ea/(Keq x Kg) + (PI x t)/(De x Keq)**1/2) (grams/second)

COMPOUND SPECIFIC EMISSIONS

Excavation Rate= 0.042 (m3/sec)
Emitting Surface Area= 290 (m2)
Time of emission= 7130 (sec)

Volatile Organic Compounds	*Concentration* (g/cm^3)	*Molecular Weight* (g/g-mole)	*Vapor Pressure* (mm Hg)	*Equilibrium Coefficient Keq*	*Diffusivity in Air Da* (cm^2/s)	*Effective Diffusivity De* (cm^2/s)	*Kg*	*ERPS* (g/s)	*ERDIFF* (g/s)	*ER* (g/s)
Acetone	1.20E-08	58	230.7	1.50E-03	0.124	0.0434	0.6507	1.68E-04	1.82E-06	1.70E-04
Tetrachloroethene	6.00E-09	165.83	19.0	7.24E-01	0.072	0.0252	0.4521	8.40E-05	1.57E-05	9.97E-05
Toluene	2.70E-06	92.4	30.0	2.63E-01	0.087	0.0304	0.5132	3.78E-02	4.67E-03	4.25E-02
TPH (mg/kg)	4.50E-04	168.0	6.5	1.55E-02	0.08	0.0273	0.4770	4.48E-01	1.78E-01	6.26E-01
							Total VOC Emissions	**0.4860**	**0.18**	**0.67**

Note:
All assumptions and calculated parameters from Table 1 are valid for Table 2.

TABLE 4A

ESTIMATED MAXIMUM GROUND LEVEL VOC CONCENTRATIONS - DURING EXCAVATION
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

Volatile Organic Compound	*Estimated Maximum Emission Rate Due To Undisturbed Soil (g/s)*	*Estimated Maximum Ground Level Concentrations*		*ACGIH TLV*
		1-hour ($\mu g/m^3$)	*8-hour ($\mu g/m^3$)*	*8-hour ($\mu g/m^3$)*
Acetone	1.09E-02	1.17E+03	8.16E+02	1.19E+06
Carbon disulfide	6.99E-04	7.48E+01	5.24E+01	3.10E+04
1,2-Dichloroethene (total)	9.88E-05	1.06E+01	7.40E+00	7.93E+05
1,2-Dichloroethane	1.55E-04	1.66E+01	1.16E+01	4.00E+04
2-Butanone	1.07E-03	1.15E+02	8.04E+01	5.90E+05
1,2-Dichloropropane	4.53E-05	4.84E+00	3.39E+00	3.47E+05
Trichloroethene	3.13E-04	3.35E+01	2.35E+01	2.69E+05
Benzene	1.04E-01	1.11E+04	7.78E+03	1.60E+03
4-Methyl-2-pentanone	4.84E-03	5.17E+02	3.62E+02	2.05E+05
Tetrachloroethene	1.27E-03	1.36E+02	9.52E+01	1.70E+05
Toluene	2.39E-01	2.56E+04	1.79E+04	1.88E+05
Chlorobenzene	2.30E-02	2.46E+03	1.72E+03	4.60E+04
Ethylbenzene	5.42E-02	5.80E+03	4.06E+03	4.34E+05
Styrene	3.07E-02	3.29E+03	2.30E+03	8.50E+04
Xylenes (total)	5.18E-01	5.54E+04	3.88E+04	4.34E+05
TPH (mg/kg)	2.15E+00	2.30E+05	1.61E+05	NA
TIC Volatile Organics				
Cyclohexane	2.08E-03	2.22E+02	1.56E+02	1.03E+06
Methyl-cyclohexane isomer	5.01E-04	5.37E+01	3.76E+01	1.61E+06
2-Butoxy-ethanol	6.09E-04	6.52E+01	4.56E+01	1.21E+05
Trimethylpentane isomer	3.85E-04	4.12E+01	2.88E+01	NA
Propylbenzene	1.50E-01	1.61E+04	1.13E+04	NA
Methyl-pentene isomer	2.56E-04	2.74E+01	1.92E+01	NA
Ethyl-cyclopentane	1.12E-03	1.20E+02	8.40E+01	NA

Notes:
ACGIH - American Conference of Governmental Industrial Hygienists.
TLV - Threshold Limit Value.
NA - Threshold Limit Value (TLV) Not Available.

TABLE 4B

ESTIMATED MAXIMUM GROUND LEVEL VOC CONCENTRATIONS - DURING EXCAVATION OF LAGOON 6
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

Volatile Organic Compound	*Estimated Maximum Emission Rate Due To Undisturbed Soil (g/s)*	*Estimated Maximum Ground Level Concentrations*		*ACGIH TLV*
		1-hour ($\mu g/m^3$)	*8-hour ($\mu g/m^3$)*	*8-hour ($\mu g/m^3$)*
Acetone	1.70E-04	1.82E+01	1.27E+01	1.19E+06
Tetrachloroethene	9.97E-05	1.07E+01	7.47E+00	1.70E+05
Toluene	4.25E-02	4.54E+03	3.18E+03	1.88E+05
TPH (mg/kg)	6.26E-01	6.69E+04	4.69E+04	NA

Notes:
ACGIH - American Conference of Governmental Industrial Hygienists.
TLV - Threshold Limit Value.
NA - Threshold Limit Value (TLV) Not Available.

TABLE 5A

PARTICULATE, METALS, PCB AND SVOC EMISSIONS DURING EXCAVATION AND ESTIMATED MAXIMUM GROUND LEVEL CONCENTRATIONS
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

	Bulk Conc. mg/kg	*Emission Rate (1) (g/s)*	*Estimated Maximum Ground Level Concentration*		*ACGIH TLV*
			1-Hour ($\mu g/m^3$)	*8-Hour ($\mu g/m^3$)*	*8-hour ($\mu g/m^3$)*
Inorganics					
Aluminum	3.14E+04	2.86E-04	3.06E+01	2.15E+01	1.00E+04
Antimony	1.80E+00	1.64E-08	1.76E-03	1.23E-03	5.00E+02
Arsenic	1.13E+01	1.03E-07	1.11E-02	7.74E-03	1.00E+01
Cadmium	9.13E-01	8.33E-09	8.92E-04	6.24E-04	1.00E+01
Chromium	3.04E+01	2.77E-07	2.97E-02	2.08E-02	5.00E+02
Copper	2.18E+02	1.99E-06	2.13E-01	1.49E-01	1.00E+03
Iron	2.80E+04	2.56E-04	2.73E+01	1.91E+01	NA
Manganese	9.30E+02	8.49E-06	9.08E-01	6.36E-01	2.00E+02
Mercury	4.67E+00	4.26E-08	4.56E-03	3.19E-03	2.50E+01
Thallium	7.58E-01	6.92E-09	7.40E-04	5.18E-04	1.00E+02
Vanadium	3.17E+01	2.90E-07	3.10E-02	2.17E-02	NA
Pesticides/PCBs					
ALDRIN	1.63E-02	1.49E-10	1.59E-05	1.11E-05	2.50E+02
DIELDRIN	3.41E-02	3.11E-10	3.33E-05	2.33E-05	2.50E+02
Aroclor-1254	2.59E+00	2.36E-08	2.52E-03	1.77E-03	NA
Arochlor-1260	7.33E-01	6.69E-09	7.16E-04	5.01E-04	NA
Semi Volatile Organics					
4-Chloro-3-Methylphenol	4.20E-02	3.83E-10	4.10E-05	2.87E-05	NA
Bis(2-Ethylhexyl)phthalate	1.66E+01	1.51E-07	1.62E-02	1.13E-02	NA
Benzo(a)pyrene	2.00E-01	1.83E-09	1.95E-04	1.37E-04	NA
Alpha-picoline	2.53E+00	2.31E-08	2.47E-03	1.73E-03	NA
2-Aminopyridine	2.34E+01	2.13E-07	2.28E-02	1.60E-02	1.90E+03
TIC Semi-Volatile Organics					
1,4-Benzenediamine	4.00E-01	3.65E-09	3.91E-04	2.73E-04	NA
1H-indazole, 4,5,6,7-tetrahydro	2.00E-01	1.83E-09	1.95E-04	1.37E-04	NA
1-Nitroethyl-benzene	3.00E-02	2.74E-10	2.93E-05	2.05E-05	NA
2-Chloroethyl benzene	1.00E+01	9.13E-08	9.77E-03	6.84E-03	NA
2-Chloro-5-(trifluoro)-benzeneamine	1.10E+01	1.00E-07	1.07E-02	7.52E-03	NA
2,2'-Methylenedithiophene	1.50E+01	1.37E-07	1.46E-02	1.03E-02	NA
2,4-Bipyridyl	2.00E-01	1.83E-09	1.95E-04	1.37E-04	NA
3-Methyl-2-pyridinamine	3.00E-01	2.74E-09	2.93E-04	2.05E-04	NA
3,5-Dimethyl-1-phenypyrazole	5.00E-01	4.56E-09	4.88E-04	3.42E-04	NA
5-Ethyl-2-methyl-Pyridine	2.01E+00	1.83E-08	1.96E-03	1.37E-03	NA
5-Methyl-2-pyridinamine	3.00E-01	2.74E-09	2.93E-04	2.05E-04	NA
6-Methyl-2-pyridinamine	2.00E-01	1.83E-09	1.95E-04	1.37E-04	NA

TABLE 5A

PARTICULATE, METALS, PCB AND SVOC EMISSIONS DURING EXCAVATION AND ESTIMATED MAXIMUM GROUND LEVEL CONCENTRATIONS
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

	Bulk Conc. mg/kg	*Emission Rate (1) (g/s)*	*Estimated Maximum Ground Level Concentration*		*ACGIH TLV*
			1-Hour ($\mu g/m^3$)	*8-Hour* ($\mu g/m^3$)	*8-hour* ($\mu g/m^3$)
Acetophenone	7.60E+00	6.94E-08	7.42E-03	5.20E-03	4.91E+04
Benzaldehyde	5.80E-01	5.29E-09	5.66E-04	3.96E-04	NA
Benzamide	8.20E+00	7.48E-08	8.01E-03	5.61E-03	NA
Benzene, 1,1'-oxybis-	3.00E-01	2.74E-09	2.93E-04	2.05E-04	NA
Benzene, 1-chloro-4(1-methyl	8.00E-01	7.30E-09	7.81E-04	5.47E-04	NA
Bicyclo(3.1.1)hept-2-ene, 3,	2.00E-01	1.83E-09	1.95E-04	1.37E-04	NA
Bipyridine Isomer	5.79E+00	5.29E-08	5.66E-03	3.96E-03	NA
Bromohexane isomer	4.00E-01	3.65E-09	3.91E-04	2.73E-04	NA
Chlorothioxanthenone isomer 1	2.00E-01	1.83E-09	1.95E-04	1.37E-04	NA
Chlorothioxanthenone isomer 2	1.00E-01	9.13E-10	9.77E-05	6.84E-05	NA
Diisopropylether	4.00E-02	3.65E-10	3.91E-05	2.73E-05	NA
Diethylbenzeneamine isomer	2.00E+00	1.83E-08	1.95E-03	1.37E-03	NA
Dimethylbenzeneamine isomer	3.00E-01	2.74E-09	2.93E-04	2.05E-04	NA
Diphenyl ether	1.00E+00	9.13E-09	9.77E-04	6.84E-04	NA
Ethyl methyl benzene isomer	2.00E-02	1.83E-10	1.95E-05	1.37E-05	NA
Ethylmethylpyridine isomer	2.88E+00	2.63E-08	2.82E-03	1.97E-03	NA
Ethenyl pyridine isomer	6.00E+00	5.48E-08	5.86E-03	4.10E-03	NA
Ethyl pyridine isomer	7.00E-01	6.39E-09	6.84E-04	4.79E-04	NA
Methyl-pyridinamine isomer	2.00E-01	1.83E-09	1.95E-04	1.37E-04	NA
Methyl pyridine isomer	1.00E-01	9.13E-10	9.77E-05	6.84E-05	NA
Naphthalene, 1,2,3,4-tetrahydro	4.40E+01	4.02E-07	4.30E-02	3.01E-02	NA
Nonane	1.00E-02	9.13E-11	9.77E-06	6.84E-06	1.05E+06
Phenothiazine	1.30E+00	1.19E-08	1.27E-03	8.89E-04	5.00E+03
Sulfur, mol (S8)	2.00E+00	1.83E-08	1.95E-03	1.37E-03	NA

Notes:

(1) The PM-10 emission factor (EF) calculations are attached.
(2) The emission rate for metals/PCBs is based on the metal/PCB concentration in the bulk soil, an enrichment factor, and the calculated PM-10 emission factor.
(3) American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value (TLV).

TABLE 5A

PARTICULATE, METALS, PCB AND SVOC EMISSIONS DURING EXCAVATION AND ESTIMATED MAXIMUM GROUND LEVEL CONCENTRATIONS
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

The PM-10 emission factor calculated using the USEPA document "Estimation of Air Impacts from Area Sources of Particulate Matter Emissions at Superfund Sites".

EF = (k (0.0016) M (U/2.2)^(1.3))/((XH2O/2)^(1.4))

Where:	EF = PM emissions (g/s)	
	k = particle size multiplier for PM10 =	0.35
	0.0016 = empirical constant (g/Kg)	0.0016
	M = mass of waste handled (Kg/s) =	63
	U = mean wind speed (m/s) =	4.4
	2.2 = empirical constant (m/s) =	2.2
	XH2O = percent moisture content (%) =	10

EF = 9.13E-03 g/s

The SVOC/pesticide/metal/PCB emission rates were calculated as follows:

ER = C*Z*EF/10^6

Where:
ER = emission rate of contaminant (g/s)
C = concentration of metal/PCB in the bulk soil (ug/g)
Z = enrichment factor

TABLE 5B

PARTICULATE, METALS, PCB AND SVOC EMISSIONS DURING EXCAVATION OF LAGOON 6 AND ESTIMATED MAXIMUM GROUND LEVEL CONCENTRATIONS
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

	Bulk Conc. mg/kg	*Emission Rate (1) (g/s)*	*Estimated Maximum Ground Level Concentration*		*ACGIH TLV*
			1-Hour ($\mu g/m^3$)	*8-Hour ($\mu g/m^3$)*	*8-hour ($\mu g/m^3$)*
TIC Semi-Volatile Organics					
1,2-Propanedione, 1-phenyl	2.00	1.83E-08	1.95E-03	1.37E-03	NA
1-Propanone, 1-(3-pyridinyl)	20.00	1.83E-07	1.95E-02	1.37E-02	NA
2-Butyl pyridine	1.00	9.13E-09	9.77E-04	6.84E-04	NA
2-Ethenyl pyridine	7.00	6.39E-08	6.84E-03	4.79E-03	NA
Dichlorobiphenyl isomer	20.00	1.83E-07	1.95E-02	1.37E-02	NA
Methyl ester benzoic acid	5.00	4.56E-08	4.88E-03	3.42E-03	NA
Methyl phenanthrene isomer	0.50	4.56E-09	4.88E-04	3.42E-04	NA
Pesticides/PCBs					
ALDRIN	0.19	1.73E-09	1.86E-04	1.30E-04	2.50E+02
Aroclor-1254	9.20	8.40E-08	8.98E-03	6.29E-03	NA
Inorganics					
Aluminum	19600.00	1.79E-04	1.91E+01	1.34E+01	1.00E+04
Arsenic	47.60	1.30E-06	1.39E-01	9.76E-02	1.00E+01
Cadmium	4.40	4.02E-08	4.30E-03	3.01E-03	1.00E+01
Chromium	60.70	5.54E-07	5.93E-02	4.15E-02	5.00E+02
Iron	37445.00	3.42E-04	3.66E+01	2.56E+01	NA
Manganese	699.00	6.38E-06	6.83E-01	4.78E-01	2.00E+02
Mercury	23.40	2.14E-07	2.29E-02	1.60E-02	2.50E+01
Thallium	1.61	1.47E-08	1.57E-03	1.10E-03	1.00E+02
Vanadium	26.80	2.45E-07	2.62E-02	1.83E-02	NA

Notes:
(1) The PM-10 emission factor (EF) calculations are attached.
(2) The emission rate for metals/PCBs is based on the metal/PCB concentration in the bulk soil, an enrichment factor, and the calculated PM-10 emission factor.
(3) American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value (TLV).

The PM-10 emission factor calculated using the USEPA document "Estimation of Air Impacts from Area Sources of Particulate Matter Emissions at Superfund Sites".

EF = (k (0.0016) M (U/2.2)^(1.3))/((XH2O/2)^(1.4))

Where: EF = PM emissions (g/s)

k = particle size multiplier for PM10 =	0.35
0.0016 = empirical constant (g/Kg)	0.0016
M = mass of waste handled (Kg/s) =	63
U = mean wind speed (m/s) =	4.4
2.2 = empirical constant (m/s) =	2.2
XH2O = percent moisture content (%) =	10

EF = 9.13E-03 g/s

The SVOC/pesticide/metal/PCB emission rates were calculated as follows:

ER = C*Z*EF/10^6

Where:
ER = emission rate of contaminant (g/s)
C = concentration of metal/PCB in the bulk soil (ug/g)
Z = enrichment factor

TABLE 6A

RTI LAGOON MODEL
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

Lagoon Surface Area =	1.00E+06	cm^2
Temperature =	20 °C	
Ideal Gas Constant =	8.20E-05	atm m^3/mol K

	Concentration Max (ug/L) (1)	*Molecular Weight (g/mol)*	*Liquid Phase Mass Transfer Coefficient (cm/s)*	*Gas Phase Mass Transfer Coefficient (cm/s)*	*Henry's Law Constant (atm m^3/mol)*	*Overall Mass Transfer Coefficient (cm/s)*	*Emission Rate (g/s)*
VOCs							
1,1-Dichloroethane	9.00E-01	98.96	1.12E-03	4.63E-01	5.62E-03	1.11E-03	9.97E-07
2-Butanone	2.80E+01	72.10	1.31E-03	5.15E-01	1.30E-04	8.91E-04	2.50E-05
4-Methyl-2-pentanone	6.40E+00	100.20	1.11E-03	4.61E-01	2.40E-04	8.96E-04	5.73E-06
Acetone	2.90E+02	58.00	1.46E-03	5.54E-01	6.42E-03	1.45E-03	4.20E-04
Benzene	3.32E+02	78.11	1.26E-03	5.01E-01	5.55E-03	1.25E-03	4.14E-04
Carbon disulfide	1.18E+00	76.10	1.28E-03	5.06E-01	1.32E-02	1.27E-03	1.50E-06
Chlorobenzene	2.67E+01	112.60	1.05E-03	4.43E-01	3.76E-03	1.03E-03	2.76E-05
cis-1,2-Dichloroethene	8.00E-01	96.95	1.13E-03	4.66E-01	4.17E-03	1.11E-03	8.92E-07
Ethylbenzene	1.23E+02	106.20	1.08E-03	4.52E-01	7.88E-03	1.07E-03	1.32E-04
Methyl Tert Butyl Ether	6.16E+00	83.10	1.22E-03	4.91E-01	6.30E-04	1.12E-03	6.87E-06
Toluene	8.93E+00	92.40	1.16E-03	4.74E-01	6.42E-03	1.15E-03	1.03E-05
Vinyl chloride	1.01E+00	62.50	1.41E-03	5.40E-01	2.21E-02	1.40E-03	1.41E-06
Xylene (total)	2.73E+02	106.20	1.08E-03	4.52E-01	7.01E-03	1.07E-03	2.92E-04
VOC TICs							
Diisopropyl ether	7.07E+00	102.17	1.10E-03	4.58E-01	2.04E-03	1.07E-03	7.57E-06
n-Butyl ether	9.00E+00	130.22	9.75E-04	4.22E-01	5.88E-03	9.66E-04	8.70E-06
2-Ethyl-1-hexanol	5.00E+00	130.22	9.75E-04	4.22E-01	6.17E-05	5.13E-04	2.57E-06
Tetrahydrofuran	6.00E+00	72.12	1.31E-03	5.15E-01	1.05E-05	1.92E-04	1.15E-06
Dimethyl disulfide	3.00E+01	94.20	1.15E-03	4.71E-01	1.04E-03	1.09E-03	3.26E-05
Methanethiol	1.00E+01	48.10	1.60E-03	5.89E-01	3.16E-03	1.57E-03	1.57E-05
1,2,3,4-Tetrahydronaphthalene	1.50E+01	132.20	9.68E-04	4.20E-01	1.89E-03	9.40E-04	1.41E-05

Notes:
(1) From Table 1C.

TABLE 6B

ESTIMATED MAXIMUM GROUND LEVEL VOC CONCENTRATIONS
DUE TO EMISSIONS FROM EXPOSED GROUNDWATER
FORMER LAGOON SITE
HAMPTONBURGH, NEW YORK

	Estimated Maximum Emission Rate (1)	*Estimated Maximum Ground Level Concentration*		*ACGIH TLV (2)*
		1-hour	*8-hour*	*8-hour*
	(g/s)	*($\mu g/m^3$)*		*($\mu g/m^3$)*
VOCs				
1,1-Dichloroethane	9.97E-07	2.58E-01	1.81E-01	4.05E+05
2-Butanone	2.50E-05	6.46E+00	4.52E+00	5.90E+05
4-Methyl-2-pentanone	5.73E-06	1.48E+00	1.04E+00	2.05E+05
Acetone	4.20E-04	1.09E+02	7.61E+01	1.19E+06
Benzene	4.14E-04	1.07E+02	7.50E+01	1.60E+03
Carbon disulfide	1.50E-06	3.89E-01	2.73E-01	3.11E+04
Chlorobenzene	2.76E-05	7.15E+00	5.01E+00	4.60E+04
cis-1,2-Dichloroethene	8.92E-07	2.31E-01	1.62E-01	7.93E+05
Ethylbenzene	1.32E-04	3.42E+01	2.39E+01	4.34E+05
Methyl Tert Butyl Ether	6.87E-06	1.78E+00	1.24E+00	1.80E+05
Toluene	1.03E-05	2.65E+00	1.86E+00	1.88E+05
Vinyl chloride	1.41E-06	3.66E-01	2.56E-01	2.56E+03
Xylene (total)	2.92E-04	7.57E+01	5.30E+01	4.34E+05
VOC TICs				
Diisopropyl ether	7.57E-06	1.96E+00	1.37E+00	1.04E+06
n-Butyl ether	8.70E-06	2.25E+00	1.58E+00	NA
2-Ethyl-1-hexanol	2.57E-06	6.65E-01	4.65E-01	NA
Tetrahydrofuran	1.15E-06	2.98E-01	2.09E-01	5.90E+05
Dimethyl disulfide	3.26E-05	8.43E+00	5.90E+00	NA
Methanethiol	1.57E-05	4.07E+00	2.85E+00	9.84E+02
1,2,3,4-Tetrahydronaphthalene	1.41E-05	3.65E+00	2.56E+00	NA

Notes:
(1) From Table 6A.
(2) American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value (TLV).

1-Hour Dispersion Factor = 258,900 *($\mu g/m^3/g/s$)*

ATTACHMENT A

SCREEN3 MODEL OUTPUT

06/29/99
10:48:13

*** SCREEN3 MODEL RUN ***
*** VERSION DATED 96043 ***

June 29, 1999 #3698

SIMPLE TERRAIN INPUTS:

SOURCE TYPE	=	AREA
EMISSION RATE (G/(S-M**2))	=	.344800E-02
SOURCE HEIGHT (M)	=	.0000
LENGTH OF LARGER SIDE (M)	=	17.0300
LENGTH OF SMALLER SIDE (M)	=	17.0300
RECEPTOR HEIGHT (M)	=	.0000
URBAN/RURAL OPTION	=	URBAN

THE REGULATORY (DEFAULT) MIXING HEIGHT OPTION WAS SELECTED.
THE REGULATORY (DEFAULT) ANEMOMETER HEIGHT OF 10.0 METERS WAS ENTERED.

MODEL ESTIMATES DIRECTION TO MAX CONCENTRATION

BUOY. FLUX = .000 M**4/S**3; MOM. FLUX = .000 M**4/S**2.

*** FULL METEOROLOGY ***

****
*** SCREEN AUTOMATED DISTANCES ***
****

*** TERRAIN HEIGHT OF 0. M ABOVE STACK BASE USED FOR FOLLOWING DISTANCES ***

DIST (M)	CONC (UG/M**3)	STAB	U10M (M/S)	USTK (M/S)	MIX HT (M)	PLUME HT (M)	MAX DIR (DEG)
1.	.8545E+05	6	1.0	1.0	10000.0	.00	45.
100.	3612.	6	1.0	1.0	10000.0	.00	41.
200.	1045.	6	1.0	1.0	10000.0	.00	1.
300.	505.9	6	1.0	1.0	10000.0	.00	30.
400.	305.9	6	1.0	1.0	10000.0	.00	2.
500.	208.6	6	1.0	1.0	10000.0	.00	18.
600.	153.6	6	1.0	1.0	10000.0	.00	34.
700.	119.1	6	1.0	1.0	10000.0	.00	18.
800.	96.02	6	1.0	1.0	10000.0	.00	8.
900.	79.65	6	1.0	1.0	10000.0	.00	1.
1000.	67.51	6	1.0	1.0	10000.0	.00	5.
MAXIMUM 1-HR CONCENTRATION AT OR BEYOND 1. M:							
12.	.1070E+06	6	1.0	1.0	10000.0	.00	45.

****
*** SUMMARY OF SCREEN MODEL RESULTS ***
****

CALCULATION PROCEDURE	MAX CONC (UG/M**3)	DIST TO MAX (M)	TERRAIN HT (M)
SIMPLE TERRAIN	.1070E+06	12.	0.

APPENDIX Q.3

STATISTICAL METHODS USED TO CALCULATE EPC VALUES

TABLE OF CONTENTS

LIST OF TABLES
(Following Text)

1.0 INTRODUCTION

Two estimates of exposure were used in the risk assessment process: (i) the mean, or central tendency (CT), exposure; and (ii) the reasonable maximum exposure (RME). The CT exposure scenario uses the mean value to represent probable exposure conditions. The RME scenario generally uses a conservative 95 percent upper confidence limit (UCL) of the mean to estimate a reasonable maximum exposure. The determinations of the CT and RME estimates are statistically based and driven by characteristics of the data. Key factors determining the statistical methodologies employed include (i) the probability distribution of the observed data (e.g., normal vs. lognormal, etc.); and (ii) the degree of censored data (non-detected results) present.

The following sections present the procedures used to determine the CT and RME values of the chemicals of potential concern (COPCs) in this risk assessment. A number of guidance documents were consulted in developing the statistical methodologies including Ministry of the Environment (MOE) (1997), United States Environmental Protection Agency (USEPA) (1989), USEPA (1992) updated by USEPA (2002), USEPA (1997), USEPA (2000), and USEPA (2003).

2.0 STATISTICAL PROCEDURES

The development of COPC exposure estimates for each parameter and area of concern is a three step process consisting of (i) determining the percentage of non-detects present, (ii) data distribution testing, and (iii) selecting the appropriate statistical method for exposure estimate calculations.

The first step of the statistical evaluation was to determine the percentage of the non-detects present in each data set. Suggested approaches to account for the presence of non-detect analytical results are outlined in USEPA (2000), and USEPA (2002), and these guidelines are summarized in Table 1.

The second step of the statistical analysis to establish COPC exposure estimates was to determine the data distribution. Each data set was tested for normality and lognormality using either the Shapiro-Wilk W-test (1965) (for sample sizes up to 50) or the Shapiro-Francia W'-test (1972) (for sample sizes of 50 to 100). Additional tests of normality for larger data sets, if needed, are presented in USEPA (2000).

Methods for determining the CT and RME values are discussed in USEPA 2002 (which updates USEPA 1992), USEPA 1997 and USEPA 2003. The alternative procedures suggested are listed in Table 2. A summary of the selected statistical methods used to determine the CT and RME values, based on the observed distribution of the data and the proportion of non-detect values is given in Tables 3 and 4.

The following sections discuss the calculation procedures used to develop the CT and RME estimates. Section 2.1 deals with the statistical methods used for normally distributed data sets, Section 2.2 discusses the statistical methods used for the lognormally distributed data sets, and Section 2.3 discusses statistical methods used for non-normal data sets. Each section is organized into separate divisions to deal with the cases of a low degree of censored (non-detect) data (0 to 15 percent), moderately censored (16 to 50 percent), highly censored (51 to 75 percent), very highly censored (76 to 99 percent), and 100 percent non-detected data.

2.1 NORMAL DISTRIBUTION

2.1.1 UP TO 15 PERCENT NON-DETECTS

In order to calculate the CT and RME values, the non-detect values were replaced with one-half the reported detection limit. The arithmetic mean and standard deviation of

this substituted data set were then calculated. The calculated mean was taken as the CT value. The RME value was established by calculating the 95 percent UCL of the arithmetic mean for the normal distribution using the following equation.

$$95\%\text{UCL} = \bar{x} + t_{(0.05,n-1,1)} \bullet s / \sqrt{n}$$

Where:

$\bar{x}$ = mean of the substituted data set;

$t_{(0.05, n-1, 1)}$ = student *t*-statistic for a one-tailed 95 percent confidence (α=0.05) and n-1 degrees of freedom;

s = standard deviation of the substituted data set; and

n = number of samples.

2.1.2 NON-DETECTS GREATER THAN 15 PERCENT UP TO 50 PERCENT

In this case, the mean and standard deviation of the censored data set were adjusted using Cohen's method, as recommended in USEPA 2002. This method is presented in McBean & Rovers (1998) and USEPA (2000). Cohen's method adjusts the sample mean and sample standard deviation to account for the censored data below the detection limit as follows.

Step 1) Compute the sample mean $\bar{x}_d$ using detected data only.

Step 2) Compute the sample variance s_d^2 using detected data only.

Step 3) Compute the two parameters *h* (proportion of non-detects) and γ as:

$$h = \frac{n - m}{n} \qquad \gamma = \frac{s_d^2}{(\bar{x} - DL)^2}$$

where *m* is the number of detected data points, *n* is the total number of samples and DL is the detection limit.

Step 4) Determine the value of the parameter $\hat{\lambda}$ from the Table 5 based on h and γ.

Step 5) Estimate the corrected sample mean ($\bar{x}$) and standard deviation (s) as:

$$\bar{x} = \bar{x}_d - \hat{\lambda}(\bar{x}_d - DL) \quad \text{and} \quad s = \sqrt{s_d^2 + \hat{\lambda}(\bar{x}_d - DL)^2}\ .$$

The Cohen-adjusted mean was taken as the CT value. The RME value was established using the Cohen-adjusted mean and standard deviation to calculate the 95 percent UCL of the arithmetic mean using the equation presented in Section 2.1.1.

2.1.3 NON-DETECTS GREATER THAN 50 PERCENT UP TO 74 PERCENT

When more than half of a data set consists of non-detect results, estimates of the mean value and standard deviation become uncertain. If the data set contained greater than 50 percent non-detects (up to 75 percent), the CT and RME values were calculated using a bounding method estimating maximum values for the mean and 95 percent UCL, as described in Section 3.2 and Appendix A of USEPA (2002).

The CT value was calculated as the mean of the data set, substituting non-detect values with the full reported detection limit. This provides a conservative maximum value for the CT estimate.

For the RME value, an optimization process (USEPA's (2002) bounding method) was applied to find a conservative maximum bound for the 95 percent UCL of the arithmetic mean. This involved re-calculating the normal UCL (see Section 2.1.1) iteratively, allowing the non-detect values to vary between zero and the reported detection limit until a maximum value for the 95 percent UCL was obtained.

2.1.4 NON-DETECTS GREATER THAN 75 PERCENT UP TO 99 PERCENT

According to USEPA (2002), for highly censored data sets (greater than 75 percent non-detects), the recommended approach to calculate exposure estimates is to substitute non-detect results with their full detection limits and report the resulting exposure terms as values likely to be overestimated. In this case, the CT value was calculated as described in Section 2.1.3 substituting non-detects with their full detection limits. The RME value was calculated by substituting non-detects with their full detection limits and calculating the 95 percent UCL of the arithmetic mean using the equation presented in Section 2.1.1.

2.1.5 100 PERCENT NON-DETECTS

In any cases where all analytical data for a COPC were non-detect results, then the maximum detection limit was taken for both CT and RME scenarios.

2.2 LOGNORMAL DISTRIBUTION

USEPA (2003) presents three recommended methods for establishing CT and RME estimate from lognormally distributed data depending on the standard deviation of the log-transformed data. These methods are (i) the Student's *t* method, (ii) the Land (H-statistic) method, and (iii) the Chebyshev Inequality method.

The Student's *t* method was presented in Section 2.1.1. If the standard deviation of the lognormal data is small (less than 0.5), then USEPA recommends using the Student's *t* method.

The Land method is appropriate for calculating UCLs of lognormally distributed data. However, as USEPA (2002) notes, the method is very sensitive to deviations from lognormality, large variance or skewness of the dataset, and small datasets (fewer than 30 data points). The Land method can be used in conjunction with a modified Cohen's procedure (USEPA, 2002; Gilbert, 1987) to account for non-detect data.

The Chebyshev Inequality method may provide a more useful estimate (i.e., lower) of the UCL than obtained using the Land Method (USEPA, 2002). It is a distribution-free method that is applicable to a wide variety of data sets (not only lognormal data), as long as the skewness of the dataset is not large. The Chebyshev Inequality method using minimum variance unbiased estimators (MVUEs) of the mean and standard deviation of lognormal data sets is recommended for use by USEPA (2002). For small, moderately skewed datasets, a 99 percent UCL calculation using the Chebyshev Inequality is recommended (as opposed to the 95 percent value typically used).

A list of specific methods recommended for calculating RME estimates for lognormally distributed data sets are given in Table 4 (USEPA, 2002).

2.2.1 UP TO 15 PERCENT NON-DETECTS

In order to calculate the CT and RME values, the non-detect values were replaced with one-half the reported detection limit.

For the CT exposure estimate, the arithmetic mean was calculated using a bootstrap method. The bootstrap was carried out using 2000 re-sampled data sets of the same sample size as the original data set. The CT value was then taken as the average of the bootstrap means.

For the RME exposure estimate, the standard deviation of the log-transformed data was calculated, and Table 4 used to select the UCL method to use. The selected method was either i) the Student's t UCL (see Section 2.1.1 above), ii) Land's H-UCL, or iii) Chebyshev Inequality UCL.

Land's H-UCL is calculated as follows:

Step 1) Compute the arithmetic mean $\overline{x}_{\log}$ of the log-transformed data.

Step 2) Compute the standard deviation $s_{\log}$ of the log-transformed data.

Step 3) Look up the $H_{1-\alpha}$ statistic from Table 6.

Step 4) Compute the one-sided $(1-\alpha)$ upper confidence limit on the mean as:

$$UCL_{1-\alpha} = e^{\left(\overline{x}_{\log} + \frac{s^2_{\log}}{2} + \frac{H_{1-\alpha} s_{\log}}{\sqrt{n-1}}\right)}$$

where n is the number of samples.

The Chebyshev Inequality UCL is calculated as follows:

Step 1) Compute the arithmetic mean $\overline{x}_{\log}$ of the log-transformed data.

Step 2) Compute the variance $s^2_{\log}$ of the log-transformed data.

Step 3) Look up the g_n statistic from Table 7.

Step 4) Compute the minimum-variance unbiased estimator (MVUE) of the population mean for a lognormal distribution as:

$$\hat{\mu}_{\log} = e^{\overline{x}_{\log} g_n \frac{s^2_{\log}}{2}}$$

where n is the number of samples.

Step 5) Calculate the MVUE of the variance of this mean as:

$$\sigma_{\mu}^{2} = e^{2\bar{x}_{\log}}\left[\left(g_n \frac{s_{\log}^2}{2}\right)^2 - g_n\left(\frac{n-2}{n-1}s_{\log}^2\right)\right].$$

Step 6) Compute the one-sided $(1-\alpha)$ upper confidence limit on the mean as:

$$UCL_{1-\alpha} = \hat{\mu}_{\log} + \sqrt{\left(\frac{1}{\alpha} - 1\right)\sigma_{\mu}^{2}}\ .$$

2.2.2 NON-DETECTS GREATER THAN 15 PERCENT UP TO 50 PERCENT

When a moderate proportion of non-detect results is present in a data set, in order to calculate the CT estimate, a correction for non-detects was made using Gilbert's modified Cohen's method (USEPA, 2002). Gilbert (1987, page 182) suggests extending Cohen's method to account for non-detect values in lognormally distributed concentrations. Cohen's method (USEPA, 2000, page 4-43) assumes the data are normally distributed, so it must be applied to the log-transformed concentration values. If $\hat{\mu}_y$ and $\hat{\sigma}_y$ are the Cohen-corrected (see Section 2.1.2) sample mean and standard deviation, respectively, of the log-transformed concentrations, then the corrected estimates of the mean and standard deviation of the underlying lognormal distribution can be obtained from the following expressions:

$$\hat{\mu} = e^{\left(\hat{\mu}_y + \frac{\hat{\sigma}_y^2}{2}\right)}$$

$$\hat{\sigma} = \hat{\mu}\sqrt{e^{\hat{\sigma}_y^2} - 1}$$

This method assumes a single detection level for all the data values. During CT calculations, if the detection limit varied, then the highest detection limit was used for the calculations to provide a conservative estimate.

For the RME value, USEPA's bounding methodology (2002) was applied to untransformed data to find a maximum value for the mean, standard deviation, and skewness. The 95 percent UCL was then calculated using Hall's Bootstrap.

The use of Gilbert's modified Cohen's method for lognormal data was evaluated for use in calculating RME estimates for moderately censored data sets. However, attempts to use the procedure in conjunction with the lognormal UCL methods (e.g., Land's method,

Chebyshev Inequality) most often resulted with unusable values. This resulted from either calculating UCLs much higher than the maximum data point observed, or by data characteristics being unsuitable for the required calculation (e.g., needing to use a Cohen's parameter λ that was far outside existing tabulated values for this method). As a result of persistent issues with these methods, RME estimates for lognormal, moderately censored data were calculated using Hall's Bootstrap procedure. This procedure takes into account sample bias and skewness (such as present in lognormal distributions), and may be used with a bounding methodology to provide upper bonds on the UCL (USEPA, 2002). Hall's Bootstrap is calculated as follows.

Step 1) Compute the arithmetic mean $\overline{x}$.

Step 2) Compute the standard deviation s.

Step 3) Compute the skewness k.

Step 4) Re-sample the data a very large number of times (thousands of re-sample sets of the same size as the initial data set were used in this case), and calculate the each bootstrap set's mean $\overline{x}_b$, standard deviation s_b and skewness k_b.

Step 5) For each bootstrap set, calculate the studentized mean:

$$W = \frac{(\overline{x}_b - \overline{x})}{s_b}$$

Step 6) For each bootstrap set, calculate Hall's statistic:

$$Q = W + \frac{k_b W^2}{3} + \frac{k_b^2 W^3}{27} + \frac{k_b}{6n}$$

Step 7) Sort all the Q values (lowest to highest) and select the lower α^{th} quantile of the B re-sample sets. This is the $(\alpha B)^{th}$ lowest value (e.g., for 10,000 resample sets, and an α=0.05, select the 500th lowest value).

Step 8) Compute the one-sided $(1-\alpha)$ upper confidence limit on the mean as:

$$W(Q) = \frac{3}{k}\left[\sqrt[3]{1 + \left(Q_\alpha - \frac{k}{6n}\right)} - 1\right]$$

where n is the number of samples.

Step 9) Compute the one-sided $(1-\alpha)$ upper confidence limit on the mean as:

$$UCL_{1-\alpha} = \overline{x} - W(Q_\alpha)s.$$

In calculating Hall's bootstrap, five replicate calculations of the 10,000 resample sets each were generated, and the median UCL value used. These replicates were used to

determine whether or not each given data set was sensitive to small differences with the random re-sampling algorithm used by the procedure.

2.2.3 NON-DETECTS GREATER THAN 50 PERCENT UP TO 74 PERCENT

In order to calculate exposure estimates for highly-censored data sets (i.e., greater than 50 percent non-detect up to 75 percent), conservative bounding assumptions were made, as described below.

The CT value was determined by substituting the full detection limit for non-detect values and applying the bootstrap procedure introduced in Section 2.2.1. The bootstrap was carried out using 2000 re-sampled data sets of the same sample size as the original data set, and the CT estimate was then taken as the average of the bootstrap means.

In this case of a highly censored data set, Hall's Bootstrap procedure fails with increasing degrees of non-detect data due to undefined skewness values if a re-sampled data set by random chance contains only non-detects. For the RME value, USEPA's Bootstrap *t* methodology (2003) was therefore applied to calculate the 95 percent UCL. A modified bounding methodology was applied by considering four non-detect substitution scenarios: i) zero, ii) one-half detection limit, iii) full detection limit, and iv) alternating zero and full detection limit. These scenarios were considered because attempting bounding procedures on each individual re-sample set in computationally impractical. The bootstrap *t* calculation was applied under each of the four scenarios and the largest resulting UCL was selected as the RME estimate.

The bootstrap t is calculated as follows (USEPA, 2003):

Step 1) Calculate the arithmetic mean $\overline{x}$ of the original data

Step 2) Re-sample the original data a very large number of times (in this case thousands of times) and calculate each resample set's mean ($\overline{x}_b$) and standard deviation (s_b).

Step 3) For each re-sample set calculate the value:

$$t_b = \frac{(\overline{x}_b - \overline{x})}{s_b} \times \sqrt{n}$$

where n is the number of samples.

Step 4) Sort the t_b values from the lowest to the highest, and select the pivotal quantity $t_{(\alpha^*N)}$, where N is the number of bootstrap sets (e.g., if 10,000 bootstrap sets are generated and α=0.05, select the 500th lowest t_b value)

Step 5) Calculate the UCL of the population mean as:

$$UCL = \overline{x} - \frac{t_{(\alpha \bullet N)} s}{\sqrt{n}}$$

2.2.4 NON-DETECTS GREATER THAN 75 PERCENT UP TO 99 PERCENT

For very highly censored data sets (greater than 75 percent non-detects), USEPA (2002) recommends calculating exposure estimates substituting non-detects with their full detection limits, and reporting the resulting values as likely to be overestimated. The CT value was calculated using the bootstrap procedure introduced in Section 2.2.1, setting non-detects as their detection limits. For the RME calculation, the non-detects were substituted with the full detection limit, the standard deviation of the log-transformed data calculated, and Table 4 was consulted to select an appropriate UCL method. The selected methods are presented in Section 2.1.1 (Student's *t* method) and Section 2.2.1 (Land's Method and Chebyshev Inequality Procedure).

2.2.5 100 PERCENT NON-DETECTS

As for the normal case, in any situations where all analytical data for a COPC with a lognormal distribution were non-detect results, then the maximum detection limit was taken for both CT and RME scenarios.

2.3 NON-NORMAL DATASETS

For any data sets that were neither normally, nor lognormally distributed, the non-parametric/distribution-free methods presented in USEPA (2002) were used to calculate CT and RME exposures. The specific methods applied are presented below.

2.3.1 UP TO 15 PERCENT NON-DETECTS

For the CT exposure estimate, the arithmetic mean was calculated substituting non-detects with one-half the detection limit and using a bootstrap method to estimate the arithmetic mean. The same method used for log-normal data was applied (refer to Section 2.2.1), setting non-detect values as one-half their detection limits and taking the mean of 2000 bootstrap resample sets' averages as the CT value.

For the RME exposure estimate, non-detects were substituted with one-half the detection limit, and the standard deviation calculated. If the standard deviation was below 0.75 and the number of samples was 30 or greater, then the adjusted central limit theorem (CLT) UCL was calculated. Otherwise, Hall's bootstrap 95 percent UCL was used.

If sample size is sufficiently large, the Central Limit Theorem (CLT) states that the mean will be normally distributed, no matter how complex the underlying distribution of concentrations might be (USEPA, 2002). An adjusted CLT UCL method is presented in USEPA (2002) and is calculated as follows.

Step 1) Compute the arithmetic mean $\overline{x}$.

Step 2) Compute the standard deviation s.

Step 3) Compute the skewness β.

Step 4) Let z_α be the $(1-\alpha)^{th}$ quantile of the standard normal distribution (for 95 percent confidence, $z_\alpha = 1.645$).

Step 5) Compute the one-sided $(1-\alpha)$ upper confidence limit on the mean as:

$$UCL_{1-\alpha} = \overline{x} + (z_\alpha + \frac{\beta}{6\sqrt{n}}(1+2z_\alpha^2))\frac{s}{\sqrt{n}}$$

where n is the number of samples.

The Hall's Bootstrap procedure is calculated as described in Section 2.2.2.

2.3.2 NON-DETECTS GREATER THAN 15 PERCENT AND LESS THAN 50 PERCENT

For CT exposure estimates, a conservative approach was taken substituting non-detects with the full detection limit and calculating the bootstrap arithmetic mean (see Section 2.2.1).

For RME exposure estimates, Hall's bootstrap procedure (see Section 2.2.2) was used, applying bounding methodology to find maximum mean, standard deviation and skewness values for the original data set prior to re-sampling. These bounded estimates were used to calculate the Hall's Bootstrap UCL for the data using 5 sets of 10,000 resamples each (as in Section 2.2.1), and the median of these five UCLs taken as the RME estimate.

2.3.3 NON-DETECTS GREATER THAN 50 PERCENT UP TO 75 PERCENT

For CT exposure estimates, the conservative method used for the moderately censored case (described in Section 2.3.2) was used. For RME exposure estimates, the Bootstrap *t* method with modified bounding procedure described in Section 2.2.3 was applied.

2.3.4 NON-DETECTS GREATER THAN 75 PERCENT UP TO 99 PERCENT

As noted for the normal (Section 2.1.4) and lognormal (Section 2.2.4) cases for very highly censored data sets (greater than 75 percent non-detects), USEPA (2002) recommends substituting non-detects with their full detection limits and reporting exposure estimates as likely to be overestimated. Both CT and RME values were calculated accordingly, as follows.

For CT exposure estimates, a conservative approach was taken substituting non-detects with the full detection limit and calculating the bootstrap arithmetic mean (see Section 2.2.1). This is the same method used for the moderately censored 15 to 50 percent non-detect case (Section 2.3.2).

For RME estimates, the non-detects were substituted with the full detection limit and Bootstrap *t* was used to calculate the UCL (refer to Section 2.2.3).

2.3.5 100 PERCENT NON-DETECTS

In any cases where all analytical data for a COPC were non-detect results, then the maximum detection limit was taken for both CT and RME estimates.

3.0 MAXIMUM DETECTED VALUE

USEPA (1992 and 2002) allow an optional use of the maximum observed concentrations for the RME estimate in cases where the calculated UCL exceeds the maximum value. However, USEPA (2002) warns that this may not be appropriate for data sets with very small sample sizes, because the observed maximum may be below the population mean.

If the RME estimate calculated using any of the statistical methods presented in Section 2.0 was larger than the maximum detected value, then the maximum detected value was used for the RME.

4.0 REFERENCES

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TABLE 1

GUIDELINES FOR ANALYZING DATA WITH NON-DETECTS [1]

Percentage of Non-detects	*Statistical Analysis Method*
<15%	Replace non-detects with detection limit/2, detection limit, or a very small number.
15% - 50%	Trimmed mean, Cohen's adjustment, Winsorized mean and standard deviation, bounding method[2], probability substitution based on specific distribution[2].
>50% - 90%	Use tests for proportions, bounding method[2][3].

Notes:

(1) adapted from USEPA, (2000), Guidance for Data Quality Assessment Practical Methods for Data Analysis EPA QA/G-9, EPA/600/R-96/084, July 2000.

(2) USEPA, (2002), Calculating Upper Confidence Limits for Exposure Point Concentrations at Hazardous Waste Sites, Office of Emergency and Remedial Response, OSWER 9285.6-10, December 2002.

(3) When greater than 75 percent non-detects present and the sample size is small (less than five samples), the bounding method should be conservatively applied setting non-detects at the detection limit (USEPA, 2002).

TABLE 2

RECOMMENDED METHODS FOR CALCULATING UPPER CONFIDENCE LIMITS (UCLs)

Method	*Applicability*	*Advantages*	*Disadvantages*	*Reference*
(i) *For Normal or Lognormal Distributions*				
Student's *t*	means normally distributed, samples random	simple, robust if n is large	distribution of means must be normal	Gilbert 1987; EPA 1992
Land's *H*	lognormal data, small variance, large n, samples random	good coverage[1]	sensitive to deviations from lognormality, produces very high values for large variance or small n	Gilbert 1987; EPA 1992
Chebyshev Inequality (MVUE)	skewness and variance small or moderate, samples random	often smaller than Land	may need to resort to higher confidence levels for adequate coverage	Singh *et al*. 1997
Wong	gamma distribution	second order accuracy[2]	requires numerical solution of an improper integral	Schulz and Griffin 1999; Wong 1993
(ii) *Nonparametric/Distribution-free Methods*				
Central Limit Theorem - Adjusted	large n, samples random	simple, robust	sample size may not be sufficient	Gilbert 1987; Singh *et al*. 1997
Bootstrap *t* Resampling	sampling is random and representative	useful when distribution cannot be identified	inadequate coverage for some distributions; computationally intensive	Singh *et al*. 1997; Efron 1982
Hall's Bootstrap Procedure	sampling is random and representative	useful when distribution cannot be identified; takes bias and skewness into account	inadequate coverage for some distributions; computationally intensive	Hall 1988; Hall 1992; Manly 1997; Schultz and Griffin 1999
Jackknife Procedure	sampling is random and representative	useful when distribution cannot be identified	inadequate coverage for some distributions; computationally intensive	Singh *et al*. 1997
Chebyshev Inequality	skewness and variance small or moderate, samples random	useful when distribution cannot be identified	inappropriate for small sample sizes when skewness or variance is large	Singh *et al*. 1997; EPA 2000c

Notes:

This Table was taken from USEPA, 2002.

(1) Coverage refers to whether a UCL method performs in accordance with its definition.

(2) As opposed to maximum likelihood estimation, which offers first order accuracy.

TABLE 3

STATISTICAL METHODS USED FOR DETERMINING EXPOSURE ESTIMATES UNDER CENTRAL TENDENCY (CT) AND REASONABLE MAXIMUM EXPOSURE (RME) SCENARIOS

Percentage of Non-detect Values	*Data Distribution*		
	Normal	*Lognormal*	*Not Normal*
I) Central Tendency (CT) Exposure Scenarios			
0-15 percent	Substitute non-detect results with one-half detection limit. Calculate arithmetic mean.	Substitute non-detect results with one-half detection limit. Calculate arithmetic mean of 2000 bootstrap resample set means.	Substitute non-detect results with one-half detection limit. Calculate arithmetic mean of 2000 bootstrap resample set means.
>15-50 percent	Use Cohen's method to determine non-detect-adjusted estimate of arithmetic mean.	Use Gilbert's modified Cohen's method to determine non-detect-adjusted estimate of arithmetic mean for lognormal data.	Substitute non-detect results with full detection limit. Calculate arithmetic mean of 2000 bootstrap resample set means.
>50-74 percent	Substitute non-detect results with full detection limit. Calculate arithmetic mean.	Substitute non-detect results with full detection limit. Calculate arithmetic mean of 2000 bootstrap resample set means.	Substitute non-detect results with full detection limit. Calculate arithmetic mean of 2000 bootstrap resample set means.
>75-99 percent	Substitute non-detect results with full detection limit. Calculate arithmetic mean.	Substitute non-detect results with full detection limit. Calculate arithmetic mean of 2000 bootstrap resample set means.	Substitute non-detect results with full detection limit. Calculate arithmetic mean of 2000 bootstrap resample set means.
100 percent	Use maximum detection limit.	Use maximum detection limit.	Use maximum detection limit.

TABLE 3

STATISTICAL METHODS USED FOR DETERMINING EXPOSURE ESTIMATES UNDER CENTRAL TENDENCY (CT) AND REASONABLE MAXIMUM EXPOSURE (RME) SCENARIOS

Percentage of Non-detect Values	*Data Distribution*		
	Normal	*Lognormal*	*Not Normal*
II) Reasonable Maximum Exposure (RME) Scenarios [1]			
0-15 percent[2]	Substitute non-detect results with one-half detection limit. Calculate Student's *t* 95-percent UCL of arithmetic mean.	Substitute non-detect results with one-half detection limit. Calculate standard deviation of log-transformed data. Use Table 4 to select UCL method.	Substitute non-detect results with one-half detection limit. If $s > 0.75$ and $n>29$: Use Adjusted Central Limit Theorem 95-percent UCL of mean. Otherwise, calculate Hall's bootstrap 95-UCL.
>15-50 percent[2]	Use Cohen's method to determine non-detect-adjusted estimates of mean and standard deviation. Calculate Student's *t* 95-percent UCL of arithmetic mean.	Use bounding methodology[3] on untransformed data to find maximum mean, standard deviation and skewness. Calculate Hall's bootstrap 95-percent UCL.	Use bounding methodology[3] to find maximum mean, standard deviation and skewness. Calculate Hall's bootstrap 95-percent UCL.
>50-74 percent[2]	Use a bounding methodology[3] to find maximum Student's *t* 95-percent UCL of arithmetic mean.	Considering data set with ND=0, ND=0.5 DL, ND=DL and alternating NDs 0 and DL. Calculate bootstrap-*t* 95-percent UCL for each of the four data sets. Select the largest value as "bounded" UCL.	Considering data set with ND=0, ND=0.5 DL, ND=DL and alternating NDs 0 and DL. Calculate bootstrap-*t* 95-percent UCL for each of the four data sets. Select the largest value as "bounded" UCL.
>75-99 percent[2]	Substitute non-detects with their full detection limit. Calculate Student's *t* UCL of arithmetic mean (likely to be overestimated - per USEPA 2002).	Substitute non-detects with their full detection limit. Calculate standard deviation of log-transformed data. Use Table 4 to select UCL method (likely to be overestimated - per USEPA 2002).	Substitute non-detects with their full detection limit. Calculate bootstrap-*t* 95-percent UCL (likely to be overestimated - per USEPA 2002).
100 percent	Use maximum detection limit.	Use maximum detection limit.	Use maximum detection limit.

Notes:

[1] RMEs are calculated as 95 percent upper confidence limits of the mean. Specific UCL methods were chosen based on Figure 1 and the text of USEPA (2002) and (2003).

[2] As per USEPA 2002, if the calculated UCL value exceeds the maximum detected value and a sufficient number of samples have been collected to meet data quality objectives, then the maximum detected value is used for the UCL.

[3] See Appendix A of USEPA 2002 for description of bounding methodology (note that "Step 9" of the appendix should say "less than", not "greater than").

[4] For Student's *t* UCL, use Cohen's method; for Land's H UCL, use Gilbert's modified Cohen's method; for Chebyshev UCL, use Cohen's method on log-transformed data.

TABLE 4

95 PERCENT UPPER CONFIDENCE LIMIT (UCL) CALCULATION METHODS FOR LOGNORMAL DATA

Standard deviation of log-transformed data (s)	*Number of Samples (n)*	*Selected Upper Confidence Limit Method* [1]
$0 \le s < 0.5$	For all n (≥ 5)	Student's *t* UCL
$0.5 \le s < 1.0$	For all n	Land's H-UCL
$1.0 \le s < 1.5$	$n < 25$	Chebyshev UCL (95% MVUE)
	$n \ge 25$	Land's H-UCL
$1.5 \le s < 2.0$	$n < 20$	Chebyshev UCL (99% MVUE)
	$20 \le n \le 50$	Chebyshev UCL (95% MVUE)
	$n \ge 50$	Land's H-UCL
$2.0 \le s < 2.5$	$n < 25$	Chebyshev UCL (99% MVUE)
	$25 \le n \le 70$	Chebyshev UCL (95% MVUE)
	$n \ge 70$	Land's H-UCL
$2.5 \le s < 3.0$	$n < 30$	Chebyshev UCL (max of 99% MVUE or 99% mean)
	$30 \le n \le 70$	Chebyshev UCL (max of 95% MVUE or 95% mean)
	$n \ge 70$	Land's H-UCL
$s \ge 3.0$	Small n	Further investigation required
	$n > 100$	Land's H-UCL

Note:

[1] Source: Table A1 of USEPA (2003) -- ProUCL User's Guide Version 2.1, February, 2003.

TABLE 5

VALUES OF LAMBDA (λ) FOR COHEN'S METHOD

	Percentage of Non-detects (h)						
λ	*0.01*	*0.05*	*0.10*	*0.15*	*0.25*	*0.40*	*0.50*
0.01	0.0102	0.0530	0.1111	0.1747	0.3205	0.5989	0.8403
0.05	0.0105	0.0547	0.1143	0.1793	0.3279	0.6101	0.8540
0.10	0.0110	0.0566	0.1180	0.1848	0.3366	0.6234	0.8703
0.15	0.0113	0.0584	0.1215	0.1898	0.3448	0.6361	0.8860
0.20	0.0116	0.0600	0.1247	0.1946	0.3525	0.6483	0.9012
0.30	0.0122	0.0630	0.1306	0.2034	0.3670	0.6713	0.9300
0.40	0.0128	0.0657	0.1360	0.2114	0.3803	0.6927	0.9570
0.50	0.0133	0.0681	0.1409	0.2188	0.3928	0.7129	0.9826
0.60	0.0137	0.0704	0.1455	0.2258	0.4045	0.7320	1.0070
0.70	0.0142	0.0726	0.1499	0.2323	0.4156	0.7502	1.0303
0.80	0.0146	0.0747	0.1540	0.2386	0.4261	0.7676	1.0527
0.90	0.0150	0.0766	0.1579	0.2445	0.4362	0.7844	1.0743
1.00	0.0153	0.0785	0.1617	0.2502	0.4459	0.8005	1.0951
1.10	0.0157	0.0803	0.1653	0.2557	0.4553	0.8161	1.1152
1.20	0.0160	0.0820	0.1688	0.2610	0.4643	0.8312	1.1347
1.30	0.0164	0.0836	0.1722	0.2661	0.4730	0.8458	1.1537
1.40	0.0167	0.0853	0.1754	0.2710	0.4815	0.8600	1.1721
1.50	0.0170	0.0868	0.1786	0.2758	0.4897	0.8738	1.1901
1.60	0.0173	0.0883	0.1817	0.2805	0.4977	0.8873	1.2076
1.70	0.0176	0.0898	0.1846	0.2851	0.5055	0.9005	1.2248
1.80	0.0179	0.0913	0.1876	0.2895	0.5132	0.9133	1.2415
1.90	0.0181	0.0927	0.1904	0.2938	0.5206	0.9259	1.2579
2.00	0.0184	0.0940	0.1932	0.2981	0.5279	0.9382	1.2739
2.10	0.0187	0.0954	0.1959	0.3022	0.5350	0.9502	1.2897
2.20	0.0189	0.0967	0.1986	0.3062	0.5420	0.9620	1.3051
2.30	0.0192	0.0980	0.2012	0.3102	0.5488	0.9736	1.3203
2.40	0.0194	0.0992	0.2037	0.3141	0.5555	0.9850	1.3352
2.50	0.0197	0.1005	0.2062	0.3179	0.5621	0.9962	1.3498
2.60	0.0199	0.1017	0.2087	0.3217	0.5686	1.0072	1.3642
2.70	0.0202	0.1029	0.2111	0.3254	0.5750	1.0180	1.3784
2.80	0.0204	0.1040	0.2135	0.3290	0.5812	1.0287	1.3924
2.90	0.0206	0.1052	0.2158	0.3326	0.5874	1.0392	1.4061
3.00	0.0209	0.1063	0.2182	0.3361	0.5935	1.0495	1.4197
3.10	0.0211	0.1074	0.2204	0.3396	0.5995	1.0597	1.4330
3.20	0.0213	0.1085	0.2227	0.3430	0.6054	1.0697	1.4462

TABLE 5

VALUES OF LAMBDA (λ) FOR COHEN'S METHOD

λ	*Percentage of Non-detects (h)*						
	0.01	***0.05***	***0.10***	***0.15***	***0.25***	***0.40***	***0.50***
3.30	0.0215	0.1096	0.2249	0.3464	0.6112	1.0796	1.4592
3.40	0.0217	0.1107	0.2270	0.3497	0.6169	1.0894	1.4720
3.50	0.0219	0.1118	0.2292	0.3529	0.6226	1.0990	1.4847
3.60	0.0221	0.1128	0.2313	0.3562	0.6282	1.1086	1.4972
3.70	0.0223	0.1138	0.2334	0.3594	0.6337	1.1180	1.5096
3.80	0.0225	0.1148	0.2355	0.3625	0.6391	1.1273	1.5218
3.90	0.0227	0.1158	0.2375	0.3656	0.6445	1.1364	1.5339
4.00	0.0229	0.1168	0.2395	0.3687	0.6498	1.1455	1.5458
4.10	0.0231	0.1178	0.2415	0.3717	0.6551	1.1545	1.5577
4.20	0.0233	0.1188	0.2435	0.3747	0.6603	1.1634	1.5693
4.30	0.0235	0.1197	0.2454	0.3777	0.6654	1.1722	1.5809
4.40	0.0237	0.1207	0.2473	0.3806	0.6705	1.1809	1.5924
4.50	0.0239	0.1216	0.2492	0.3836	0.6755	1.1895	1.6037
4.60	0.0241	0.1225	0.2511	0.3864	0.6805	1.1980	1.6149
4.70	0.0242	0.1235	0.2530	0.3893	0.6855	1.2064	1.6260
4.80	0.0244	0.1244	0.2548	0.3921	0.6903	1.2148	1.6370
4.90	0.0246	0.1253	0.2567	0.3949	0.6952	1.2230	1.6479
5.00	0.0248	0.1262	0.2585	0.3977	0.7000	1.2312	1.6587
5.10	0.0249	0.1270	0.2603	0.4004	0.7047	1.2394	1.6694
5.20	0.0251	0.1279	0.2621	0.4031	0.7094	1.2474	1.6800
5.30	0.0253	0.1288	0.2638	0.4058	0.7141	1.2554	1.6905
5.40	0.0255	0.1296	0.2656	0.4085	0.7187	1.2633	1.7010
5.50	0.0256	0.1305	0.2673	0.4111	0.7233	1.2711	1.7113
5.60	0.0258	0.1313	0.2690	0.4137	0.7278	1.2789	1.7215
5.70	0.0260	0.1322	0.2707	0.4163	0.7323	1.2866	1.7317
5.80	0.0261	0.1330	0.2724	0.4189	0.7368	1.2943	1.7418
5.90	0.0263	0.1338	0.2741	0.4215	0.7412	1.3019	1.7518
6.00	0.0264	0.1346	0.2757	0.4240	0.7456	1.3094	1.7617

Source: McBean & Rovers, 1998

TABLE 6

VALUES OF H(0.95) FOR LAND'S METHOD

Standard deviation of log-transformed data (S_{log})	*Number of Samples*												
	3	*5*	*7*	*10*	*12*	*15*	*21*	*31*	*51*	*101*	*301*	*601*	*1001*
0.10	2.750	2.035	1.886	1.802	1.775	1.749	1.722	1.701	1.684	1.670	1.659	1.656	1.654
0.20	3.295	2.198	1.992	1.881	1.843	1.809	1.771	1.742	1.718	1.697	1.680	1.674	1.671
0.30	4.109	2.402	2.125	1.977	1.927	1.882	1.833	1.793	1.761	1.733	1.709	1.700	1.696
0.40	5.220	2.651	2.282	2.089	2.026	1.968	1.905	1.856	1.813	1.777	1.746	1.734	1.728
0.50	6.495	2.947	2.465	2.220	2.141	2.068	1.989	1.928	1.876	1.830	1.790	1.776	1.769
0.60	7.807	3.287	2.673	2.368	2.271	2.181	2.085	2.010	1.946	1.891	1.843	1.825	1.816
0.70	9.120	3.662	2.904	2.532	2.414	2.306	2.191	2.102	2.025	1.960	1.902	1.881	1.870
0.80	10.43	4.062	3.155	2.710	2.570	2.443	2.307	2.202	2.112	2.035	1.968	1.944	1.931
0.90	11.74	4.478	3.420	2.902	2.738	2.589	2.432	2.310	2.206	2.117	2.040	2.012	1.997
1.00	13.05	4.905	3.695	3.103	2.915	2.744	2.564	2.423	2.306	2.205	2.117	2.085	2.068
1.25	16.33	6.001	4.426	3.639	3.389	3.163	2.923	2.737	2.580	2.447	2.330	2.288	2.266
1.50	19.60	7.120	5.184	4.207	3.896	3.612	3.311	3.077	2.881	2.713	2.566	2.514	2.486
1.75	22.87	8.250	5.960	4.795	4.422	4.081	3.719	3.437	3.200	2.997	2.820	2.757	2.723
2.00	26.14	9.387	6.747	5.396	4.962	4.564	4.141	3.812	3.533	3.295	3.088	3.013	2.974
2.50	32.69	11.67	8.339	6.621	6.067	5.557	5.013	4.588	4.228	3.920	3.650	3.553	3.503
3.00	39.23	13.97	9.945	7.864	7.191	6.570	5.907	5.388	4.947	4.569	4.238	4.119	4.057
3.50	45.77	16.27	11.56	9.118	8.326	7.596	6.815	6.201	5.681	5.233	4.842	4.700	4.627
4.00	52.31	18.58	13.18	10.38	9.469	8.630	7.731	7.024	6.424	5.908	5.456	5.293	5.208
4.50	58.85	20.88	14.80	11.64	10.62	9.669	8.652	7.854	7.174	6.590	6.077	5.892	5.796
5.00	65.39	23.19	16.43	12.91	11.77	10.71	9.579	8.688	7.929	7.277	6.704	6.497	6.390
6.00	78.47	27.81	19.68	15.45	14.08	12.81	11.44	10.36	9.449	8.661	7.968	7.718	7.588
7.00	91.55	32.43	22.94	18.00	16.39	14.90	13.31	12.05	10.98	10.05	9.242	8.949	8.797
8.00	104.6	37.06	26.20	20.55	18.71	17.01	15.18	13.74	12.51	11.45	10.52	10.19	10.01
9.00	117.7	41.68	29.46	23.10	21.03	19.11	17.05	15.43	14.05	12.85	11.81	11.43	11.23
10.00	130.8	46.31	32.73	25.66	23.35	21.22	18.93	17.13	15.59	14.26	13.10	12.67	12.45

Sources: Land (1975) and Gilbert (1987).

TABLE 7

VALUES OF g_n FOR CHEBYSHEV'S METHOD

Variance ÷ 2 of log-transformed data ($S_{log}^2 \div 2$)	*Number of Samples*																
	2	*5*	*8*	*10*	*13*	*15*	*20*	*25*	*30*	*50*	*70*	*90*	*100*	*150*	*200*	*500*	*∞*
0.05	1.025	1.041	1.045	1.046	1.047	1.048	1.049	1.049	1.049	1.050	1.050	1.051	1.051	1.051	1.051	1.051	1.051
0.10	1.050	1.082	1.091	1.093	1.096	1.097	1.099	1.100	1.101	1.103	1.103	1.104	1.104	1.104	1.105	1.105	1.105
0.15	1.076	1.125	1.138	1.143	1.147	1.149	1.152	1.154	1.155	1.158	1.159	1.160	1.160	1.161	1.161	1.161	1.162
0.20	1.102	1.169	1.187	1.194	1.200	1.203	1.207	1.210	1.212	1.216	1.217	1.218	1.218	1.219	1.220	1.221	1.221
0.25	1.128	1.214	1.238	1.247	1.255	1.259	1.265	1.268	1.271	1.276	1.278	1.280	1.280	1.281	1.282	1.283	1.284
0.30	1.154	1.260	1.291	1.302	1.312	1.317	1.325	1.330	1.333	1.340	1.342	1.344	1.345	1.346	1.347	1.349	1.350
0.35	1.180	1.307	1.345	1.359	1.372	1.378	1.387	1.393	1.398	1.406	1.410	1.412	1.412	1.415	1.416	1.418	1.419
0.40	1.207	1.356	1.401	1.418	1.433	1.441	1.453	1.460	1.465	1.476	1.480	1.483	1.484	1.486	1.488	1.490	1.492
0.45	1.234	1.406	1.459	1.479	1.498	1.506	1.521	1.530	1.536	1.548	1.554	1.557	1.558	1.562	1.563	1.566	1.568
0.50	1.261	1.457	1.519	1.542	1.564	1.574	1.592	1.602	1.610	1.625	1.631	1.635	1.637	1.641	1.643	1.646	1.649
0.55	1.288	1.509	1.581	1.608	1.633	1.645	1.666	1.678	1.687	1.705	1.713	1.717	1.719	1.724	1.726	1.730	1.733
0.60	1.315	1.563	1.645	1.675	1.705	1.719	1.743	1.757	1.768	1.789	1.798	1.803	1.805	1.811	1.813	1.819	1.822
0.65	1.343	1.618	1.711	1.746	1.780	1.796	1.823	1.840	1.852	1.876	1.887	1.893	1.895	1.902	1.905	1.911	1.916
0.70	1.371	1.675	1.779	1.818	1.857	1.876	1.907	1.926	1.940	1.968	1.981	1.988	1.990	1.998	2.002	2.009	2.014
0.75	1.399	1.733	1.849	1.894	1.938	1.959	1.994	2.016	2.032	2.064	2.079	2.087	2.090	2.099	2.103	2.111	2.117
0.80	1.427	1.792	1.922	1.971	2.021	2.045	2.085	2.110	2.128	2.165	2.182	2.191	2.194	2.205	2.210	2.219	2.226
0.85	1.456	1.853	1.996	2.052	2.108	2.134	2.179	2.208	2.228	2.270	2.289	2.300	2.304	2.316	2.322	2.332	2.340
0.90	1.485	1.915	2.074	2.135	2.197	2.227	2.278	2.310	2.333	2.381	2.402	2.415	2.419	2.432	2.439	2.451	2.460
0.95	1.514	1.979	2.153	2.221	2.291	2.323	2.380	2.417	2.442	2.496	2.521	2.535	2.539	2.555	2.562	2.576	2.586
1.00	1.543	2.044	2.235	2.310	2.387	2.424	2.487	2.528	2.557	2.617	2.645	2.660	2.666	2.683	2.692	2.707	2.718
1.05	1.573	2.111	2.320	2.403	2.487	2.528	2.598	2.644	2.676	2.744	2.774	2.792	2.798	2.818	2.827	2.845	2.858
1.10	1.602	2.180	2.407	2.498	2.591	2.636	2.714	2.765	2.800	2.876	2.911	2.930	2.938	2.959	2.970	2.990	3.004
1.15	1.632	2.250	2.497	2.596	2.699	2.748	2.834	2.891	2.930	3.014	3.053	3.075	3.083	3.108	3.120	3.143	3.158
1.20	1.662	2.321	2.589	2.698	2.810	2.864	2.960	3.022	3.066	3.159	3.203	3.227	3.236	3.263	3.277	3.303	3.320
1.25	1.693	2.395	2.685	2.803	2.926	2.985	3.090	3.159	3.207	3.311	3.359	3.387	3.397	3.427	3.442	3.471	3.490
1.30	1.724	2.470	2.783	2.911	3.045	3.111	3.226	3.301	3.354	3.470	3.523	3.554	3.565	3.599	3.616	3.648	3.669
1.35	1.754	2.547	2.884	3.023	3.169	3.241	3.367	3.450	3.508	3.636	3.695	3.729	3.741	3.779	3.798	3.833	3.857
1.40	1.786	2.626	2.988	3.139	3.298	3.376	3.513	3.604	3.669	3.809	3.875	3.912	3.926	3.968	3.989	4.028	4.055
1.45	1.817	2.706	3.096	3.259	3.431	3.515	3.666	3.766	3.836	3.991	4.063	4.105	4.120	4.166	4.190	4.233	4.263
1.50	1.849	2.788	3.206	3.382	3.569	3.661	3.825	3.933	4.011	4.181	4.260	4.307	4.323	4.374	4.400	4.448	4.482
1.55	1.880	2.873	3.320	3.510	3.711	3.811	3.990	4.108	4.193	4.379	4.467	4.518	4.536	4.592	4.621	4.675	4.711
1.60	1.913	2.959	3.437	3.642	3.859	3.967	4.161	4.291	4.383	4.587	4.683	4.739	4.759	4.821	4.853	4.912	4.953
1.65	1.945	3.047	3.558	3.777	4.012	4.129	4.339	4.480	4.581	4.804	4.910	4.971	4.993	5.062	5.097	5.162	5.207
1.70	1.977	3.137	3.682	3.918	4.171	4.297	4.525	4.678	4.788	5.031	5.147	5.215	5.239	5.314	5.353	5.424	5.474
1.75	2.010	3.229	3.810	4.062	4.334	4.471	4.717	4.883	5.003	5.269	5.395	5.469	5.496	5.578	5.621	5.700	5.755
1.80	2.043	3.323	3.942	4.212	4.504	4.651	4.917	5.097	5.227	5.517	5.655	5.736	5.766	5.856	5.903	5.990	6.050
1.85	2.077	3.420	4.077	4.366	4.680	4.838	5.125	5.320	5.461	5.776	5.927	6.016	6.048	6.147	6.198	6.294	6.360
1.90	2.110	3.518	4.216	4.525	4.861	5.031	5.341	5.552	5.705	6.048	6.212	6.309	6.344	6.453	6.509	6.613	6.686
1.95	2.144	3.619	4.359	4.688	5.049	5.232	5.566	5.794	5.959	6.331	6.511	6.617	6.655	6.773	6.834	6.949	7.029
2.00	2.178	3.721	4.506	4.858	5.243	5.439	5.799	6.045	6.224	6.628	6.823	6.938	6.980	7.109	7.176	7.302	7.389

Source: After Gilbert (1987).

APPENDIX Q.4

TOXICITY PROFILES FOR COPCs

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1.0 VOLATILE ORGANIC COMPOUNDS

1.1 1,2-DICHLOROETHANE (1,2-DCA)

1. Constituent Properties

A. Physical and Chemical Properties

Molecular Weight:	98.96
Melting Point:	-35.3°C
Boiling Point:	83.5°C
Specific Density:	1.23 (@ 20°C)
Water Solubility (mg/L):	8,690 (@ 20°C)
Vapor Pressure (mm Hg):	79.1 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	1.1 x 10^{-3} (@ 20°C)

Reference: ATSDR, 2001. Toxicological Profile for 1,2-Dichloroethane.

B. Chemical Transformation

Air: 1,2-DCA is photooxidized by reaction with photochemically produced hydroxyl radicals. The atmospheric life-time of 1,2-DCA is reported to be >5 months with formyl chloride, chloroacetyl chloride, hydrogen chloride and chloroethanol reported as degradation products (ATSDR, 2001).

Water: Volatilization dominates the fate of 1,2-DCA in surface water. In groundwater, biodegradation contributes to the removal of 1,2-DCA. There is strong evidence from studies that the co-metabolism of 1,2-DCA occurs under aerobic conditions. Abiotic degradation processes, such as oxidation and hydrolysis, are too slow to be environmentally significant (ATSDR, 2001).

Soil: 1,2-DCA is expected to partition to the atmosphere or be transported to groundwater. The primary transformation process of 1,2-DCA is biodegradation. 1,2-DCA was completely dechlorinated to ethane under anaerobic conditions. Soils exposed to methane biodegraded 1,2-DCA to carbon dioxide (ATSDR, 2001).

2. Toxicological Properties

A. Metabolism

There are no studies regarding metabolism of 1,2-DCA in humans. Convincing evidence from animal studies suggest that reactive intermediates are formed by conjugation with glutathione. Studies in rats and mice indicate that 1,2-DCA may be metabolized to 2-chloroacetaldehyde, *S*-(2-chloroethyl)glutathione, and other putative reactive intermediates capable of binding covalently to cellular macromolecules in the liver, kidney and other tissues. Thus, the severity of 1,2-DCA-induced toxicity in humans may be dependent on the extent to which 1,2-DCA is metabolized and what intermediates are formed. It

appears that at lower dose levels that do not saturate the metabolic pathways, metabolic detoxification prevents the toxic effect of 1,2-DCA. 1,2-DCA is rapidly excreted from the body. Following inhalation and oral exposure, elimination of 1,2-DCA occurred primarily via excretion of soluble metabolites in the urine and parent compound and carbon dioxide in the expired air. In animals, 1,2-DCA and its metabolites were excreted within 48 hours of exposure (ATSDR, 2001)

B. Acute Toxicity

Available information regarding the health effects of 1,2-DCA in humans came from death reports following acute exposures to high levels by inhalation or ingestion. Symptoms included central nervous system depression, nausea and vomiting, corneal opacity, bronchitis, respiratory distress, lung congestion, myocardial lesions, hemorrhagic gastritis and colitis, increased blood clotting time, hepatocellular damage, renal necrosis, and histopathological changes in brain tissue (ATSDR, 2001). Death was most often attributed to cardiac arrhythmia. Autopsy results showed hemorrhagic colitis and gastritis in the gastrointestinal tract of people who died after acute oral exposure. Similar effects have been reported in animals; vomiting and diarrhea preceded death in dogs given acute high-level inhalation exposure (ATSDR, 2001).

C. Subacute and Chronic Toxicity

There is very limited studies regarding exposure to 1,2-DCA. Intermediate duration intermittent exposures caused death in guinea pigs, rats and mice exposed to 200 parts per million (ppm), rats and rabbits exposed to 400 ppm, and dogs, cats and monkeys exposed to 1,000 ppm. Necropsy of these animals showed liver, kidney, heart and lung effects similar to those seen in acute exposures (ATSDR, 2001).

D. Carcinogenicity

1,2-DCA is classified by the USEPA as Group B2, a probable human carcinogen based on the induction of several tumor types in rats and mice treated by gavage and lung papillomas in mice after topical application (USEPA, 1991a).

E. Mutagenicity

Evidence from genotoxic studies indicates that 1,2-DCA is capable of interacting with DNA to produce genotoxic effects *in vitro*. Results were positive in assays for point mutations in human cells, animal cells, and bacteria. By itself, 1,2-DCA is a weak mutagen, however, it can be activated to a more effective mutagen with a metabolic activation system (ATSDR, 2001). 1,2-DCA was mutagenic for Salmonella in assays wherein excessive evaporation was prevented; exogenous metabolism by mammalian systems enhanced the response. Both somatic cell mutations and sex-linked recessives were induced in Drosphila. Metabolites of

1,2-DCA have been shown to form adducts with DNA after *in vitro* or *in vivo* exposures (USEPA, 1991a).

F. Teratogenicity/Reproductive Effects

A single study on reproductive effects of exposure to 1,2-DCA in humans is suggestive of a reduction in gestation duration, but co-exposure to other chemicals occurred in most cases. Results in animal studies indicate that 1,2-DCA does not cause reproductive effects. One and two generation studies found no chemical-related effects on fertility indices in long-term oral studies in mice and rats, but at extremely high doses caused increases in nonsurviving implants and resorptions in rats that also caused maternal toxicity. Histological examinations of the testes, ovaries, and other reproductive system tissues had negative results (ATSDR, 2001).

There are only two studies regarding developmental effects in humans. One study reported adverse birth outcomes of increased odds ratios for exposure to 1,2-DCA in drinking water and major cardiac defects but not neural tube defects and the other reported for residence had neural tube defects but no heart defects. Because of mixed chemical exposure, lack of dose-response and inconsistency between the findings, the effects are only suggestive. Available inhalation and oral studies in rats, mice and rabbits indicate that 1,2-DCA is not fetotoxic or teratogenic, although indications of embryolethality and maternal toxic doses have been reported (ATSDR, 2001).

G. Other Health Effects

Immunological effects have not been reported in humans, however, in mice the immune system was the most sensitive target for short-term exposure to 1,2-DCA following both acute inhalation and oral exposure. Effects observed included reduced humoral immunity and cell-mediated immunity (ATSDR, 2001).

H. Epidemiological Evidence

The only known human health effects of 1,2-DCA, seen in cases of acute high exposure are neurotoxicity, nephrotoxicity and hepatotoxicity as well as death due to cardiac arrythmia (ATSDR, 2001).

I. Toxicity

1,2-DCA is classified by the USEPA as Group B2, a probable human carcinogen based on sufficient evidence in animal studies. The chronic cancer slope factors are 0.091 (mg/kg-day)-1 and 0.091 (mg/kg-day)-1 for the oral and inhalation routes, respectively. The chronic reference doses are 0.02 and 0.0014 mg/kg-day for the oral and inhalation routes, respectively. The aforementioned toxicity values were taken from USEPA, 2004a.

1.2 CIS- AND TRANS-1,2-DICHLOROETHENE (CIS- AND TRANS-1,2-DCE)

1. Constituent Properties

A. Physical and Chemical Properties

cis-1,2-DCE

Atomic Weight (g/mol):	96.94
Boiling Point:	60.3°C
Melting Point:	-80.5°C
Specific Density:	1.2837 (@ 20°C)
Water Solubility (mg/L):	3,500 (@ 25°C)
Vapor Pressure (mm Hg):	215 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	4.08×10^{-3} (@ 25°C)

trans-1,2-DCE

Atomic Weight (g/mol):	96.94
Boiling Point:	47.5°C
Melting Point:	-50°C
Specific Density:	1.2565 (@ 20°C)
Water Solubility (mg/L):	6,260 (@ 25°C)
Vapor Pressure (mm Hg):	336 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	9.38×10^{-3} (@ 25°C)

Reference: ATSDR, 1995a. Toxicological Profile for 1,2-Dichloroethylene.

B. Chemical Transformation

Air: The dominant removal process for 1,2-DCA is predicted to be reaction with photochemically produced hydroxyl radicals, with formyl chloride being one positively identified by-products of the reaction (ATSDR, 1995a).

Water: In surface waters, 1,2-DCE is assumed to be rapidly transferred to the atmosphere. Hydrolysis, photolysis, and oxidation are not important fate processes for 1,2-DCE in surface waters (ATSDR, 1995a). 1,2-DCE and other chlorinated ethenes generally resist biodegradation under aerobic conditions. In aerobic conditions, such as groundwater, 1,2-DCE undergoes slow reductive dechlorination (ATSDR, 1995a). The cis isomer degrades to chloroethane and vinyl chloride, while the trans isomer degrades to vinyl chloride only.

Soil: Studies suggest the 1,2-DCE isomers undergo anaerobic biodegradation in soil and that this process may be the sole mechanism by which 1,2-DCE degrades in soil, again producing vinyl chloride (ATSDR, 1995a).

2. Toxicological Properties

A. Metabolism

Although 1,2-DCE is relatively lipophilic, there is good evidence that it can accumulate in important organs such as liver, brain, kidney, and adipose tissue. It is more likely that 1,2-DCE will be metabolized to more hydrophilic by-products, and therefore, eliminated quickly as metabolites (ATSDR, 1995a). It has been reported that both the cis and trans isomers of 1,2-DCE are converted to dichloroethanol and dichloroacetic acid by rat liver (ATSDR, 1995a). The metabolism of the cis isomer is believed to occur at a greater rate than the trans isomer. As a result, the cis isomer also exhibits a higher rate of metabolic elimination under saturation conditions, in comparison to the trans isomer (ATSDR, 1995a).

B. Acute Toxicity

Human symptoms reported from exposure to high levels (1,715 to 2,220 ppm) of 1,2-DCE in air include nausea, drowsiness, fatigue, intracranial pressure and ocular irritation (ATSDR, 1995a). Only one human fatality has been reported. No information is available on oral toxicity for 1,2-DCE in humans, or on the relative toxicities of the cis and trans isomers in humans. Mortality in animals exposed orally to cis- and trans-1,2-DCE involve central nervous system depression (ATSDR, 1995a). Results of acute studies in which animals have inhaled or ingested trans-1,2-DCE implicate the heart, liver, and lung as potential targets for toxicity. Acute exposure of the skin causes irritation and other mild skin effects that are readily reversible.

C. Subacute and Chronic Toxicity

No data available.

D. Carcinogenicity

To date, cancer effects of cis- and trans-1,2-DCE have not been studied in humans or animals (ATSDR, 1995a).

E. Mutagenicity

1,2-DCE has been examined in a variety of test systems. *In vivo* tests indicate that cis-1,2-DCE, but not trans-1,2-DCE, is genotoxic (ATSDR, 1995a). Genotoxic effects of cis- and trans-1,2-DCE in humans are unknown.

F. Teratogenicity/Reproductive Effects

No information was located.

G Other Health Effects

Pathological lesions of the heart (3,000 ppm for 8 hours), liver (200 ppm for 8 hours), and lung (200 ppm for a single exposure) have been reported

in rats exposed to trans-1,2-DCE in air. Trans-1,2-DCE is an ocular irritant in humans.

H. Epidemiological Evidence

No data available.

I. Toxicity

USEPA has classified cis-1,2-DCE and trans-1,2-DCE as not classifiable as to human carcinogenicity based on no data in humans or animals. The chronic oral reference dose for cis-1,2-DCE is 0.01 mg/kg-day. The aforementioned toxicity value was taken from USEPA, 2004b. The chronic oral reference dose for trans-1,2-DCE is 0.02 mg/kg-day. The aforementioned toxicity value was taken from USEPA, 2004a.

1.3 1,2-DICHLOROPROPANE (1,2-DCP)

1. Constituent Properties

A. Physical and Chemical Properties

Molecular Weight:	112.99
Melting Point:	-100.4°C
Boiling Point:	96.37°C
Specific Density:	1.16 (@ 20°C)
Water Solubility (mg/L):	2,700 (@ 20°C)
Vapor Pressure (mm Hg):	49.67 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	2.07 x 10^{-3} (@ 20°C)

Reference: ATSDR, 1989. Toxicological Profile for 1,2-Dichloropropane.

B. Chemical Transformation

Air: 1,2-DCP is photooxidized by reaction with photochemically produced hydroxyl radicals. The atmospheric life-time of 1,2-DCP is reported to be >23 days (ATSDR, 1989).

Water: Volatilization dominates the fate of 1,2-DCP in surface water. Half-lives have been reported up to 9 hours (ATSDR, 1989).

Soil: 1,2-DCP is expected to partition to the atmosphere when applied to surface soil. 1,2-DCP is not expected to sorbed to soil and as such leaching to groundwater is a predominant fate in soils based on the high solubility (ATSDR, 1989).

2. Toxicological Properties

A. Metabolism

There are no studies regarding metabolism of 1,2-DCP in humans. In laboratory animals, 1,2-DCP is readily and extensively absorbed by the gastrointestinal tract following oral and inhalation exposure. 1,2-DCP is

primarily metabolized to N-acetyl-S-(2-hydroxyporpyl)cysteine and is rapidly excreted in the urine, feces and expired air (ATSDR, 1989).

B. Acute Toxicity

Available information regarding the health effects of 1,2-DCP in humans and animals report the central nervous system, liver and kidneys as the primary target organs however; respiratory and hemetopoietic system alterations were also observed (ATSDR, 1989). The health effects in humans that were exposed to 1,2-DCP after the accidental spill of 2000 gallons were reported to be chest discomfort, dyspnea, cough, and some complained of chest pain or discomfort and fatigue (USEPA, 1991b). There are two reported cases of intoxication following 1,2-DCP exposure. The first case resulted in severe renal failure, acute liver damage, hemolytic anemia, disseminated intravascular coagulation. Symptoms included abdominal pain, vomiting, fever, facial edema and erythema. The second case resulted in severe liver failure, hemolytic anemia, and slight disseminated intravascular coagulation. In both cases, the effects were reversed once exposure was ceased and with treatment (USEPA, 1991b). The few deaths observed in humans as a result of intentional ingested of 1,2-DCP, reported that ultimate cause of death to be cardiac arrest and septic shock (ATSDR, 1989).

C. Subacute and Chronic Toxicity

The available animal studies have reported that the liver is the primary target organ for toxic effects of 1,2-DCP. As well, hematological and central nervous system effects were observed in the studies (ATSDR, 1989).

D. Carcinogenicity

USEPA has not evaluated the carcinogenicity of 1,2-DCP (USEPA, 1991b).

E. Mutagenicity

Available genotoxicity studies conducted with bacteria, fungus, and mammalian cell lines suggest that 1,2-DCP to be mutagenic (ATSDR, 1989).

F. Teratogenicity/Reproductive Effects

Animal studies indicate that fetotoxicity, developmental effects and reproductive effects are found at doses at or nearing dosages that adversely affect the blood and liver (USEPA, 1991b). Animals exposed orally to 1,2-DCP resulted in inflammation of the uterus and ovary, as well as hyperplasia of the mammary gland (USEPA, 1991b). Developmental effects were reported in animal studies to include delayed ossification of the bones (ATSDR, 1989).

G. Other Health Effects

Animal studies have reported 1,2-DCP to be a skin and eye irritant. Chronic dermal exposure to 1,2-DCP in aerosol form resulted in dermatitis (ATSDR, 1989).

H. Epidemiological Evidence

Case studies of humans exposed to 1,2-DCP confirm it primarily effects the central nervous system, liver and kidneys. There is only one occupational exposure reported, where women were dermally exposed to liquid 1,2-DCP and developed dermatitis (ATSDR, 1989).

I. Toxicity

1,2-DCP has not been evaluated for carcinogenicity. The chronic oral cancer slope factor is 0.068 (mg/kg-day)-1 (USEPA, 1997). The chronic inhalation reference dose is 0.00114 mg/kg-day (USEPA, 2004a).

1.4 METHYL ETHYL KETONE (MEK, 2-BUTANONE)

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	72.11
Melting Point:	79.6°C
Boiling Point:	-86.3°C
Specific Density:	0.8054 (@ 20°C)
Water Solubility (mg/L):	136,000 (@ 25°C)
Vapor Pressure (mm Hg):	90.6 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	5.77 x 10^{-5} (@ 25°C)

Reference: ATSDR, 1992a. Toxicological Profile for 2-Butanone.

B. Chemical Transformation

Air: MEK is expected to undergo atmospheric destruction by reaction with photochemically produced hydroxyl radicals (ATSDR, 1992a).

Water: Numerous investigations have concluded that MEK is removed from water by microbial degradation under both aerobic and anaerobic conditions (ATSDR, 1992a). One byproduct of MEK degradation is propionate. In a study of biodegradation potential using anaerobic biotechnology, MEK was completely reduced to methane within 8 days (ATSDR, 1992a).

Soil: MEK is expected to degrade under aerobic and anaerobic conditions (ATSDR, 1992a).

2. Toxicological Properties

A. Metabolism

Two metabolites identified in human urine after inhalation exposure are 3-hydroxy-2-butanone and 2,3-butanediol, but the extent of metabolism appears to be small (ATSDR, 1992a). The urinary concentration of these metabolites represents only about 0.1 percent (%) to 2% of the absorbed MEK. 2-Butanol has been reported to be a metabolite identified in the blood of male volunteers (ATSDR, 1992a).

B. Acute Toxicity

As with other small molecular weight, aliphatic or aromatic chemicals, acute exposure to high concentrations of MEK results in reversible central nervous system depression. Data from a series of NIOSH-sponsored studies involving acute, 4-hour exposures of volunteers found no exposure-related changes in performance of psychomotor and mood tests or incidences of irritation. Evidence for neurotoxic effects in humans repeatedly exposed to MEK is limited to a few case reports of neurological impairment in workers (USEPA, 2003a).

C. Subacute and Chronic Toxicity

Chronic inhalation exposure resulted in minimal to mild lesions in the upper or lower respiratory tract of MEK-exposed rats and were coded as chronic respiratory disease consisting of "multifocal accumulation of lymphoid cells in the bronchial wall and peribronchial tissues with occasional polymorphonuclear cells (eosinophils) in the perivascular areas of small veins". In addition, the study reported an increased prevalence of nasal inflammation (including submucosal lymphocytic infiltration and luminal exudate) across control and all exposure groups. Other effects noted include reduced body weight gain, statistically significant increases in relative liver weight (males and females) and altered serum liver enzymes (females), and decreased brain weight (females). It was noted in the report, reported liver effects are more likely indicative of a physiological adaptive response than toxicity. The finding of decreased brain weight observed in female rats raises concerns, but was difficult to interpret (USEPA, 2003a).

D. Carcinogenicity

Under the final draft revised guidelines for carcinogen risk assessment, USEPA concludes the *data are inadequate for an assessment of human carcinogenic potential* of MEK. Studies of humans chronically exposed to MEK are inconclusive, and MEK has not been tested for carcinogenicity in animals by the oral or inhalation routes (USEPA, 2003a).

E. Mutagenicity

MEK has not exhibited mutagenic activity in a number of conventional short-term test systems. *In vitro* tests showed that MEK was not

genotoxic in the Salmonella assay, mouse lymphoma assay, and the BALB/3T3 cell transformation assay, and did not induce unscheduled DNA synthesis in rat primary hepatocytes, chromosome aberrations, or sister chromatic. No induction of micronuclei was found in the erythrocytes of mice or hamsters after intraperitoneal injection with MEK. In general, studies of MEK yielded little or no evidence of mutagenicity (USEPA, 2003a).

F. Teratogenicity/Reproductive Effects

A study was conducted on the multigenerational reproductive and developmental toxicity of 2-butanol. Identification of the critical effect for MEK is based on its metabolic precursor, 2-butanol. The available pharmacokinetic and toxicologic data support the use of 2-butanol as an appropriate surrogate for MEK. In summary, the results of the study demonstrate that the administration of 2-butanol in drinking water to rats at a concentration of 3% produced maternal toxicity accompanied by developmental effects, but did not affect reproductive performance (with the possible exception of effects on male rat copulatory success). Decreased parental weight gain prior to mating, decreased pup survival, and decreased pup weights among survivors at postnatal days 4 and 21 were observed in the groups exposed to 3% 2-butanol in the drinking water. At the 2% level, the following effects were noted: decreased maternal body weight gain during the second pregnancy of dams, decreased fetal weights when pregnancy was terminated at gestation day 20, and decreased pup weights at postnatal days 4 and 21. At the next lower dose level (1%), reduced pup weight was observed, but the reduction was not observed in subsequent generations at the same exposure level. Developmental endpoints were not affected at the 0.3% exposure levels in any of the generations. The finding of developmental toxicity in rats exposed orally to 2-butanol in the study is consistent with inhalation developmental toxicity studies (fetal weight deficits) of MEK. Inhalation exposure of experimental animals to approximately 3,000 ppm MEK (7 hours/day) during gestation resulted in developmental effects (USEPA, 2003a).

G. Other Health Effects

No data available.

H. Epidemiological Evidence

The main neurological complaints of humans exposed occupationally to MEK are headaches, dizziness, nausea, and fatigue. However, these symptoms were not reported in a study where volunteers were exposed to 200 ppm of MEK for 4 hours (ATSDR, 1992a). Two retrospective epidemiological studies of industrial workers chronically exposed to MEK in dewaxing plants reported that deaths due to cancer were less than expected (ATSDR, 1992a).

I. Toxicity Data

USEPA has not classified MEK, as to human carcinogenicity due to inadequate or no evidence of carcinogenicity in animal studies. The chronic reference doses are 0.6 and 1.4 mg/kg-day for the oral and inhalation routes, respectively. All of the aforementioned toxicity values were taken from USEPA, 2004a.

1.5 4-METHYL-2-PENTANONE (METHYL ISOBUTYL KETONE - MIBK)

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	100.16
Melting Point	-85°C
Boiling Point:	115.8°C
Specific Density:	0.8024 (@ 20°C)
Water Solubility (mg/L):	19,000 (@ 25°C)
Vapor Pressure (mm Hg):	19.9 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	1.38×10^{-4}

Reference: Hazardous Substances Databank (HSDB, 2004).

B. Chemical Transformation

Air: Vapor-phase MIBK is degraded in the atmosphere by reaction with photochemically produced hydroxyl radicals; the half-life is estimated to be 27 hours (HSDB, 2004).

Water: MIBK is not likely to adsorb to particulates in water. The predominant fate of MIBK is volatilization from surface waters and biodegradation (HSDB, 2004).

Soil: Volatilization from soil surfaces is a predominant fate of MIBK. Biodegradation is expected to be an important environmental fate under both aerobic and anaerobic conditions (HSDB, 2004).

2. Toxicological Properties

A. Metabolism

Ketones are readily absorbed through intact skin or from the lungs into the bloodstream. MIBK is metabolized by oxidation to metabolites 4-hydroxy-2-methylpentanone and 4-methyl-2-pentanol. Ketones are rapidly excreted in expired air as the parent compound or its metabolites excreted in the urine (HSDB, 2004).

B. Acute Toxicity

The most likely exposures by humans are by inhalation of vapors and by skin and eye contact. Exposure to 50 to 105 ppm for 15 to 30 minutes provoked gastrointestinal disturbances and central nervous system

impairment in workers. One group of workers exposed to 100 ppm MIBK developed headaches and nausea, whereas another group complained of respiratory tract irritation. Tolerance to MIBK increased during the work week but lost over the weekend (HSDB, 2004).

One study where guinea pigs were exposed to MIBK, reported respiratory effects due to central nervous system depression. Eye and nose irritation was also reported followed by salivation, lacrimation, ataxia and death at a high dosage of 16,000 ppm. The highest dosage (28,000 ppm) caused death in 50% of the animals within 45 minutes. Fatty livers, congestion of the brain, lungs and spleen were noted.

Undiluted MIBK (0.1 mL) produced some irritation within 10 minutes when applied to the rabbit eye. Inflammation and swelling occurred in 8 hours, and inflammation, swelling, and exudate were present at 24 hours. A single application of MIBK to the skin of rabbits produced only transient erythema, but daily applications of 10 mL for 7 days caused drying and flaking of the skin (HSDB, 2004).

C. Subacute and Chronic Toxicity

Although the relative toxicity of most ketones is low and the effects of acute exposures are well recognized, the effects of chronic exposure are less well understood. It was reported that an individual developed cerebral dysfunction as indicated by blurred vision, dysarthria, nystagmus, slight intention tremor, staggering gait and abnormal electroencephalogram after an 8-month period of sniffing a lacquer thinner containing MIBK (HSDB, 2004).

A sub-chronic inhalation study in animals exposed to 100 ppm of MIBK reported liver and kidney weights increase (HSDB, 2004). Effects associated with adverse changes in the liver and kidney generally occurred at lower concentrations than neurological effects in repeated exposure animal studies. The principal effects associated with neurological impairment in animals were behavioral changes (e.g., hypoactivity, ataxia, and unsteady gait) that were only observed during exposure events in repeated exposure studies. Two subchronic oral exposure studies: one 13-week gavage exposure study and one 90-day *ad libitum* drinking water study reported kidney and liver effects associated with concentrations at approximately 1,000 mg/kg-day in both studies and at 250 mg/kg-day in the gavage study (USEPA, 2003b).

D. Carcinogenicity

Under the USEPA draft revised cancer guidelines, the data for MIBK are inadequate for an assessment of human carcinogenic potential. No data were located regarding the existence of an associated between cancer and MIBK exposure in humans or animals (USEPA, 2003b).

E. Mutagenicity

Studies of *in vivo* and *in vitro* genotoxicity tests of MIBK overwhelmingly provided negative responses (USEPA, 2003b).

F. Teratogenicity/Reproductive Effects

Animal studies have reported maternal toxicity and fetotoxicity when exposed to MIBK. Critical effects observed were skeletal variations in mice and rats and reduced fetal body weight and increased fetal death in mice. Several additional effects observed included hypoactivity, ataxia, and lacrimation in mouse and rat dams and reduced maternal body weight and body weight gain in rats (USEPA, 2003b).

In a two-generation reproductive toxicity assay in rats, no neonatal developmental effects (number of offspring with gross external malformations at birth, number of stillbirths, number of live births, body weight on post-natal day 1, or survival to post-natal day 4) were seen in either generation of rats exposed to air concentrations of MIBK up to 8,219 mg/m^3 for 6 hours/day for 70 days prior to mating, throughout mating, and during most of gestation and lactation (USEPA, 2003b).

G. Other Health Effects

No data available.

H. Epidemiological Evidence

The sensory threshold of MIBK was studied in 12 men and women who were subjected to various concentrations of MIBK for 15-minute periods. The highest concentration that most subjects evaluated as satisfactory for 8-hour continuous exposure was 100 ppm. The MIBK odor was objectionable and the vapor was irritating to the eyes at 200 ppm (HSDB, 2004).

I. Toxicity Data

USEPA has not classified MIBK as to human carcinogenicity, due to inadequate evidence of carcinogenicity in studies. A chronic inhalation reference dose is reported to be 0.86 mg/kg-day for MIBK. The aforementioned toxicity value was taken from USEPA, 2004a.

1.6 ACETONE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	58.08
Melting Point:	-95.35°C
Boiling Point:	56.2°C
Specific Density:	0.7899 (@ 20°C)

Water Solubility (mg/L): miscible
Vapor Pressure (mm Hg): 181.72 (@ 20°C)
Henry's Law Constant (atm-m^3/mol): 4.26×10^{-5}
Reference: ATSDR, 1994a. Toxicological Profile for Acetone.

B. Chemical Transformation

Air: The two significant processes in determining the fate of acetone in the atmosphere are reaction with hydroxyl radicals and photolysis. The estimated half-life of atmospheric acetone was reported at 22 days due to hydroxyl radical reaction and photolysis. Products of acetone photolysis are carbon dioxide and acetylperoxynitrate (ATSDR, 1994a).

Water: Acetone primarily volatilizes when in aquatic sources. The volatilization half-life of acetone from water sources is reported to be within hours. Acetone is easily biodegradable with acclimatized microorganisms (ATSDR, 1994a)

Soil: The two significant transport mechanisms of acetone in soil are volatilization and leaching to groundwater. In dry soils the volatilization rate of acetone is much higher than in wet soils. Studies indicate that biodegradation of acetone in soils will be significant (ATSDR, 1994a).

2. Toxicological Properties

A. Metabolism

Acetone is readily absorbed by the lungs and gastrointestinal tract, taken up the blood and widely distributed to organs and tissues. Acetone is metabolized mainly in the liver, which involves three separate gluconeogenic pathways. Ultimately carbon atoms are incorporated into glucose and other products of intermediary metabolism, with the production of carbon dioxide and adenosine triphosphate. Acetone and its metabolites are excreted mainly in expired breath with very little acetone excreted in urine. Elimination of acetone is generally complete within 1 to 3 days (ATSDR, 1994a).

B. Acute Toxicity

As a solvent, acetone is irritating to mucous membranes and exposure to vapors irritates the respiratory system and eyes. Acetone has anesthetic properties and causes headaches, lightheadedness, confusion, dizziness, and can lead to unconsciousness and coma in humans at high exposure levels. Acute inhalation exposure of acetone can also lead to increased pulse rates, gastrointestinal irritation, nausea, vomiting and hemorrhage. Accidental or intentional ingestion of acetone caused erosions in the mouth, coma and diabetes-like symptoms (excessive thirst, frequent urination). Acute dermal exposure of humans to liquid acetone resulted in degenerative changes in the epidermis (ATSDR, 1994a).

C. Subacute and Chronic Toxicity

In a 6-week study with humans, no effects were found on respiratory, cardiovascular, hematological, hepatic and renal systems at concentrations <1,250 ppm. Chronic inhalation of acetone at concentrations greater than 1,006 ppm can again result in lung and throat irritation, headache and lightheadedness in workers. Intermediate-duration studies on rats have reported liver effects, hematological effects and nephropathy. However, these effects seem to be greater in males then females suggesting the male population might be more likely to have effects of exposure to acetone than women (ATSDR, 1994a).

D. Carcinogenicity

In accordance with the Draft Revised Guidelines for Carcinogen Risk Assessment (USEPA, 2003c), USEPA has classified acetone as data are inadequate for an assessment of the human carcinogenic potential of acetone. This weight-of-evidence determination is based on the availability of one human study of limited utility, no chronic animal studies, and no additional information on structural analogues with known carcinogenic potential (USEPA, 2003d).

E. Mutagenicity

The genotoxicity of acetone has been well studied *in vitro* assays, with the results almost entirely negative. All studies cited in the GENE-TOX data base were negative, with the exception of one study for which no conclusion was drawn (USEPA, 2003d).

F. Teratogenicity/Reproductive Effects

Only one human study suggested any reproductive effects to humans. Women exposed to 1,000 ppm of acetone reported their period came earlier than expected. Available studies on the reproductive or developmental effects of acetone to animals are limited. Studies where pregnant rats were exposed to acetone via inhalation or ingestion reported increased liver weights, lower body weights and produced fewer fetuses. Other studies reported findings of male rats had abnormal sperm when ingesting acetone (ATSDR, 1994a).

G. Other Health Effects

One effect of acetone seen in animals is an increase in chemicals in the body that help break down natural substances in the body and chemicals entering the body. The increase in enzymes caused by acetone exposure can make some chemicals more harmful such as halogenated alkanes and alkenes, benzene, dichlorobenzene, ethanol, nitrosamines, acetonitrile and acetaminophen (ATSDR, 1994a).

H. Epidemiological Evidence

Most of the information comes from medical exams or lab studies. Workers and volunteers exposed to acetone complained of irritation to their nose, throat, lungs and eyes. Some people felt the irritation at levels of 100 ppm of acetone in the air, while others felt the irritation at higher levels. Workers exposed to acetone at 12,000 ppm complained of headaches, dizziness, lightheadedness, unsteadiness, and confusion depending on length of exposure (from 2 minutes to 4 hours). Two workers exposed for 4 hours became unconscious. Typically, workers are not exposed to levels higher than 750 ppm of acetone due to current government regulations. However, the odor threshold of acetone (100 to 140 ppm) and the feelings of irritancy are warning properties that generally preclude inhalation over-exposure to acetone (ATSDR, 1994a).

I. Toxicity Data

USEPA has not classified acetone as to human carcinogenicity, due to inadequate evidence of carcinogenicity in studies. A chronic oral reference dose is reported to be 0.9 mg/kg-day for acetone. The aforementioned toxicity value was taken from USEPA, 2004a.

1.7 BENZENE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	78.11
Boiling Point:	80.1°C
Melting Point:	5.5°C
Water Solubility (mg/L):	1,780 (@ 25°C)
Vapor Pressure (mm Hg):	95.2 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	5.5×10^{-3}

Reference: ATSDR, 1997a. Toxicological Profile for Benzene.

B. Chemical Transformation

Air: Benzene in the atmosphere predominantly exists in the vapor phase. The most significant degradation process for benzene is its reaction with hydroxyl radicals. Half-lives for benzene in the atmosphere have been reported from hours to days. Some of the products of the reaction of benzene with nitrogen dioxide include nitrobenzene, o- and p-nitrophenol, and 2,4- and 2,6-dinitrophenol (ATSDR, 1997a).

Water: Benzene is biodegradable in surface water and groundwaters. Microbial degradation in aquatic sources is influenced by many factors such as microbial population, dissolved oxygen,

nutrients and other sources of carbon, inhibitors, temperature and pH. Half-lives for benzene in surface water and groundwater of 16 and 28 days, respectively were reported (ATSDR, 1997a).

Soil: The predominant fate of benzene released to soils would be volatilization. Other fates of benzene in soils include leaching to groundwater, adherence to soil particles and degradation. A model developed to predict the fate of benzene following leakage of gasoline to soils reported 67% of benzene would volatilize, 29% would leach to groundwater, 3% remain in the soil and 1% would be degraded (ATSDR, 1997a).

2. Toxicological Properties

A. Metabolism

The liver is the major site of benzene metabolism. Most of the available evidence suggests that benzene toxicity is produced by one or more metabolites rather than by benzene itself. Major metabolites of benzene are phenolic compounds (i.e., phenol, hydroquinone, and catechol). Conjugated phenolic metabolites appear in the urine mainly as etheral sulfates and glucuronides. Benzene can be stored in the bone marrow, liver, and body fat. The respiratory elimination of benzene in humans is described as triplastic. The initial component has a half-life of ~0.9 hours. The second, slower phase has a half-life of 3 hours, and the third has a half-life of >15 hours (ATSDR, 1997a).

B. Acute Toxicity

Brief exposure (5 to 10 minutes) to extremely high levels of benzene (10,000 to 20,000 ppm) has been reported to result in death. At lower levels, acute inhalation exposure can cause drowsiness, dizziness, rapid heart rate, headaches, tremors, confusion, and unconsciousness. In most cases, symptoms are reduce when exposure is stopped. Ingestion of benzene at high levels may result in vomiting, gastrointestinal irritation, dizziness, drowsiness, rapid heart rate, coma and death. Dermal exposure has been reported to cause redness and sores. Ocular exposure to benzene may cause general irritation and cornea damage (ATSDR, 1997a).

C. Subacute and Chronic Toxicity

The most noted systemic effect resulting from intermediate and chronic benzene exposure is hematotoxicity. Hematoxicity is harmful effects in the tissues that form blood cells, especially in the bone marrow. These effects can disrupt normal blood production and cause a decrease in important blood components. A decrease in red blood cells can lead to anemia. Reduction in other components in the blood may cause excessive bleeding. Excessive exposure to benzene have been reported to be

harmful to the immune system, increasing the chance for infection and perhaps lowering the body's defense against cancer (ATSDR, 1997a).

D. Carcinogenicity

Under the proposed revised Carcinogen Risk Assessment Guidelines (USEPA, 2003c), benzene is characterized as a known human carcinogen for all routes of exposure based upon convincing human evidence as well as supporting evidence from animal studies. Significantly increased risks of leukemia, chiefly acute myelogenous leukemia (AML), have been reported in benzene-exposed workers in the chemical industry, shoemaking, and oil refineries (USEPA, 2003e).

E. Mutagenicity

Data from both humans and animals indicate that benzene and/or its metabolites are genotoxic. Chromosomal aberrations in peripheral lymphocytes and bone marrow cells are the predominant effects seen in humans (ATSDR, 1997a).

F. Teratogenicity/Reproductive Effects

There is some evidence from human epidemiological studies of reproductive and developmental toxicity of benzene, but the data did not provide conclusive evidence of a link between exposure and effects. Some test animal studies provide limited evidence that exposure to benzene affects reproductive organs; however, these effects were limited to high exposure concentrations that exceeded the maximum tolerated dose. Results of inhalation studies conducted in test animals are fairly consistent across species and have demonstrated that at concentrations of greater than 150 mg/m^3 (47 ppm) benzene is fetotoxic and causes decreased fetal weight and/or minor skeletal variants. Exposure of mice to benzene in utero has also been shown to cause changes in the hematogenic progenitor cells in fetuses, 2-day neonates, and 6-week-old adults (USEPA, 2003e).

G. Other Health Effects

Both gastrointestinal (i.e., toxic gastritis and pyloric stenosis) and dermal effects (i.e., swelling and edema) have been reported to occur in a human who has swallowed benzene. Chronic oral and inhalation exposure to benzene has been associated with distal neuropathy in humans, difficulty in sleeping and memory loss (ATSDR, 1997a).

H. Epidemiological Evidence

Many epidemiological and case studies correlate benzene exposure with leukemia (USEPA, 2003e).

I. Toxicity Data

USEPA has classified benzene as a human carcinogen with sufficient evidence in human epidemiological studies. The chronic cancer slope

factors are 0.055 (mg/kg-day)-1 and 0.027 (mg/kg-day)-1 for the oral and inhalation routes, respectively. The chronic reference doses are 0.004 and 0.0086 mg/kg-day for the oral and inhalation routes, respectively. All of the aforementioned toxicity values were taken from USEPA, 2004a.

1.8 CARBON DISULFIDE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	76.14
Boiling Point:	46.5°C
Melting Point:	-111.5°C
Density:	1.2632 (@ 20°C)
Water Solubility (mg/L):	2,940 (@ 20°C)
Vapor Pressure (mm Hg):	260 (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	1.22 x 10^{-2}

Reference: ATSDR, 1996a. Toxicological Profile for Carbon Disulfide.

B. Chemical Transformation

Air: The main degradation pathway for carbon disulfide is reaction with hydroxyl radicals and may include the following by-products: carbonyl sulfide, carbon monoxide and sulfur dioxide (ATSDR, 1996a).

Water: Carbon disulfide is stable to hydrolysis and oxygenation in surface waters. The compound apparently does not undergo biodegradation at rates that are competitive with its volatilization from surface waters (ATSDR, 1996a).

Soil: Carbon disulfide volatilizes rapidly and is highly mobile in soil, and therefore, it is unlikely that it remains in the soil long enough to be significantly biodegraded (ATSDR, 1996a).

2. Toxicological Properties

A. Metabolism

In animals and humans the proposed metabolic pathways involved in the metabolism of carbon disulfide are not completely known. Metabolism studies in animals clearly indicate that there are two distinctly different pathways: it can form dithiocarbamates and glutathione conjugates, or it can be metabolized to generate reactive sulfur. Both metabolic pathways suggest several potential mechanisms of toxicity. Appreciable amounts of carbon disulfide are excreted unchanged in breath regardless of route of exposure (ATSDR, 1996a).

B. Acute Toxicity

Sufficient animal data have reported the central nervous system, cardiovascular system and the liver as target organs after inhalation exposure. Lethality, decreased respiratory rate, and increased post-implantation loss have also been reported in acute inhalation studies. Three case reports indicate that half an ounce of carbon disulfide caused death after ingestion. Animal studies identified the cardiovascular system and the liver as target organs after acute oral exposure. Adverse effects include decreased body weight gain, hind-limb paralysis, and decreased norepinephrine levels. Dermal exposure to carbon disulfide can lead to burns at the contact site. Workers in a rayon plant who handled fibers made with carbon disulfide for more than 14 days developed blisters on their fingers. Rabbits developed blisters and ulcers on the treated areas of the ears (ATSDR, 1996a).

C. Subacute and Chronic Toxicity

The primary target organs for carbon disulfide is the nervous system, cardiovascular system, the liver and the eye. It is clear in human and animal studies that the nervous system is the primary target organ for carbon disulfide exposure for the inhalation route. There are behavioral, histopathological and neurophysiological effects. Several human studies have reported increased incidence of elevated blood pressure indicating cardiovascular effects. In humans, inhalation and oral exposure to carbon disulfide causes inhibition of microsomal enzymes and enlarged livers. Ophthalmologic changes were noted in workers exposed to chronic low levels of carbon disulfide. Effects include retinal micro-aneurysms, retinopathy, and burning eyes (ATSDR, 1996a).

D. Carcinogenicity

This substance has not undergone a complete evaluation and determination under USEPA's IRIS program for evidence of human carcinogenic potential (USEPA, 1995a).

E. Mutagenicity

Most studies reviewed regarding mutagenic toxicity from carbon disulfide exposure were negative. There are no mutagenicity data on humans exposed occupationally to carbon disulfide, so that at this time it is difficult to predict effects of carbon disulfide at the DNA level (ATSDR, 1996a).

F. Teratogenicity/Reproductive Effects

Decreased sperm count and decreased libido in men and menstrual irregularities in women exposed to carbon disulfide in the workplace have been the most frequently reported effects. However, community and workplace studies have not shown a decrease in fertility rate, an increase in the time between live births, or an effect on semen quality

with carbon disulfide exposure (ATSDR, 1996a). Carbon disulfide has been reported to cause malformations in the offspring of rats exposed via inhalation, in addition; neurobehavioral effects have been noted in offspring of exposed animal mothers. There is some evidence that carbon disulfide will cross over the placenta membrane and distribute to the brain, blood, liver, and eyes of fetuses (ATSDR, 1996a).

G. Other Health Effects

Gastrointestinal symptoms are commonly reported in workers exposed to carbon disulfide. Symptoms include nausea, vomiting, stomach distress, and loss of appetite (ATSDR, 1996a).

H. Epidemiological Evidence

There are many epidemiological studies that address the effects of inhalation exposure to carbon disulfide. These studies include occupational- and community-based cohorts and have reported positive association between carbon disulfide exposure and neurological and cardiovascular adverse health effects (ATSDR, 1996a).

I. Toxicity Data

USEPA has not assessed carbon disulfide as to human carcinogenicity potential. The chronic reference doses are reported at 0.1 and 0.2 mg/kg-day for both oral and inhalation routes, respectively. The aforementioned toxicity values were taken from USEPA, 2004a.

1.9 CHLOROBENZENE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	112.56
Boiling Point:	132°C
Melting Point:	-45.6°C
Specific Density:	1.1058 (@ 20°C)
Water Solubility (mg/L):	500 (@ 20°C)
Vapor Pressure (mm Hg):	9 (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	3.93 x 10^{-3}

Reference: ATSDR, 1990a. Toxicological Profile for Chlorobenzene.

B. Chemical Transformation

Air: Air is an important and perhaps dominant medium for transport and transformation of chlorobenzene.

Water: Biodegradation is the major degradative process in oxygenated waters while evaporation will play an additional removal role.

Soil: Biodegradation plays an important role.

2. Toxicological Properties

A. Metabolism

The main metabolites of chlorobenzene are p-chlorophenylmercapturic acid and 4-chlorocatechol. The excretion of p-chlorophenylmercapturic acid was markedly less than that of 4-chlorocatechol in humans who received chlorobenzene orally or by inhalation. Pharmacokinetic modeling of the respiratory elimination kinetics of chlorobenzene revealed a biphasic elimination pattern, with a fast phase of up to 4 hours post-exposure and a slower phase lasting longer than 48-hours (ATSDR, 1990a).

B. Acute Toxicity

No case studies of human fatalities have been reported following exposure to chlorobenzene by inhalation, ingestion, or dermal contact (ATSDR, 1990). Deaths have been reported in animals following acute exposures to extremely high doses. For example, cats died within 2 hours after removal from exposure to 8,000 ppm and after 7 hours at 3,700 ppm. This animal data suggest that lethality may not be a concern for humans unless the exposure level is very high (ATSDR, 1990a).

No studies were located demonstrating hepatic toxicity of chlorobenzene in humans by any route of exposure. Acute and intermediate exposures in animals demonstrated that chlorobenzene caused changes in liver weights and enzyme levels, degeneration, necrosis, and alterations in microsomal enzymes (ATSDR, 1990a).

Acute studies in animals confirm that chlorobenzene is potentially neurotoxic. Narcosis was reported at 1,200 ppm and restlessness, tremor and muscular spasms at 2,400 to 2,900 ppm. Unconsciousness, lack of response to skin stimuli, and muscle spasms were noted following accidental ingestion (ATSDR, 1990a).

C. Subacute and Chronic Toxicity

No studies were located demonstrating that chlorobenzene causes renal effects in humans by any route of exposure. Intermediate studies in animals showed effects on the kidney at doses comparable to those causing liver effects. These effects were evident in animals exposed to 75 ppm chlorobenzene via inhalation for 5 days/week for 24 weeks. Similar effects were observed in animals that ingested chlorobenzene at >250 mg/kg/day for 91 days. Typical effects included tubular degeneration and necrosis as well as changes in organ weight. Based on animal studies, renal toxicity may be an area of concern in humans (ATSDR, 1990a).

Case reports of humans demonstrated that chlorobenzene caused disturbances of the central nervous system, but there were no reports of changes in the structure of the brain and other parts of the nervous system. Effects were observed in humans who inhaled vapors of

chlorobenzene in the workplace for up to 2 years. Effects included headaches, dizziness, and sleepiness. While there is qualitative evidence for central nervous system effects in humans, a quantitative assessment can not be made since exposure levels were not reported (ATSDR, 1990a).

D. Carcinogenicity

No studies were found regarding the carcinogenicity of chlorobenzene in humans. In a chronic bioassay in animals, chlorobenzene (up to 120 mg/kg-day) did not produce increased tumor incidence in mice of both sexes or in female rats. It was noted, however, that male rats showed a statistically significant increase in neoplastic nodules of the liver (ATSDR, 1990a). While there is strong evidence for neoplastic nodules developing into carcinomas, existing data are inadequate to characterize the potential for chlorobenzene to cause cancer in humans (ATSDR, 1990a). USEPA has classified chlorobenzene as Group D, not classifiable as to human carcinogenicity due to inadequate or no evidence.

E. Mutagenicity

No data available.

F. Teratogenicity/Reproductive Effects

No data available.

G. Other Health Effects

No data available.

H. Epidemiological Evidence

No data available.

I. Toxicity

Chlorobenzene is classified by the USEPA as Group D, not classifiable as a human carcinogen based on insufficient evidence in animal studies. The chronic oral reference dose is 0.02 mg/kg-day (USEPA, 2004a) and the chronic inhalation reference dose is 0.017 mg/kg-day (USEPA, 2004b).

1.10 ETHYLBENZENE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	106.17
Boiling Point:	136.2°C
Melting Point:	-95°C
Specific Density:	0.867 (@ 20°C)
Water Solubility (mg/L):	152 (@ 20°C)

Vapor Pressure (mm Hg): 7.0 (@ 20°C)
Henry's Law Constant (atm-m^3/mol): 6.6×10^{-3} (@ 20°C)
Reference: ATSDR, 1999a. Toxicological Profile for Ethylbenzene.

B. Chemical Transformation

Air: Ethylbenzene undergoes atmospheric transformations through the reaction with photolytically-generated hydroxyl radicals. Atmospheric half-life has been reported at less than 3 days. Oxidation byproducts from the reaction with hydroxyl radicals and nitrogen oxides include ethylphenols, benzaldehyde, acetophenone, and m- and p-nitroethylbenzene (ATSDR, 1999a).

Water: Transformations in surface water may occur through photooxidation and biodegradation. The byproducts of the photooxidation reaction include 1-phenylethanone, 1-phenylethanol, and benzaldehyde (ATSDR, 1999a).

Soil: Aerobic biodegradation of ethylbenzene in soils has been reported to produce phenylacetic acid, 1-phenylethanol, fumaric acid, and acetoacetic acids, among others. Biodegradation in soil will compete with other migration processes such as volatilizing to the atmosphere and leaching to groundwater (ATSDR, 1999a).

2. Toxicological Properties

A. Metabolism

The metabolism of ethylbenzene has been studied in humans and other mammalian species. The data demonstrate that ethylbenzene rapidly undergoes a complex series of biotransformations from which numerous metabolites have been isolated. The major urinary metabolites have been traced to the liver, and evidence suggests that the adrenal cortex may be a major site for extra-hepatic ethylbenzene metabolism. Major metabolites include 1-phenylethanol, mandelic acid, phenylglyoxylic acid, and acetophenone. It's been reported that most of ethylbenzene and its metabolites leave in the urine within 2 days (ATSDR, 1999a).

B. Acute Toxicity

The primary symptoms resulting from acute exposure to ethylbenzene in animals are neurological and respiratory depression. Several studies suggest that the target organs of ethylbenzene toxicity in animals may be the liver, kidney, and hematopoietic system, however these results are inconclusive. No deaths have been reported in humans following ethylbenzene exposure, but death has occurred in laboratory animals following acute exposure to high levels of ethylbenzene administered via the inhalation, oral and dermal routes. The dose of ethylbenzene necessary to cause death in animals have been shown to be relatively high (1,200 to 13,367 ppm, inhalation exposure; 4728 mg/kg-day, oral

exposure; 15,415 mg ethylbenzene/kg body weight, dermal exposure) (ATSDR, 1999a).

The principal effect in humans acutely exposed to high concentrations (460 to 1,200 ppm) of ethylbenzene has been central nervous system toxicity (dizziness, vertigo). Complete recovery has been shown to occur if the exposure is not prolonged. Moderate upper respiratory irritation accompanied by chest constriction has been reported in humans exposed by inhalation to ethylbenzene concentrations as low as 460 ppm. Symptoms become more extreme following exposure to higher doses (ATSDR, 1999a).

C. Subacute and Chronic Toxicity

Exposure to ethylbenzene may result in hepatic and renal effects. Animal studies have reported biochemical changes, changes in liver weight, and histopathological alterations in the liver. Renal effects, manifested as enzyme changes, increase in organ weight, tubular swelling or hyperplasia, have been reported in animal studies. These studies suggest that chronic exposure to ethylbenzene may result in liver or kidney effects for humans (ATSDR, 1999a).

D. Carcinogenicity

No association between increased cancer incidence in humans and exposure to ethylbenzene has been reported in current literature. Two long-term studies in animals suggest ethylbenzene may cause tumors. On study had many weaknesses, therefore conclusions could not be drawn. The second study provided clear evidence that ethylbenzene cause cancer in one species after inhalation exposure (ATSDR, 1999a). At the present moment, USEPA has included ethylbenzene in Group D, not classified as to human carcinogenicity due to inadequate or no evidence (USEPA, 1991c).

E. Mutagenicity

Available data indicate that ethylbenzene is not mutagenic in bacteria, yeast cells, or hamster cells. It has, however, caused mutagenic effects in mouse lymphoma cells and other tests. Although the majority of data suggest that ethylbenzene is not mutagenic, the few positive results suggest that ethylbenzene may cause an increase potential for genotoxicity in humans (ATSDR, 1999a).

F. Teratogenicity/Reproductive Effects

No human studies were located. Animal studies indicate that inhalation exposure of pregnant rats to ethylbenzene can produce fetotoxic effects. The relevance of these findings with regard to developmental effects in humans cannot be ascertained. There are studies that suggest that adverse reproductive effects might occur in women exposed to ethylbenzene in the workplace or near hazardous waste sites. However, no definitive conclusions can be drawn from these studies. Oral

administration to female rats resulted in blockage of the estrus cycle. Decreased fertility was reported in female rats after inhalation exposure to ethylbenzene. One study in male rats reported an increase in interstitial cell adenoma and bilateral testicular ademona. Therefore, reproductive effects may be a target for ethylbenzene toxicity for both males and females (ATSDR, 1999a).

G. Other Health Effects

No data available.

H. Epidemiological Evidence

Little information concerning human exposure to ethylbenzene is available. Most of the information concerning health effects is reported in occupations studies which are difficult to interpret given the limitations of the studies. No data were available concerning human health effects following oral or dermal exposures. Dermal effects in humans are limited to respiratory and/or ocular irritation after exposure to ethylbenzene vapor (ATSDR, 1999a).

I. Toxicity Data

USEPA has not classified ethylbenzene as to human carcinogenicity, due to inadequate evidence of carcinogenicity in studies. The chronic reference doses are reported at 0.1 and 0.29 mg/kg-day for both oral and inhalation routes, respectively. The aforementioned toxicity values were taken from USEPA, 2004a.

1.11 METHYLENE CHLORIDE (DICHLOROMETHANE - DCM)

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	84.93
Boiling Point:	40°C
Melting Point:	-95.1°C
Specific Density:	1.3182 (@ 25°C)
Water Solubility (mg/L):	16,700 (@ 25°C)
Vapor Pressure (mm Hg):	349 (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	2.03×10^{-3}

Reference: ATSDR, 2000a. Toxicological Profile for Methylene Chloride.

B. Chemical Transformation

Air: DCM undergoes atmospheric transformations through the reaction with photolytically-generated hydroxyl radicals. An average atmospheric half-life has been calculated to be 130 days (ATSDR, 2000a).

Water: DCM undergoes slow hydrolysis in water. Biodegradation may be an important fate process for DCM in water under both aerobic and anaerobic conditions (ATSDR, 2000a).

Soil: Most of the DCM released to soils will volatilize into the atmosphere. Degradation of DCM in soils was found to occur under both aerobic and anaerobic conditions. DCM has a low tendency to adsorb to particles, therefore leaching to groundwater is a potential fate for this analyte (ATSDR, 2000a).

2. Toxicological Properties

A. Metabolism

After oral or inhalation exposure, DCM is rapidly absorbed through the lungs and gastrointestinal tract. Dermal exposure results in absorption but at a slower rate. Once absorbed, DCM is rapidly metabolized in the liver in part to carbon monoxide. DCM is removed rapidly from the body primarily in expired air and urine, usually occurring with 48 hours after exposure (ATSDR, 2000a).

B. Acute Toxicity

The central nervous system is a potential target in humans and animals at exposure levels of 800 ppm or higher. Effects were also reported on the liver and kidney at concentrations of 25 ppm or greater. Acute inhalation exposure of humans (usually during paint stripping) has caused death. Exposure levels were not known and the biologic cause of death was not certain. No cases of death from ingestion of DCM have been observed in humans. High oral doses (2,100 mg/kg or greater) in animals has caused death (ATSDR, 2000a).

Human data are limited on the effects of DCM on the liver. However, the liver appears to be a major target organ following DCM exposure in animals. Histomorphological and biochemical changes of the liver occur following acute inhalation (6 hours to 7 days) at high dose levels (5,200 ppm) (ATSDR, 2000a). Studies in humans and animals indicate that the central nervous system is a potentially important target for DCM. For the most part, anesthetic responses were reported and effects subsided once exposure ceased. A decrease in behavioral performance and various psychomotor tasks was evident in humans acutely exposed to DCM (300 ppm or greater) in experimental studies (ATSDR, 2000a).

C. Subacute and Chronic Toxicity

Mortality risk was not increased in humans chronically exposed occupationally to 30 to 120 ppm DCM. Fatty changes and biochemical alterations (altered cytochrome levels) of the liver were also observed at lower DCM concentrations (100 ppm) for continuous, 24-hour intermediate-duration exposure (100 days). No studies were found regarding renal effects in humans by any route of exposure. In rats, DCM produced non-specific renal tubular and degenerative changes after

continuous intermediate-duration exposure to DCM vapors (100 to 5,000 ppm) (ATSDR, 2000a).

D. Carcinogenicity

USEPA classifies DCM as a probable human carcinogen (Group B2) with sufficient evidence of carcinogenicity in animal studies. This is based on studies where increased incidence of hepatocellular neoplasms and alveolar/bronchiolar neoplasms in male and female mice, and increased incidence of benign mammary tumors in both sexes of rats, salivary gland sarcomas in male rats and leukemia in female rats was reported (USEPA, 1995b).

E. Mutagenicity

No human studies were located. DCM has been evaluated in several animal assays for its potential to induce gene mutation and cause DNA damage, with many negative and some positive results (ATSDR, 2000a).

F. Teratogenicity/Reproductive Effects

No studies were located regarding developmental effects in humans after inhalation, oral, or dermal exposure. Animal studies demonstrated that inhalation of DCM vapors at concentrations of 1,250 ppm produced minor skeletal effects. Fetal weight was reduced and behavioral changes occurred in rat pups following exposure to 4,500 ppm methylene chloride. Although fetal body weights were decreased, the absence of other fetotoxic effects, including embryo lethality and major malformations, suggest that DCM is not likely to cause developmental effects and behavioral changes at levels encountered at hazardous waste sites (ATSDR, 2000a).

Data on reproductive effects in humans is limited. One case study reported genital pain, testicular atrophy, infertility, and low or abnormal sperm count in workers who inhaled vapors or had direct contact with DCM. A case-control occupational study reported a significant association between DCM exposure and spontaneous abortions. Uterine, ovarian and testicular atrophy were observed in rats following chronic exposure to DCM for 2 years at 4,000 ppm (ATSDR, 2000a).

G. Other Health Effects

Respiratory effects to include cough, breathlessness, chest tightness, and in a fatal case, pulmonary congestion with focal hemorrhage were observed in several occupational cases. Gastrointestinal effects (nausea and vomiting) were reported in several cases of occupational exposure to DCM. Direct contact with DCM causes intense burning and mild redness of the skin. In a workplace accident where an individual loss consciousness and partly fallen into an open vat of DCM, extended direct contact with the liquid resulted in severe burns of the skin and eyes (cornea) (ATSDR, 2000a).

H. Epidemiological Evidence

Epidemiology studies have not revealed a causal relationship between deaths due to cancer and occupational exposure to DCM. Asphyxia and eventually death occurred in a subject acutely exposed to a high concentration of DCM in the air. Exposure to lower concentrations affects primarily the central nervous system, signs and symptoms include dizziness, incoordination, loss of balance, unconsciousness, and decreased performance in tests of sensory and motor functions. General population may be exposed to DCM (i.e., paint removal, aerosol use) but levels would be low to not produce adverse neurological effects (ATSDR, 2000a).

I. Toxicity Data

USEPA (1995b) has classified DCM as a probably human carcinogen with sufficient evidence in animal studies. The chronic oral and inhalation cancer slope factors are 0.0075 (mg/kg-day)-1 and 0.00165 (mg/kg-day)-1, respectively. The chronic reference doses are 0.06 and 0.86 mg/kg-day for the oral and inhalation routes, respectively. The cancer slope factors and oral reference dose toxicity values were taken from USEPA, 2004a. The inhalation reference dose was taken from USEPA, 1997.

1.12 METHYL *TERT*-BUTYL ETHER (MTBE)

1. Constituent Properties

A. Physical and Chemical Properties

Molecular Weight:	88.15
Melting Point:	-109°C
Boiling Point:	55.2°C
Specific Density:	0.7405 (@ 20°C)
Water Solubility (mg/L):	4,800 (@ 20°C)
Vapor Pressure (mm Hg):	245 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	5.87 x 10^{-4} (@ 20°C)

Reference: ATSDR, 1996b. Toxicological Profile for Methyl Tert-Butyl Ether.

B. Chemical Transformation

Air: MTBE is photooxidized by reaction with photochemically produced hydroxyl radicals. The atmospheric life-time of MTBE is reported to be 3 to 6 days with acetone and tert-butyl formate reported as degradation products (ATSDR, 1996b).

Water: Volatilization dominates the fate of MTBE in surface water. In groundwater, MTBE is persistence due to its high solubility with little tendency to sorb to soil particles (ATSDR, 1996b).

Soil: MTBE predominantly partitions to the atmosphere or is transported to groundwater when released to soils. When released to subsurface soils, MTBE may be fairly persistent since it has been reported to have a low tendency for either abiotic or biotic degradation processes (ATSDR, 1996b).

2. Toxicological Properties

A. Metabolism

MTBE is rapidly absorbed from the respiratory and gastrointestinal tract. Much of the absorbed MTBE is rapidly excreted as unchanged MTBE in expired air. The remainder is metabolized to *tert*-butanol, formaldehyde, methanol, formic acid, carbon dioxide, 2-methyl-1,2-propanediol, and alpha-hydroxyisobutyric acid. MTBE and its metabolites distribute to tissues but show little tendency to accumulate. Metabolism of MTBE has been reported to not be route specific (ATSDR, 1996b).

B. Acute Toxicity

MTBE vapors are irritating to the eyes and upper respiratory tract. Once absorbed in may cause central nervous system and respiratory depression. Effects include malaise, dizziness, fatigue, headache, and lightheadedness, coughing and burning sensation of the nose and throat, all of which disappear soon after exposure cease. Dermal exposure to MTBE may result in defatting of the skin and subsequent dermatitis. Following ingestion, there is potential risk for aspiration leading to airway and pulmonary damage, (ATSDR, 1996b).

C. Subacute and Chronic Toxicity

Mortality in animals was reported in studies with the cause of death reported to be chronic progressive nephropathy in rats and obstructive uropathy in mice. Animal studies have reported central nervous system effects after inhalation, oral and dermal exposure which are characteristic of ether anesthesia and ethyl alcohol intoxication and include lacrimation, ataxia, loss of righting reflex, hyperpnea, labored breathing, incoordination, prostation, drowsiness, hypoactivity, decreased startle and pain reflexes, decreased muscle tone, anesthesia, blepharospasm, and stereotypy. Other effects reported include increase liver and kidney weights, induction of liver and kidney microsomal enzymes, gastrointestinal irritation, and decreased BUN levels (ATSDR, 1996b).

D. Carcinogenicity

The availability of carcinogenic studies of MTBE indicate it to produce tumors in multiple species of animals, however; USEPA has not yet

reviewed MTBE to its carcinogenic potential (ATSDR, 1996b; USEPA, 1993a).

E. Mutagenicity

Most of the *in vitro* and *in vivo* animal studies reported negative results indicating MTBE to have very little genotixicity activity (ATSDR, 1996b).

F. Teratogenicity/Reproductive Effects

Reproductive animal studies either by inhalation or oral routes of exposure reported negative results. Only one oral study reported testicular Leydig cell tumors in male rats. In inhalation developmental studies, MTBE didn't cause structural effects in the offsprings during gestation. However in one study, MTBE was reported to be a developmental toxicant in offspring mice. Decreased weight gain of developing pups was reported in multigeneration reproductive inhalation studies in rats (ATSDR, 1996b).

G. Other Health Effects

Humans are acutely exposed to MTBE as a part of a medical treatment to dissolve cholesterol gallstones. Injection of the gall bladder with a high dose of MTBE can be associated with several types of health effects to include nausea, vomiting, drowsiness, mild sedation, confusion, vertigo and coma. Minor transient mucosal damage in the gallbladder has been demonstrated with extensive exposure, but no clinically significant consequences have been reported. One patient has been reported to have developed intravascular hemolysis and renal failure following inadvertent extravasation of a large bolus of MTBE (ATSDR, 1996b; USEPA, 1993a).

H. Epidemiological Evidence

The information regarding the human health effects noted above were located in studies of motorists, gas station attendants, and mechanics as well in experimental inhalation studies to MTBE for 1 hour, and in clinical studies of patients receiving therapy for dissolution of gallstones (ATSDR, 1996b).

I. Toxicity

MTBE has not been evaluated for carcinogenicity by USEPA. The chronic oral cancer slope factor is 0.004 (mg/kg-day)-1 (USEPA, 2004b). The chronic inhalation reference dose is 0.857 mg/kg-day (USEPA, 2004a).

1.13 STYRENE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	104.16
Melting Point:	-30.6°C
Boiling Point:	145.2°C
Specific Density:	0.906 (@ 20°C)
Water Solubility (mg/L):	300 (@ 20°C)
Vapor Pressure (mm Hg):	5 (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	2.61 x 10^{-3} (@ 25°C)

Reference: ATSDR, 1992b. Toxicological Profile for Styrene.

B. Chemical Transformation

Air: Styrene may be photooxidized by ozone and hydroxyl radicals with reported half-lives ranging from 0.5 to 17 hours. Transformation products include various oxygen-containing and saturated hydrocarbons (ATSDR, 1992b).

Water: Styrene has been reported to biodegrade in aquatic systems. It was found that the rate of biodegradation in groundwater was slow with estimated half-life reported at 6 weeks up to 7.5 months (ATSDR, 1992b).

Soil: Styrene biodegrades in soil. Biodegradation products include phenylethanol and phenylacetic acid (ATSDR, 1992b).

2. Toxicological Properties

A. Metabolism

There are several studies regarding the metabolism of styrene. Styrene is initially metabolized to styrene oxide. The styrene oxide is then hydrated to phenylethylene glycol (styrene glycol). This transformation is catalyzed by microsomal epoxide hydratase. The styrene glycol is then metabolized to mandelic acid (MA) or benzoic acid and then hippuric acid. Mandelic acid is also metabolized to PGA. The MA, PGA and hippuric acid are excreted in the urine (ATSDR, 1992b).

B. Acute Toxicity

Illness or injury has been reported in workers who have been acutely exposed to styrene. The most common health effect is injury to the nervous system. The health effects include depression, concentration problems, muscle weakness, tiredness, and nausea. It has been reported that styrene also causes respiratory effects which include irritation of the nose, throat, and eyes. There have been no reports of death as a result of acute styrene exposure. Recovery from the ill effects of short-term exposure is rapid after styrene exposure cease (ATSDR, 1992b).

C. Subacute and Chronic Toxicity

Chronic inhalation studies are available that investigated adverse health effects of styrene on workers in the plastics industry. Although the lung and liver are both effected by chronic inhalation exposure, neurological effects such as decreased short-term memory or impaired visuomotor performances seem to be the most sensitive indicators of toxicity. Chronic oral exposure to styrene is only available through animal studies. These animal studies have reported red blood cell and liver effects (ATSDR, 1992b).

USEPA has calculated an oral RfD based on a study using beagles. In this study, four beagle dogs/sex were gavaged with doses of 0, 200, 400, or 600 mg styrene/kg bw/day in peanut oil for 560 days. No adverse effects were observed for dogs administered styrene at 200 mg/kg-day. In the higher dose groups, increased numbers of Heinz bodies in the RBCs, decreased packed cell volume, and sporadic decreases in hemoglobin and RBC counts were observed. In addition, increased iron deposits and elevated numbers of Heinz bodies were found in the livers. Marked individual variations in blood cell parameters were noted for animals at the same dose level. Other parameters examined were body weight, organ weights, urinalyses, and clinical chemistry. The NOAEL in this study is 200 mg/kg-day and the LOAEL is 400 mg/kg-day (USEPA, 1993b).

D. Carcinogenicity

USEPA has not reviewed styrene regarding its carcinogenic potential. There are several occupational inhalation studies that suggest an association with increased incidence of leukemia and lymphoma, however, these studies are inconclusive due to exposure to multiple chemicals and small size of cohorts. Other studies have reported negative results and there are no oral or dermal exposure studies to styrene. Animal studies have reported limited evidence of styrene to be weakly carcinogenic in some mice and rats. Overall, human and animal studies suggest that styrene may be a weak human carcinogen (ATSDR, 1992b).

E. Mutagenicity

Genotoxicity tests for styrene both *in vivo* and *in vitro* have reported conflicting results and the genotoxic potential of styrene has not been clarified (ATSDR, 1992b).

F. Teratogenicity/Reproductive Effects

Some studies of female workers exposed to elevated air concentrations of styrene have suggested that styrene may cause lower birth weights and produce an increased risk of spontaneous abortions. A single three generation study showed no-styrene related reproductive effects, however, another animal study indicated altered testicular function (ATSDR, 1992b). Developmental effects were not generally observed in

animal studies, however, some fetal-toxicity and embryo-toxicity was observed (ATSDR, 1992b).

G. Other Health Effects

No other significant health effects were noted.

H. Epidemiological Evidence

Epidemiological and clinical studies in occupational settings have demonstrated that inhalation exposure to styrene may cause alterations of central nervous system functions. The symptoms are typical of central nervous system depression and appear to be the most sensitive end point for styrene exposure (ATSDR, 1992b).

I. Toxicity Data

USEPA has not reviewed styrene as to human carcinogenicity. A chronic oral reference dose is reported to be 0.2 mg/kg-day and a chronic inhalation reference dose is reported to be 0.286 mg/kg-day. The aforementioned toxicity values were taken from USEPA, 2004a.

1.14 TETRACHLOROETHYLENE (PERCHLOROETHYLENE OR PCE)

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	165.83
Boiling Point:	121°C
Melting Point:	-19°C
Specific Density:	1.6227 (@ 20°C)
Water Solubility (mg/L):	150 (@ 25°C)
Vapor Pressure (mm Hg):	18.47 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	1.8×10^{-2} (@ 25°C)

Reference: ATSDR, 1997b. Toxicological Profile for Tetrachloroethylene.

B. Chemical Transformation

Air: PCE undergoes atmospheric transformations through the reaction with photolytically-generated hydroxyl radicals. The degradation products of this reaction include phosgene and chloroacetylchlorides (ATSDR, 1997b).

Water: Predominant fate of PCE in aquatic environments is primarily volatilization into the atmosphere. Existing evidence indicates that PCE does not readily transform in water (ATSDR, 1997b). Biodegradation of PCE may be the most important transformation process in natural waters with the suspected byproducts being cis & trans-1,2-dichloroethene and trichloroethene (ATSDR, 1997b).

Soil: The predominant fates of PCE in soils is either volatilization to the atmosphere and leaching to groundwater. Biodegradation of PCE in soils appear to occur only under certain conditions and only to a limited extent (ATSDR, 1997b).

2. Toxicological Properties

A. Metabolism

Following inhalation or ingestion of PCE in humans, the primary metabolites identified in urine and blood were trichloroacetic acid and trichloroethanol (ATSDR, 1997b). The metabolites account for only 3% of the absorbed PCE by humans. The remaining absorbed PCE is exhaled unchanged. The metabolism of PCE is believed to be mediated by a cytochrome P-450 catalyzed oxidation reaction involving the formation of an epoxide intermediate.

B. Acute Toxicity

The primary targets of PCE toxicity include the brain, liver, and kidneys. There is also some evidence that suggest reproductive effects may be induced in women exposed to PCE (ATSDR, 1997b). Exposure to high concentrations (>1,000 ppm) of PCE vapor results in collapse, unconsciousness, and death in humans. The cause of death may be related to depression of respiratory centers of the central nervous system and possibly due to cardiac arrhythmia and heart block. Animal studies of oral exposure suggest that anesthesia and death would be likely occurrences in humans if high concentrations were swallowed (ATSDR, 1997b).

PCE has been shown to cause hepatotoxic effects in humans and animals by inhalation and oral routes of exposure. The types of PCE-induced hepatic effects in humans are not well documented, and the exposures or doses producing these effects are not adequately characterized. In animals, hepatic lesions were induced by inhalation exposure to PCE (ATSDR, 1997b). Reversible kidney damage has been reported in humans accidentally exposed to acutely toxic amounts of PCE vapor.

Neurological symptoms of acute inhalation exposure to high levels of PCE is well documented in humans and include headaches, dizziness, and drowsiness. Neurological symptoms of dizziness and drowsiness occurred at exposure to 216 ppm for 45 minutes to 2 hours: loss of motor coordination occurred at exposure to 280 ppm for 2 hours or 600 ppm for 10 minutes (ATSDR, 1997b). Human data suggest that the threshold for acute effects may be in the concentration range of 100 to 200 ppm with preanesthetic/anesthetic effects occurring at a threshold of 1,000 ppm.

C. Subacute and Chronic Toxicity

There is a suggestion that long-term inhalation exposure of workers to organic solvents, including PCE, causes irreversible neurological impairment. There are no data in humans to indicate that structural brain

damage is associated with PCE exposure (ATSDR, 1997b). Despite the relatively large number of people occupationally exposed to PCE, there are few cases of PCE-associated cardiotoxicity. In one study a patient experienced cardiac arrhythmia; he had been employed in a dry cleaning facility for 7 months where he treated clothes with PCE. There is no strong evidence that people exposed to environmental levels of PCE or levels at hazardous waste sites would develop cardiovascular effects (ATSDR, 1997b).

Subtle renal perturbations have been detected in at least one study of chronically exposed workers in dry cleaning workshops. They were exposed for an average of 14 years to an estimated time-weighted average of 10 ppm of PCE (ATSDR, 1997b).

D. Carcinogenicity

Carcinogenic effects have not been documented in exposed workers; however, cancer has been induced in experimental animals exposed by inhalation and oral routes (ATSDR, 1997b). USEPA has not classified PCE as to its carcinogenicity.

E. Mutagenicity

Some studies have indicated that PCE itself is not a mutagen, however, the metabolites of PCE have been shown to be mutagenic in several studies (ATSDR, 1997b).

F. Teratogenicity/Reproductive Effects

There is no evidence that PCE is a human teratogen (ATSDR, 1997b). Results from inhalation studies in animals suggest that PCE is fetotoxic but not teratogenic. There is some evidence that suggests PCE causes reproductive effects in women exposed to PCE in the workplace, however the evidence is not conclusive (ATSDR, 1997b).

G. Other Health Effects

Intense upper respiratory tract irritation occurred in humans exposed acutely by inhalation to high concentrations (>1,000 ppm) of PCE. Respiratory irritation (irritation of the nasal passages) was reported in workers exposed to PCE vapors at levels of 232 to 385 ppm, and in volunteers exposed to concentrations as low as 216 ppm for 45 minutes to 2 hours (ATSDR, 1997b). Skin damage (burns) and intense ocular irritation have been reported in humans exposed to concentrations of PCE liquid or vapors (>1,000 ppm) high enough to cause anesthetic effects (ATSDR, 1997b). Very mild eye irritation was reported by four subjects at exposure to 216 or 106 ppm (ATSDR, 1997b).

H. Epidemiological Evidence

Epidemiological studies of women occupationally exposed to PCE suggest that they have an increased risk of adverse reproductive effects (ATSDR, 1997b). Some epidemiological studies of dry cleaning workers

suggest a possible association between chronic PCE exposure and increased cancer risk. The results of these studies are inconclusive because of the likelihood of concomitant exposure to petroleum solvents, the effects of other confounding factors, such as smoking and other life-style variables, and methodological limitations in choosing control population and maintaining complete follow-up. Occupational exposure to PCE and other solvents did not generally result in increased risk of hematopoietic neoplasms (ATSDR, 1997b).

I. Toxicity Data

The chronic cancer slope factors are 0.54 (mg/kg-day)-1 and 0.02 (mg/kg-day)-1 for the oral and inhalation routes (USEPA, 2004b), respectively. The chronic oral reference dose is 0.01 mg/kg-day (USEPA, 2004a) and the chronic inhalation reference dose is 0.14 mg/kg-day (USEPA, 2004b).

1.15 TOLUENE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	92.15
Boiling Point:	110.6°C
Melting Point:	-95°C
Specific Density:	0.8669 (@ 20°C)
Water Solubility (mg/L):	534.8 (@ 25°C)
Vapor Pressure (mm Hg):	28.4 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	5.94 x 10^{-3}

Reference: ATSDR, 2000b. Toxicological Profile for Toluene.

B. Chemical Transformation

Air: Toluene in the atmosphere was rapidly degraded by reaction with hydroxyl radicals to yield cresol and benzaldehyde, which in turn undergo ring cleavage to yield simple hydrocarbons. Half-lives have been reported to range from 10 to 104 hours depending on atmospheric conditions (ATSDR, 2000b).

Water: Toluene can be oxidized by reactions similar to those in air, but the rates of these reactions are very slow. Biodegradation of toluene in water is a more rapid process. In surface waters, the biodegradation half-life has been reported from 4 to 22 days, whereas the biodegradation half-life of toluene in groundwaters was reported from 7 to 28 days (ATSDR, 2000b).

Soil: Toluene can be biodegraded in soil by a number of bacterial species. The biodegradation process appears to occur in two

phases with the first phase producing benzoic acid, and in the second phase, the aromatic ring is cleaved and the intermediates are degraded to carbon dioxide. Biodegradation half-life for toluene in soils have been reported from 1 to 7 days (ATSDR, 2000b).

2. Toxicological Properties

A. Metabolism

Toluene is initially converted to benzyl alcohol in the liver, with benzyl alcohol being oxidized to benzaldehyde, and subsequently to benzoic acid. Benzoic acid is then conjugated with glycine to form hippuric acid, a major urinary metabolite. Benzoic acid can also react with glucuronic acid to form benzoyl glucuronide. Toluene is also catalyzed to o- and p-cresol that are then conjugated with sulfate glucuronic acid, glutathione or cysteine and are excreted in the urine. In both humans and animals, 60% to 75% of inhaled toluene that is absorbed can be accounted for as hippuric acid in the urine. Much of the remaining toluene is exhaled unchanged. The excretion of toluene and its metabolites is rapid, with the major portion occurring within 12 hours of exposure (ATSDR, 2000b).

B. Acute Toxicity

The primary effect in humans following oral and inhalation exposure to toluene is depression of the central nervous system. Acute inhalation exposure to toluene resulted in subtle neurological disorder at concentrations of 75 to 150 ppm in volunteers. Concentrations of 200 to 800 ppm produced exhilaration and light headedness, and at higher acute exposure concentrations, intellectual, psychomotor and neuromuscular abilities were impaired followed by development of narcosis (ATSDR, 2000b). Under acute exposure conditions [short exposures to greater than 10,000 ppm (37,690 mg/m^3)], toluene produces CNS narcosis (USEPA, 1994a). Neurological effects observed in animals after acute oral exposure include changes in levels of several neurotransmitters and clinical signs of ataxia, prostration, and tremors. Mortality reports in humans due to exposure to toluene have generally not provided information on dose and thus, do not provide a basis for quantitative estimates. In one fatal case following oral ingestion of toluene, the cause of death appeared to be a profound disruption of central nervous system function (ATSDR, 2000b).

C. Subacute and Chronic Toxicity

Toluene-induced neurotoxicity has been documented in humans over a broad spectrum of severity that correlates well with concentration. Numerous case studies on chronic toluene abusers [repeatedly exposed to greater than 30,000 ppm (113,000 mg/m^3)] have demonstrated functional deficits of the CNS accompanied by abnormal morphology of cerebellar and cortical areas of the brain. Lower concentrations, i.e., 800 to 400 ppm

(3,015 to 1,508 mg/m^3), have been associated with worker complaints of CNS-related effects. Clinical studies using controlled exposure to toluene have demonstrated concentration-related occurrence of complaints such as drowsiness, ataxia, visual impairment, and headache (USEPA, 1994a). A number of occupational studies where the exposure concentration of toluene ranged from 30 to 150 ppm found evidence for increased incidence of self-reported neurological symptoms, performance deficits in neurobehavioral tests, hearing loss, changes in visual-evoked brainstem potentials, and color vision impairment (ATSDR, 2000b).

D. Carcinogenicity

Human and animal studies do not support a concern for the carcinogenicity of toluene. The only available human epidemiological studies were negative, but inconclusive due to limitation in design and the animal bioassays were all negative (ATSDR, 2000b). USEPA has placed toluene in Group D, not classifiable as to human carcinogenicity due to inadequate or no evidence in animal studies (USEPA, 1994a).

E. Mutagenicity

Studies indicate that toluene is nonmutagenic and nongenotoxic (ATSDR, 2000b).

F. Teratogenicity/Reproductive Effects

In general, results from human and animal studies suggest toluene not to be a potent reproductive toxicant but may cause some reproductive problems. Women occupationally exposed or wives of men similarly exposed, have an increased risk of spontaneous abortions. Occupational exposure to concentrations of toluene of 8 to 111 ppm reported decreased plasma levels of the luteinizing hormone, follicle stimulating hormone, and testosterone levels in males. Animal studies reported decreased sperm count in males, structural changes in antral follicles in ovary of female rats and increased fetal mortality (ATSDR, 2000b).

Case reports of birth defects in children of mothers who abused toluene during pregnancy suggest that exposure to high levels of toluene may be toxic to developing fetus. Birth defects, similar to those associated with fetal alcohol syndrome include microcephaly, central nervous system dysfunction, growth deficiency, cranofacial and limb abnormalities, and reversible renal tubular acidosis. Results from animal studies indicate that toluene can retard fetal growth and skeletal development, and adversely influence behavior of offspring at exposure levels that overwhelm maternal mechanisms (ATSDR, 2000b).

G. Other Health Effects

The primary effect of toluene following inhalation exposure is irritation of the respiratory tract. Early animals studies reported respiratory irritation and pulmonary lesions in rats exposed to high concentrations of toluene. Mice exposed to concentrations of toluene from 600 to 1,000 ppm,

6.5 hours/day, 5 days/week for 2 years reported similar observations (ATSDR, 2000b).

Acute inhalation of high levels of toluene results in alterations of the heart rhythm for both humans and animals. The exposures associated with cardiac rhythm disturbances were of the short-term, high-level type experienced by substance abusers. Therefore, cardiovascular responses are not expected to occur following inhalation exposure of toluene at or near hazardous waste sites, unless some occurrence releases a high concentration of toluene into an enclosed area (ATSDR, 2000b).

It is unlikely that toluene exposure resulting from the contamination of hazardous waste sites would cause gastrointestinal effects in any exposed population (ATSDR, 2000b). Humans have reported eye irritation following exposure to toluene vapors. Human subjects exposed acutely to toluene concentrations of 100 ppm and greater developed irritation of the eyes (ATSDR, 2000b). No effects were observed at lower doses. Skin irritation can also occur in humans and animals dermally exposed to toluene. This appears to be due to the degreasing action of toluene and its removal of protective skin oils (ATSDR, 2000b).

H. Epidemiological Evidence

It is well known for solvents to affect CNS processes in humans which leaves little doubt that the brain is a principal target organ for toluene toxicity in humans. In cases of inhalation abuse of toluene, one case demonstrated diffuse cerebral, cerebellar, and brainstem atrophy in 3 of 11 toluene abusers who also had neurological abnormalities. Another case study was able to correlate neuropsychological impairment with the degree of white matter abnormality. Cerebellar and cortical functions were classified as impaired in 15/24 individuals who had abused toluene daily (425 +/- 366 mg/day) for extended periods (6.3 +/- 3.9 years). In a limited case study, it was demonstrated that brainstem atrophy through computerized tomographic scans and abnormal brainstem auditory-evoked potentials in 2/2 chronic toluene abusers (12 to 16 years of admitted, continuous abuse). These studies confirm the occurrence of severe CNS damage in response to highly abusive concentrations of toluene (USEPA, 1994a).

I. Toxicity Data

USEPA has not classified toluene as to human carcinogenicity, due to inadequate evidence of carcinogenicity in studies. The chronic reference doses are reported at 0.2 and 0.114 mg/kg-day for both oral and inhalation, respectively. The aforementioned toxicity values were taken from USEPA, 2004a.

1.16 TRICHLOROETHENE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	131.40
Boiling Point:	86.7°C
Melting Point:	-87.1°C
Specific Density:	1.465 (@ 20°C)
Water Solubility (mg/L):	1,366 (@ 25°C)
Vapor Pressure (mm Hg):	74 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	1.1 x 10^{-2} (@ 25°C)

Reference: ATSDR, 1997c. Toxicological Profile for Trichloroethylene.

B. Chemical Transformation

Air: The dominant transformation process for TCE in the atmosphere is reaction with hydroxyl radicals to form the following degradation products: phosgene, dichloroacetyl chloride, and formyl chloride (ATSDR, 1997c).

Water: Most TCE in surface waters can be expected to volatilize to the atmosphere. Microbial degradation products of TCE in groundwater were reported to be dichloroethylene and vinyl chloride (ATSDR, 1997c).

Soil: The majority of TCE in surficial soils will volatilize to the atmosphere. TCE is also highly mobile and is susceptible to leaching. In one study, methane utilizing bacteria degraded TCE to carbon dioxide, but not dichloroethylene or vinyl chloride (ATSDR, 1997). In another study, TCE was transformed 100% to vinyl chloride after 10 days in anaerobic, methanogenic conditions (ATSDR, 1997c).

2. Toxicological Properties

A. Metabolism

Inhaled doses of TCE are metabolized extensively in humans. The principal metabolites of TCE are trichloroethanol, trichloroethanol-glucoronide ("urochloralic acid"), and trichloroacetic acid (ATSDR, 1997c).

B. Acute Toxicity

Cases of human deaths have been reported as a result of acute accidental exposure in an occupational setting or by intentionally drinking or breathing large amounts of TCE (i.e., suicides). No deaths due to dermal exposure have been reported. Death is not likely to result from exposure to environmental levels or to levels of TCE found at hazardous waste sites (ATSDR, 1997c).

There are inadequate human data regarding the possible hepatic effects of TCE. People who have been acutely exposed during surgical anesthesia, and most people exposed chronically in the workplace have not had adverse liver effects. However, a few case reports do show minor effects on serum or urinary measures of liver function. People who have been acutely exposed to high levels of TCE during surgical anesthesia, or chronically in the workplace, have not had renal toxicity. However, minor changes in urinary and serum indicators of renal function have been found in other workers (ATSDR, 1997c).

In the past, TCE was used as an anesthetic, so it obviously can cause acute central nervous system depression in humans (ATSDR, 1997c). People have become unconscious after acute exposure to very high levels occasionally present in the workplace. Other nonspecific neurological effects from TCE exposure in the workplace have been reported, and include dizziness and drowsiness.

C. Subacute and Chronic Toxicity

Cardiovascular disease had not been reported in workers chronically exposed to TCE, although deaths following acute high-level exposures to TCE were attributed to cardiac arrhythmias (TCE exposure levels could not be established for these studies). It is not known whether cardiovascular effects could result from exposure to levels of TCE found at or near hazardous waste sites (ATSDR, 1997c). There are also a few case reports of persons showing hepatorenal failure following exposure to very large amounts of TCE (TCE exposure levels were not reported). It has been suggested that liver damage may result from prolonged exposure but not acute exposures, and it is unknown whether exposure to levels of TCE found in and around hazardous waste sites may result in hepatic injury (ATSDR, 1997c).

D. Carcinogenicity

Workers who have been exposed to TCE showed no higher incidence of cancer than controls. The few studies that did show some association were complicated by exposures to other known human carcinogens. Animal studies have shown increases in cancers of various types following inhalation or oral exposure to TCE. The significance of these studies for humans cannot be determined due to other circumstances. (this statement is incorrect – USEPA withdrew its classification and toxicity data, under further review).

E. Mutagenicity

The data regarding genotoxicity of TCE in humans are inconclusive (ATSDR, 1997c). The potential for gene mutations is not known, and the mechanisms for carcinogenicity are not known (ATSDR, 1997c).

F. Teratogenicity/Reproductive Effects

Limited evidence exists that would link TCE exposure to developmental toxicity in humans (ATSDR, 1997c). There is no evidence that exposure to TCE caused adverse reproductive effects in humans and the biological significance of positive animal effects is unknown (ATSDR, 1997c). Thus, TCE in air, water, or soil at hazardous waste sites is not expected to adversely affect human reproduction (ATSDR, 1997c).

G. Other Health Effects

Some humans experience dry throats and mild eye irritation following acute inhalation exposure (200 ppm for 7 hours) to TCE. Persons working with TCE for intermediate periods sometimes develop skin burns or rashes and dermatitis. TCE is not known to cause dermal effects when given via the oral route. It is possible that exposure to TCE in the air or soil at hazardous waste sites would be irritating to human eyes or skin (ATSDR, 1997c).

H. Epidemiological Evidence

No data available.

I. Toxicity Data

USEPA (1989) has withdrawn its carcinogenic classification following further review. The chronic cancer slope factors are 0.4 (mg/kg-day)-1 and 0.4 (mg/kg-day)-1 for the oral and inhalation routes, respectively. The chronic reference doses are 0.0003 and 0.01 mg/kg-day for the oral and inhalation routes, respectively. All of the aforementioned toxicity values were taken from USEPA, 2004b.

1.17 VINYL CHLORIDE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	62.5
Boiling Point:	-13.4°C
Melting Point:	-153.8°C
Specific Density:	0.9106 (@ 20°C)
Water Solubility (mg/L):	2,763 (@ 25°C)
Vapor Pressure (mm Hg):	2,600 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	2.78 x 10^{-2} (@ 25°C)

Reference: ATSDR, 2004a. Toxicological Profile for Vinyl Chloride.

B. Chemical Transformation

Air: Reaction with hydroxyl radicals is predicted to be the primary degradation mechanism for vinyl chloride, with the products of

this reaction being hydrochloric acid, formaldehyde, formyl chloride, carbon dioxide, carbon monoxide, chloroacetaldehyde, acetylene, chloroethylene epoxide, chloroacetylchloranil, and water (ATSDR, 2004a).

Water: Vinyl chloride in surface waters will primarily volatilize to the atmosphere. Oxidation, hydrolysis and biodegradation do not seem to be significant transformation processes (ATSDR, 2004a).

Soil: Vinyl chloride in surficial soils will primarily volatilize to the atmosphere. Vinyl chloride is also mobile and susceptible to leaching (ATSDR, 2004a).

2. Toxicological Properties

A. Metabolism

Metabolism of vinyl chloride appears to be a dose-dependent, saturable process. Vinyl chloride is metabolized in the liver by the action of cytochrome isozymes (P450 CYPIIE1) with the production of an epoxide intermediate, chloroethylene oxide (CEO). CEO spontaneously rearranges to form chloroacetaldehyde (CAA). These intermediates are detoxified mainly through conjugation with glutathione. These conjugated products are excreted in urine as substituted cysteine derivatives (USEPA, 2000).

B. Acute Toxicity

Human exposure to vinyl chloride occurs primarily through inhalation. Occupational studies of exposure to vinyl chloride have reported the central nervous system (CNS) as the primary target organ, reporting effects to include headaches, drowsiness, dizziness, ataxia and loss of consciousness. A threshold for CNS effects appears to be approximately 8,000 ppm. Extremely high concentrations (>25,000 ppm) of vinyl chloride resulted in respiratory irritation and deaths in both human and animal studies (ATSDR, 2004a). There is one reported case of dermal exposure to vinyl chloride. Severe frostbite with second degree burns on the hands was reported resulting from the rapid evaporation of spilled liquid vinyl chloride (ATSDR, 2004a).

C. Subacute and Chronic Toxicity

Several epidemiology and case studies have associated chronic occupational exposure with impaired liver function, biochemical evidence of liver damage or histological evidence of liver damage. Changes that are characteristic to liver damage include extensive fibrosis of the portal tracts and septa, and intralobular perisinusoidal regions, hepatocellular degeneration, sinusoidal dilation, and hypertrophy and hyperplasia of both hepatocytes and sinusoidal cells. Recent studies have demonstrated morbidity and mortality related to fibrosis, portal hypertension, and cirrhosis among workers. Other studies have reported

peripheral neuropathy may also develop in some workers exposed to vinyl chloride (ATSDR, 2004a).

D. Carcinogenicity

Vinyl chloride is regarded as a human carcinogen. The most compelling evidence for the carcinogenic potential of vinyl chloride in humans comes from the cluster of reports of greater than expected incidences of angiosarcoma of the liver in workers occupationally exposed to vinyl chloride. Vinyl chloride is classified into Group A, a known human carcinogen (USEPA, 2000).

E. Mutagenicity

There is evidence that vinyl chloride metabolites are genotoxic, interacting directly with DNA. The metabolites CEO and CAA are both mutagenic in Salmonella assay, although the metabolite CEO is much more mutagenic than the metabolite CAA. The reactive metabolite binds to DNA forming DNA adducts and if not repaired leads to mutations. Occupational exposure to vinyl chloride has resulted in chromosome aberrations, micronuclei, and sister chromatid exchanges (USEPA, 2000).

F. Teratogenicity/Reproductive Effects

A number of animal studies has shown developmental toxicity from vinyl chloride exposure consisting of decreased litter size and fetal weight, delayed ossification and dilated ureters. Studies in humans have not indicated that male reproductive function may be adversely affected by exposure to vinyl chloride. There are some studies with evidence of testicular damage in rats exposed to vinyl chloride by inhalation. However, reduced fertility was reported at concentrations well above those producing effects in the liver (USEPA, 2000).

G. Other Health Effects

Vinyl chloride disease has been reported in a small percentage of workers exposed to the chemical. One of the symptoms of this disease is condition referred to as Raynaud's phenomenon, in which fingers blanch and experience numbness and discomfort upon exposure to the cold. Skin exposure may also result in acroosteolysis and sclerodermatous skin changes. These skin changes occur almost exclusively on workers who are exposed to high concentrations and are not relevant to low level environmental exposures (USEPA, 2000).

H. Epidemiological Evidence

Studies located support the conclusion that vinyl chloride is a likely human carcinogen. There are several epidemiological studies or case reports in humans showing adverse health effects. The primary target organ for non-cancer effects is the liver (USEPA, 2000).

I. Toxicity Data

USEPA (2000) has classified vinyl chloride as a human carcinogen with sufficient evidence in human epidemiological studies. The chronic cancer slope factors for a child are 1.4 (mg/kg-day)-1 and 0.03 (mg/kg-day)-1 for the oral and inhalation routes, respectively. The chronic cancer slope factors for an adult are 0.72 (mg/kg-day)-1 and 0.015 (mg/kg-day)-1 for the oral and inhalation routes, respectively. The chronic reference doses for an adult are 0.003 and 0.028 mg/kg-day for the oral and inhalation routes, respectively. All of the aforementioned toxicity values were taken from USEPA, 2004a.

1.18 XYLENES

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	106.16
Boiling Point:	138.5°C
Melting Point:	no data
Specific Density:	0.864 (@ 20°C)
Water Solubility (mg/L):	130 (@ 25°C)
Vapor Pressure (mm Hg):	6-16 (@ 20°C)
Henry Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 1995b. Toxicological Profile for Xylenes.

B. Chemical Transformation

Air: Xylene is transformed in the atmosphere primarily by photooxidation (ATSDR, 1995b). The major photodegradation products formed by the cleavage of the aromatic ring in the presence of nitric oxide are: o-tolualdehyde, methylglyoxal, 4-nitro-o-xylene, and 2,3-dimethylphenol for o-xylene; 2,6-dimethylphenol, 2,4-dimethylphenol, methylglyoxal, and m-tolualdehyde for m-xylene; and p-tolualdehyde and 2,5-dimethylphenol for p-xylene (ATSDR, 1995b).

Water: In surface waters, volatilization is the dominant removal process. Therefore, biotransformation of xylene in surface waters is probably not significant (ATSDR, 1995b). Biodegradation of xylene is possible in aquatic systems if conditions are right.

Soil: Xylene on surface soil will rapidly volatilize, with the remaining xylene likely being photooxidized (ATSDR, 1995b). Xylene can be biodegraded under proper environmental conditions. Some studies have reported biodegradation products of xylenes as xylenols and benzoic acids.

2. Toxicological Properties

A. Metabolism

Xylene is primarily metabolized in the liver by oxidation of a methyl group to yield methylbenzoic acids, and subsequent conjugation with glycine to yield methylhippuric acids that are excreted in the urine. This metabolic pathway accounts for almost all the absorbed dose of xylene, regardless of the isomer, the route of exposure, the size of the dose, and the exposure duration. The excretion of methylhippuric acids is rapid and a significant amount is detected in the urine within 2 hours of exposure (USEPA, 2003f).

B. Acute Toxicity

Reversible symptoms of neurological impairment and irritation of the eyes and throat are well-known health hazards from acute inhalation exposure to xylenes and other aromatic solvents. In general, these acute effects are expected to involve reversible molecular interactions of the solvent itself (not metabolites) with membranes of the affected tissues, including neuronal membranes, and are most pronounced at high exposure levels in excess of 1,000 ppm. At lower concentrations, more subtle effects may occur. Human volunteers exposed under controlled conditions to xylenes concentrations in the range of 200 to 400 ppm for short time periods (15 minutes to 4 hours) have reported symptoms of irritation including watering eyes and sore throat or neurological impairment including mild nausea, headache. Animal studies have demonstrated that these effects are the most sensitive to xylenes, with measurable effects endpoints beginning at concentrations as low as 100 ppm. The available controlled-exposure human studies suggest concentrations at approximately 100 to 200 ppm are close to the threshold level for short-term reversible neurological and irritation effects from xylenes (USEPA, 2003f).

C. Subacute and Chronic Toxicity

No data on the effects of xylenes in humans following chronic exposure are available. Results from several subchronic animal studies and one chronic animal study identify decreased body weights and increased mortality as potential health hazards from repeated oral exposure to doses greater than 500 to 800 mg/kg-day. Results from subchronic animal studies identify neurological impairment effects as potential health hazards from repeated inhalation exposures. Neurological endpoints after chronic inhalation exposure to xylenes include decreased rotarod performance, decreased spontaneous motor activity, and impaired radial maze performance indicative of a learning deficit (USEPA, 2003f).

D. Carcinogenicity

Data in both humans and animals are inadequate to evaluate potential associations between xylenes and cancer. A number of human occupational studies have suggested possible carcinogenic effects of chronic inhalation exposure. However, in each case co-exposure to other chemicals was a major confounding factor, leading to an inability to adequately assess the potential effects of chronic exposure to xylenes. Animal data on the carcinogenic effects of oral exposure to xylenes provided inadequate data (USEPA, 2003f).

E. Mutagenicity

Mixed xylene, and the individual isomers, have been tested for genotoxicity in a variety of assays, with the results indicating that they are nongenotoxic (USEPA, 2003f).

F. Teratogenicity/Reproductive Effects

The most significant effects on developmental endpoints were decreased fetal body weight and fetal survival in rats at xylene isomer doses of 350 or 750 ppm or at mixed xylenes concentration of 780 ppm. Increased abortions in rabbits exposed to 230 ppm was also reported in another study. Although these are the most significant effects, others reported include neurodevelopmental effects, skeletal and visceral malformations. There are only a few studies of human or animal reproductivity reported. Human studies were of limited usefulness in assessing the potential reproductive toxicity of xylenes, because the numbers of cases of spontaneous abortions were small, and the women had been exposed to a number of chemicals. There are two animal reports regarding reproductive effects. In one study, male Sprague-Dawley rats exposed to 0 or 1,000 ppm xylene solvent for 18 hours per day, 7 days per week, for 61 days reported no differences between control and exposed rats in several testicular endpoints and fertility (USEPA, 2003f).

G. Other Health Effects

There are some case studies where xylenes was ingested either accidentally or purposely. One report where a 27-year-old man committed suicide resulted in histopathologic effects including pulmonary edema and congestion resulting in death. The probably cause of death was attributed to respiratory failure and asphyxia, a secondary response elicited by depression in the respiratory center of the brain. In another case where xylene was accidentally ingested resulted in a deep coma, hepatic impairment, acute pulmonary edema and other pulmonary complications. Health effects reported from case studies of individuals after inhalation exposure to xylenes include major and minor seizures, unconciousness, vertigo, headache, gastric discomfort, and slight drunkedness (USEPA, 2003f).

H. Epidemiological Evidence

No data available.

I. Toxicity Data

USEPA has placed xylenes into Group D, not classifiable as to human carcinogenicity, due to inadequate evidence of carcinogenicity in animal studies. The chronic reference doses are 0.2 and 0.03 mg/kg-day for the oral and inhalation routes, respectively. All of the aforementioned toxicity values were taken from USEPA, 2004a.

2.0 SEMI-VOLATILE ORGANIC COMPOUNDS

2.1 2-AMINOPYRIDINE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	94.12
Melting Point:	58.1°C
Boiling Point:	210.6°C
Specific Density:	1.065 (@ 20°C)
Water Solubility (mg/L):	1,000,000 (@ 20°C)
Vapor Pressure (mm Hg):	no data
Henry's Law Constant (atm-m^3/mol):	no data

Reference: Hazardous Substances Databank (HSDB, 2004).

B. Chemical Transformation

Air: 2-aminopyridine will exist solely as a vapor in the ambient atmosphere. Vapor-phase 2-aminopyridine will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 19 hours (HSDB, 2004).

Water: Aminopyridines may be susceptible to photochemical degradation in water. Biodegradation in water may slowly occur based upon biodegradation studies in soil (HSDB, 2004).

Soil: In one study, 2-aminopyridine was found to completely degrade in greater than 96 days under both aerobic and anaerobic conditions in soil (HSDB, 2004).

2. Toxicological Properties

A. Metabolism

No data available.

B. Acute Toxicity

Acute poisoning can occur from inhalation of the dust or vapor at relatively low concentrations, or by skin absorption following direct contact. Ingestion of as little as 60 mg in adult humans has caused severe poisoning. Typical symptoms included intense diaphoresis, weakness, nausea, dizziness and thirst followed by psychotic-like behavior, tremors, dyspnea, and tonic-clonic seizures. An ECG may show non-specific ST-T wave changes. High concentrations have resulted in elevation of cardiac action potential plateau and depression of diastolic depolarization. Respiratory arrest has been reported. Weakness, dizziness, disorientation, hyperexcitibility, tremors, periorbital paresthesias and tonic-clonic seizures may be noted. Elevations of serum SGOT, LDH, and

alkaline phosphatase have been noted. Metabolic acidosis has also been noted (HSDB, 2004).

C. Subacute and Chronic Toxicity

No data available.

D. Carcinogenicity

No data available.

E. Mutagenicity

2-Aminopyridine was evaluated for mutagenicity in the Salmonella/microsome preincubation assay using the standard protocol approved by the National Toxicology Program. 2-Aminopyridine was tested at doses of 0, 33, 100, 333, 1,000, and 10,000 µg/plate in four Salmonella typhimurium strains (TA98, TA100, TA1535, and TA1537) in the presence and absence of Aroclor-induced rat or hamster liver S9. 2-Aminopyridine was negative in these tests and the highest ineffective dose tested in any Salmonella tester strain was 10,000 µg/plate (HSDB, 2004).

F. Teratogenicity/Reproductive Effects

No data available.

G. Other Health Effects

No data available.

H. Epidemiological Evidence

Fatal intoxication occurred in a chemical operator who spilled 2-aminopyridine during distillation. He continued to work in contaminated clothing for 1 1/2 hours. Two hours later he developed dizziness, headache, respiratory distress, and convulsions that progressed to respiratory failure and death. A chemical operator milling 2-aminopyridine without protective equipment, developed severe pounding headache, nausea, flushing of extremities, and elevated blood pressure, but recovered fully next day (HSDB, 2004).

I. Toxicity Data

No data available.

2.2 4-CHLORO-3-METHYLPHENOL

1. Constituent Properties

A. Physical and Chemical Properties

Molecular Weight:	142.59
Melting Point:	67°C

Boiling Point:	235°C
Specific Density:	1.37 (@ 20°C)
Water Solubility (mg/L):	3,830 (@ 25°C)
Vapor Pressure (mm Hg):	5.0×10^{-2} (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: Hazardous Substances Databank (HSDB, 2004).

B. Chemical Transformation

Air: Vapor-phase 3-methyl-4-chlorophenol will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 15 hours (HSDB, 2004).

Water: 3-Methyl-4-chlorophenol is expected to biodegrade in water based on its complete degradation in a river water closed bottle test. Volatilization from water surfaces is expected to be an important environmental fate process based upon this compound's estimated Henry's Law constant. Estimated volatilization half-lives for a model river and model lake are 16 and 120 days, respectively. 3-Methyl-4-chlorophenol is not expected to undergo hydrolysis in the environment because phenols are generally resistant to hydrolysis (HSDB, 2004).

Soil: Volatilization from moist soil surfaces is expected to be an important environmental fate process based upon an estimated Henry's Law constant of 2.45×10^{-6} atm-cu m/mole. 3-Methyl-4-chlorophenol is not expected to volatilize from dry soil surfaces based upon its vapor pressure. Biodegradation half-lives were 21 days in both sandy clay and silty clay soils at initial concentrations of 10 and 1,000 ppm each. Thus, 3-methyl-4-chlorophenol is expected to biodegrade in soil (HSDB, 2004).

2. Toxicological Properties

A. Metabolism

Structure-activity relationship analyses suggest that the chloro and methyl groups in chlorocresols are important for the activation of the ryanodine receptor Ca2+ release channel (HSDB, 2004).

B. Acute Toxicity

Burning pain in mouth and throat. 1. White necrotic lesions in mouth, esophagus and stomach accompanied by abdominal pain, vomiting, and bloody diarrhea. 2. Pallor, sweating weakness, headache, dizziness, tinnitus. 3. Shock: Weak irregular pulse, hypotension, shallow respirations, cyanosis, pallor, and a profound fall in body temperature. 4. Possibly fleeting excitement and confusion, followed by unconsciousness. 5. Stentorous breathing, mucous rales, rhonchi, frothing at nose and mouth and other signs of pulmonary edema are

sometimes seen. Characteristic of phenol on the breath. 6. Scanty, dark-colored urine, moderately severe renal insufficiency may appear. 7. Methemoglobinemia, Heinz body hemolytic anemia and hyperbilirubinemia have been reported. 8. Death from respiratory, circulatory or cardiac failure. 9. If spilled on skin, pain is followed promptly by numbness. The skin becomes blanched, and a dry opaque eschar forms over the burn. When the eschar sloughs off, a brown stain remains (HSDB, 2004).

C. Subacute and Chronic Toxicity

p-Chloro-m-cresol has been reported to cause vesicular dermatitis in humans. Concentrations of 1.5% (aqueous) cause a pruritic vesicular dermatitis in sensitive individuals. Symptoms occur within 4 hours and regress within a week (HSDB, 2004).

D. Carcinogenicity

No increased risk to cancer was found in animal studies (HSDB, 2004).

E. Mutagenicity

Technical p-chloro-m-cresol was tested for potential to induce reverse mutations in Salmonella typhimurium strains TA 1535, TA 1537, TA 100 and TA 98. Metabolic activation was provided by an S-9 mix prepared from the livers of adult male Sprague-Dawley rats, induced with an injection of Aroclor 1254. Five dose levels, ranging from 20 to 12,500 µg/plate, were used. All dose groups were tested with S-9 mix; the highest dose was also tested in the absence of the S-9 mix. Endoxan® and Trypaflavin were used as positive controls. The two highest dose levels, 2,500 and 12,500 µg/plate were toxic to the cells. p-Chloro-m-cresol failed to induce a mutagenic response at any of the three lowest dose levels (20, 100, or 500 µg/plate) with all four tester strains. The Salmonella/mammalian microsome test was performed for technical 3-methyl-4-chlorophenol using Salmonella typhimurium strains TA-100, TA-1535, TA-1537, and TA-98. Dimethylsulfoxide acted as a negative control. Sodium azide or 2-nitrofluorene served as positive control in tests without S9; 2-antramine was the positive control in tests performed with S9. 4-Chloro-3-methylphenol (CMP) was added to plates in concentrations of 1.28, 6.4, 32, 160, or 800 µg. No mutagenic potential of CMP was evident in Salmonella tester strains, with or without S9 (HSDB, 2004).

F. Teratogenicity/Reproductive Effects

Developmental or Reproductive Toxicity of p-Chloro-m-cresol was tested by gavage in a developmental toxicity study in rats at the following dose levels: 0, 30, 100, or 300 mg/kg/day during gestation days 6 to 15 inclusive. At 300 mg/kg, six dams died. Treatment-related clinical signs of toxicity included audible breathing sounds, gasping breathing, reduced motility and high stepping gait; and after dosing: lying on side,

somnolence, abdominal position, spastic convulsions, rough coat, sunken flanks, and bloody muzzle. Statistically significant decreases in body weight gain were observed during the dosing period and significant decreases in the corrected body weight gain were also observed during the entire gestation period. In addition, the decreased food consumption was also considered to be biologically significant. Finally, two dams totally resorbed their litters (HSDB, 2004).

G. Other Health Effects

No data available.

H. Epidemiological Evidence

A case of contact urticaria caused by detergent disinfectants containing p-chloro-m-cresol and o-benzyl-p-chlorophenol was reported. A 28-year-old woman working in an aviary complained of red swollen eyelids each time she washed chicken incubators. The symptoms lasted for several hours. A 42-year-old woman who experienced more than 50 attacks of left-sided facial palsies after exposure to chlorocresol was studied. Only muscles around the left side of the mouth were affected. On neurophysiological testing during chlorocresol provocation the only abnormality was a loss of motor units during maximal contraction of the left orbicularis oris muscle. This could be explained by a peripheral as well as a central effect (HSDB, 2004).

I. Toxicity

No data available.

2.3 4-CHLOROANILINE (P-CHLOROANILINE)

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	127.57
Melting Point:	72.5°C
Boiling Point:	232°C
Specific Density:	1.429 (@ 19°C)
Water Solubility (mg/L):	3,900 (@ 20-25°C)
Vapor Pressure (mm Hg):	0.015 (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	1.07×10^{-5} (@ 25°C)

Reference: Montgomery and Welkom, 1990.

B. Chemical Transformation

Air: No data available.

Water: Under artificial sunlight, river water containing 2 to 5 ppm of 4-chloroaniline photodegraded to 4-aminophenol and unidentified polymers (Montgomery and Welkom, 1990).

Soil: No information was located.

2. Toxicological Properties

A. Metabolism

In a recent metabolism study in which radiolabelled p-chloroaniline was administered to male rats, female mice, and a male rhesus monkey, greater than 90% of the radiolabelled-carbon was eliminated through the urine in all species. A major route of metabolism was identified as ortho hydroxylation, whereby 2-amino-5-chlorphenyl sulfate is the major excretion product. In addition, p-chloroacetanilide was also found to be a major circulating metabolite (Patty's, 1994). No information on metabolic rates or elimination rates for p-chloroaniline was reported.

B. Acute Toxicity

The oral LD50 in rats is reportedly 0.31 g/kg (Patty's, 1994). It has been reported that p-chloroaniline has been associated with oxidative hemolytic anemia in humans as a result of oxidative hemolysis of the red blood cells. p-Chloroaniline is absorbed through the intact skin and may cause methemoglobinemia. A common major effect of many aromatic nitro and amino compounds is their ability to convert hemoglobin to the oxidized ferric form, methemoglobin. Methemoglobin is unable to transport oxygen and symptoms of oxygen deficiency may begin to appear, the severity depending on the amount of conversion to methemoglobin (Patty's, 1994).

C. Subacute and Chronic Toxicity

The National Cancer Institute (NCI) designed a chronic oral bioassay study. Groups of 20 and 50 F344 rats of each sex were exposed to p-chloroaniline in the diet at concentrations of 0, 250 or 500 ppm for 78 weeks followed by an observation period of 24 weeks. Gross and comprehensive histological examinations were performed on all animals after sacrifice (USEPA, 1995c).

Significantly increased mortality occurred in the high-dose male rats and decreased average body weight gain occurred in the high-dose female rats. Non-neoplastic proliferative lesions of the capsule of the spleen (focal fibrosis with subcapsular mesenchymal proliferation) occurred in most of the treated rats. Fibrosis or fatty infiltration of the splenic parenchyma occurred in some of the high-dose males and one of the high-dose females. Splenic lesions did not occur in any of the control rats (USEPA, 1995c).

This study did not define a No Observed Effect Level (NOEL). The 250 ppm level (12.5 mg/kg/day) is considered to be the Lowest Observed

Adverse Effect Level (LOAEL), which when divided by an uncertainty factor of 3000 results in a RfD of 0.004 mg/kg/day. Although the NCI bioassay is a well designed chronic oral study, confidence in the study is low because a NOEL or NOAEL was not defined. Confidence in the data base is low because corroborating data are not available. Confidence in the RfD is also low, reflecting the low confidence in the study and data base (USEPA, 1995c).

D. Carcinogenicity

An initial National Toxicological Program (NTP) bioassay reportedly demonstrated equivocal results and the compound was assigned a Class E carcinogenic rating by EPA. In a second study to reevaluate the carcinogenic potential of p-chloroaniline, groups of rats of each sex were administered by gavage doses of 0, 2, 6 or 18 mg/kg in water, 5 days/week for 103 weeks; groups of mice of each sex were given 0, 3, 10, or 30 mg/kg, 5 days/week for 103 weeks. Male rats at all dose levels showed increased fibrosis of the spleen and sarcomas; female rats at 18 mg/kg showed fibrosis of the spleen. Male mice had increased incidence of hepatocellular adenomas or carcinomas. The authors concluded that p-chloroaniline was carcinogenic in male rats and male mice (Patty's, 1994).

E. Mutagenicity

No data available.

F. Teratogenicity/Reproductive Effects

Information regarding teratogenicity or other reproductive effects of p-chloroaniline is not available (USEPA, 1995c).

G. Other Health Effects

No data available.

H. Epidemiological Evidence

No data available.

I. Toxicity Data

4-chloroaniline had not been evaluated for carcinogenicity by USEPA (1995c). The chronic reference dose for an adult is 0.004 mg/kg-day for the oral route (USEPA, 2004a).

2.4 4-METHYLPHENOL (P-CRESOL)

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol): 108.14

Melting Point:	34.74°C
Boiling Point:	85.7°C (@ 10 mm Hg)
Specific Density:	1.0178 (@ 20°C)
Water Solubility (mg/L):	21,520 (@ 25°C)
Vapor Pressure (mm Hg):	0.11 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	7.92×10^{-7} (@ 25°C)

Reference: ATSDR 1992c. Toxicological Profile for Cresols.

B. Chemical Transformation

Air: Cresols degrade rapidly in the atmosphere. During the day, the primary removal process is the reaction with hydroxyl radicals. The hydroxyl radicals react with cresols by attacking the carbon bearing the hydroxyl group. By-products from this reaction include nitrocresols and products or ring opening to include pyruvic acid, acetaldehyde, formaldehyde and peroxyacetylnitrate (ATSDR, 1992c). At night, nitrate radical reactions are the primary removal process since hydroxyl radical concentrations decrease. Nitrate radicals react with cresols by removing the hydroxyl hydrogen, yielding a phenoxy radical.

Water: Biodegradation of cresols in water appears to be the primary loss process. Based on the results of one study, biodegradability of cresols appear to exist in the order of p-cresol > o-cresol > m-cresol in waters (ATSDR, 1992c). The degradation process of p-cresol in groundwater appears to proceed by oxidation of the methyl group to first give the corresponding benzaldehyde, then benzoic acid. The hydroxybenzoic acid then can be either decarboxylated or dehydroxylated to phenol or benzoic acid, respectively (ATSDR, 1992c).

Soil: Based on all available information, cresols degrade rapidly in soils, possibly incorporated into soil microorganisms, but they mineralize slowly (ATSDR, 1992c).

2. Toxicological Properties

A. Metabolism

Cresols are absorbed across the respiratory tract, gastrointestinal system and skin. Dermal absorption is rapid, however, rate of absorption is dependant on size of area exposed rather than concentration of the chemical. The primary metabolic pathway for cresols is conjugation with glucuronic and sulfuric acid. Minor metabolic pathway for cresols include hydroxylation of the benzene ring and side-chain oxidation to hydroquinone and pyrocatechin. The major route of elimination of cresols is renal excretion in the form of conjugates. Cresols are excreted in animals (rabbit) primarily as oxygen conjugates; 60% to 70% as ether glucuronides and 10% to 15% as etheral sulfates (HSDB, 2004).

B. Acute Toxicity

Deaths, as a result of incidental or intentional ingestion of Lysol (which contained approximately 50% mixed cresols, and/or other cresol formulations), have been reported. The lethal oral exposure level for humans is estimated to be at or above 2 g/kg. Lethal doses of cresols in animals varied from 1,350 to 2,020 mg/kg in orally exposed rats and 300 to 2,830 mg/kg in dermally exposed rabbits, depending on the isomer tested (ATSDR, 1992c).

Following ingestion of highly concentrated solutions (e.g., Lysol), systemic effects reported in humans include mouth and throat burns, abdominal pain, vomiting, hemolytic anemia, and impaired kidney function. Following inhalation, mucosal irritation was observed; and anuria, elevated blood urea levels, and severe skin corrosion were reported following dermal contact with highly concentrated solutions. Comas were frequently reported in humans exposed to high levels of cresols, either by ingestion or by dermal contact (ATSDR, 1992c).

C. Subacute and Chronic Toxicity

Chronic exposure to low concentration of cresols through the skin, mucous membranes, or respiratory tract may cause chronic systemic poisoning. Symptoms include vomiting, difficulty swallowing, salivation, diarrhea, loss of appetite, headache, fatigue, dizziness, mental disturbances, and skin rash. Death may result if there has been severe damage to the liver and kidneys (HSDB, 2004).

In rats, intermediate-duration oral exposures to o- or m-cresol produced reductions in body weight gain and occasional organ weight changes. In addition to these effects, p-cresol produced more notable changes such as increased incidence of epithelia metaplasia in the trachea, mild reductions in hemoglobin, hematocrit and red blood cell counts, increased serum transaminase levels, and mild nephropathy (ATSDR, 1992c).

D. Carcinogenicity

USEPA classifies the three cresol isomers as possible human carcinogens (Group C) with limited evidence of carcinogenicity in animal studies. The basis for the classification is an increased incidence of skin papillomas in mice in an initiation-promotion study (USEPA, 1991d).

E. Mutagenicity

The genotoxic effects of cresols have been well studied. The test results in mammalian cells indicate that cresols can react with DNA of humans and other mammals to produce genotoxic effects *in vitro* (ATSDR, 1992c). USEPA toxicity profiles (1991d) reported the isomers produced positive results in genetic toxicity studies both alone and in combination.

F. Teratogenicity/Reproductive Effects

No human studies were located. Based on animal data, it is not likely that cresols pose a serious developmental hazard to humans; however, the fact that they produce some effects on the developing fetus in animals suggest that care should be taken to limit exposure in pregnant women (ATSDR, 1992c). Results in animal studies suggest that cresols are not likely to result in reproductive effects in animals or humans (ATSDR, 1992c).

G. Other Health Effects

Dermal contact of cresols can cause serious burns. Although sensation is not immediate, prickling and extensive burning occur after a few moments, note that the skin is red. This is followed by loss of sensory feeling. Then the affected skin shows wrinkling, white discoloration and softening (blisters). Depending on length of contact and concentration of the cresol, eczema and ulceration may occur. Later the white patches may turn a brown or black discoloration indicating signs of gangrene (HSDB, 2004). Ocular contact of cresols may result in extensive damage and blindness.

H. Epidemiological Evidence

Epidemiological studies is limited to reports regarding no relationship between urinary levels of endogenous p-cresol and bladder and bowel cancer. No other studies were found (ATSDR, 1992c).

I. Toxicity Data

USEPA has classified cresols as Group C, a possible human carcinogen. The chronic oral reference dose for 4-methylphenol is 0.005 mg/kg-day (HEAST, 1997).

2.5 ALPHA-PICOLINE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	93.13
Melting Point:	-70°C
Boiling Point:	128°C
Specific Density:	0.9443 (@ 20°C)
Water Solubility (mg/L):	miscible in water
Vapor Pressure (mm Hg):	11.2 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	9.96×10^{-6} (@ 25°C)

Reference: Hazardous Substances Databank (HSDB, 2004).

B. Chemical Transformation

Air: Alpha-picoline exists solely as a vapor in the ambient atmosphere. Vapor-phase 2-methylpyridine is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 15 days. Reaction with vapor-phase nitric acid may be important in a polluted atmosphere; this reaction may be dominant for pyridine in some situations and may be similarly important for 2-methylpyridine (HSDB, 2004).

Water: Volatilization half-lives for a model river and model lake are 4 and 30 days, respectively. The potential for bioconcentration in aquatic organisms is low. In aerobic environments, 2-methylpyridine should biodegrade fairly rapidly while it is expected to be resistant to biodegradation under anaerobic conditions. 2-Methylpyridine was completely biodegraded in 4 days in aerobic groundwater (HSDB, 2004).

Soil: Complete biodegradation of 2-methylpyridine occurred within the first 2 weeks of exposure to unpolluted surface soil (with no pyridine derivatives) under aerobic conditions, but was unsuccessful under anaerobic conditions (both denitrifying and sulfidogenic conditions) with only 10% biodegradation reported in 3 months. 2-Methylpyridine was completely biodegraded within 2 weeks of exposure to contaminated (with pyridine derivatives) surface and subsurface soils under aerobic conditions. Under anaerobic conditions (both denitrifying and sulfidogenic conditions) in polluted soils, only 30% of the initially present 2-methylpyridine was degraded in 3 months (HSDB, 2004).

2. Toxicological Properties

A. Metabolism

Pyridine and its alkyl derivitives are absorbed from GI tract, intraperitoneal cavity and lungs. Peritoneal absorption is apparently only slightly more rapid and complete than GI absorption. Alkyl derivatives of alpha-picoline was rapidly absorbed by blood and penetrated into liver, heart, spleen, lungs, and muscles during 1st 10 to 20 minutes following oral admin of 0.5 g/kg to rats. It was excreted in urine during the first 48 hours picoline (HSDB, 2004).

In rabbits and dogs 2-methylpyridine is oxidized to alpha-picolinic acid which is excreted in the urine. Alpha-Picolinuric acid is also excreted by frogs administered alpha-picoline, but only in amounts less than 1% of the dose. In hens it is excreted partly as alpha-pyridinornithuric acid. It was observed that 96% of a 100 mg/kg oral dose of 2-methylpyridine administered to rats was excreted in the urine as picolinuric acid picoline (HSDB, 2004).

There is evidence that 2-methylpyridine forms an N-methylated derivative in dogs. Addition of a methyl group onto the pyridine molecule increases the rate of absorption of the resultant picolines into the liver, kidney, and brain of rats. After ip injection of the picolines, pharmacokinetic parameters were greatly dependent upon the position of the methyl group. For example, the residence time for beta-picoline in liver, brain, and kidney was greater than that of alpha- or gamma-picoline. Elimination of all four pyridines was biphasic in nature, the first phase being more prolonged for pyridine and beta-picoline than for alpha- or gamma-picoline (HSDB, 2004).

B. Acute Toxicity

Exposures to the picolines may give rise to flushing of the face, skin rash, an increase in heart and respiration rates, headache, giddiness, nausea and vomiting (HSDB, 2004).

C. Subacute and Chronic Toxicity

2-Methylpyridine causes local irritation on contact with the skin, mucous membranes, and cornea. Clinical signs of intoxication caused by the methylpyridines include weight loss, diarrhea, weakness, ataxia, and unconsciousness as well as narcosis headache. Also, methylpyridine results in anemia, ocular, and facial paralysis in addition to the previously mentioned symptoms (HSDB, 2004).

D. Carcinogenicity

No data available.

E. Mutagenicity

No data available.

F. Teratogenicity/Reproductive Effects

No data available.

G. Other Health Effects

It has been alleged that workmen exposed to picoline vapors may develop diplopia as a result of disturbance of the eye muscles. Picoline vapors have also been found to be moderately toxic and an irritant (HSDB, 2004).

H. Epidemiological Evidence

No data available.

I. Toxicity Data

No data available.

2.6 BIS(2-ETHYLHEXYL)PHTHALATE (BEHP)

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	390.57
Melting Point:	-47°C
Boiling Point:	384°C
Specific Density:	0.984
Water Solubility (mg/L):	0.041 (@ 25°C)
Vapor Pressure (mm Hg):	1.0×10^{-7} (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	1.71×10^{-5} (@ 25°C)

Reference: ATSDR 2002a. Toxicological Profile for Di(2-ethylhexyl)phthalate.

B. Chemical Transformation

Air: BEHP is ubiquitous in the atmosphere at low concentrations. BEHP does not evaporate easily and so volatilization of BEHP is considered minimal. BEHP will strongly adsorb to particulates and is subject to dry and wet deposition (ATSDR, 2002a).

Water: Biodegradation may be an important fate process for BEHP under aerobic, but not anaerobic conditions (ATSDR, 2002a).

Soil: Biodegradation of BEHP also occurs in soil, but at a slightly slower rate than in water, since BEHP tends to adsorb to particulates (ATSDR, 2002a).

2. Toxicological Properties

A. Metabolism

BEHP is lipophilic and tends to accumulate in the adipose tissues. Based on data from both human and animal studies, the metabolism of BEHP involved a complex series of reactions with the production of 30 or more metabolites (ATSDR, 2002a). The first step in metabolism is the lypolitic cleavage of BEHP resulting in the formation of mono(2-ethylhexyl)phthalate (MEHP) and 2-ethylhexanol (ATSDR, 2002a). 2-ethylhexanol is further metabolized yielding primary urinary products of 2-ethylhexanoic acid and several ketone acid derivatives. MEHP primarily undergoes oxidation of the aliphatic side chain to yield several various metabolites. Although some MEHP is absorbed into the bloodstream, MEHP is poorly absorbed , so that much of the ingested BEHP is excreted in the feces (ATSDR, 2002a).

B. Acute Toxicity

There is currently no evidence of adverse health effects from studies involving human exposure to BEHP, but there are two cases where individuals ingested a single, large dose of BEHP. The individuals reported gastrointestinal distress, symptoms include mild abdominal

pain and diarrhea. Numerous dose oral studies in animals have reported that the main target organs of BEHP to be the liver and testes. Characteristic hepatic effects in rats and mice were observed in numerous studies including hypertrophy and hyperplasia, usually beginning within 24 hours of exposure (ATSDR, 2002a).

C. Subacute and Chronic Toxicity

Intermediate and chronic exposures to BEHP have profound effects on the rodent liver which may include: increased hyperplasia, increased liver weight, decreased cholesterol synthesis and degradation, decreased liver glycogen, and alterations in the morphology of the bile duct. Toxicity of BEHP in other tissues is less well characterized, although effects in the kidney, thyroid, ovaries, and blood have been reported in a few studies. Chronic exposure studies have revealed focal cystic changes in the kidney cells of rats, among other adverse effects of BEHP exposure. (ATSDR, 2002a).

D. Carcinogenicity

No studies were located regarding cancer in humans after exposure to BEHP. Orally administered BEHP produced significant dose-related increase in the incidence of hepatocellular carcinomas and combined incidence of carcinomas and adenoma were observed in female rats and both sexes of mice. The combined incidence of neoplastic nodules and hepatocellular carcinomas was statistically significantly increased in the high-dose male rats. A positive dose response trend was also noted. The USEPA has placed BEHP into Group B2, classified a probable human carcinogen, due to sufficient evidence of carcinogenicity in animal studies (USEPA, 1993c).

E. Mutagenicity

BEHP has been extensively tested for genotoxicity in a variety of *in vitro* and *in vivo* microbial and mammalian assay systems with results that are predominantly negative or false positive. The weight of evidence indicates that BEHP is not genotoxic, but exerts multiple effects by an epigenetic mechanism that can alter the expression of genes in cells (ATSDR, 2002a).

F. Teratogenicity/Reproductive Effects

Several animal studies reported a range of developmental effects including intra-uterine deaths, skeletal and cardiovascular malformations, neural tube closure defects, increased perinatal mortality and developmental delays. There are numerous studies in rats and mice where BEHP exposure has led to reproductive effects such as testicular damage and affected reproductive performance (ATSDR, 2002a). There are insufficient data for estimating the risk to humans from exposure to BEHP during gestation or the potential reproductive effects due to exposure at low levels of BEHP (ATSDR, 2002a).

G. Other Health Effects

There is limited information available regarding the health effects of BEHP following inhalation or dermal exposure. Inhalation of BEHP, lung disorders resembling hyaline membrane disease were observed during the 4th week of life in three children who had received respiratory ventilation via PVC tubing as preterm infants. Although the information is complicated by cofounding variables, the information suggests lung disorders were related to BEHP released from the walls of the respiratory tubing. One inhalation study on rats reported increased lung and liver weights and histological changes in the lungs following cessation of exposure. One dermal study found no indications of skin irritation or sensitization to BEHP (ATSDR, 2002a).

H. Epidemiological Evidence

No data available.

I. Toxicity Data

USEPA has classified BEHP as a probable human carcinogen with sufficient evidence in animal studies. The chronic oral cancer slope factor is 0.014 (mg/kg-day)-1 (USEPA, 2004a) and the chronic inhalation cancer slope factor is 0.014 (mg/kg-day)-1 (USEPA, 2004b). The chronic oral reference dose is 0.02 mg/kg-day, USEPA 2004a.

2.7 POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

1. Constituent Properties

A. Physical and Chemical Properties

- *low molecular weight PAHs* - acenaphthene, anthracene, phenanthrene
- *medium molecular weight PAHs* - fluoranthene and pyrene; and
- *high molecular weight PAHs* - benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene.

Acenaphthene

Atomic Weight (g/mol):	154.21
Melting Point:	95°C
Boiling Point:	96.2°C
Specific Density:	1.225 (@ 0°C)
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	4.47×10^{-3} (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: The chemical and physical properties for this and all subsequent PAHs are from ATSDR, 1994b. Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs).

Anthracene

Atomic Weight (g/mol):	178.2
Melting Point:	218°C
Boiling Point:	342°C
Specific Density:	no data
Water Solubility (mg/L):	virtually insoluble
Vapor Pressure (mm Hg):	1.7×10^{-5} (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	8.6×10^{-5}

Benz(a)anthracene [BAA]

Atomic Weight (g/mol):	228.29
Melting Point:	158-159°C
Boiling Point:	400°C
Specific Density:	1.274 (@ 20°C)
Water Solubility (mg/L):	virtually insoluble
Vapor Pressure (mm Hg):	2.2×10^{-8} (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	1.0×10^{-6}

Benzo(a)pyrene [BAP]

Atomic Weight (g/mol):	252.3
Melting Point:	179°C
Boiling Point:	310-312°C
Specific Density:	1.351
Water Solubility (mg/L):	3.8
Vapor Pressure (mm Hg):	5.6×10^{-9}
Henry's Law Constant (atm-m^3/mol):	4.9×10^{-7}

Benzo(b)fluoranthene [BBF]

Atomic Weight (g/mol):	252.3
Melting Point:	168.3°C
Boiling Point:	no data
Specific Density:	no data
Water Solubility (mg/L):	virtually insoluble
Vapor Pressure (mm Hg):	1.0×10^{-11} - 1.0×10^{-6} (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	1.22E-05

Benzo(k)fluoranthene [BKF]

Atomic Weight (g/mol):	252.3
Melting Point:	215.7°C
Boiling Point:	480°C
Specific Density:	no data
Water Solubility (mg/L):	virtually insoluble
Vapor Pressure (mm Hg):	5.0×10^{-7} (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	3.87E-05

Benzo(g,h,i)perylene [BP]

Atomic Weight (g/mol):	276.34
Melting Point:	273°C
Boiling Point:	222°C

Specific Density:	no data
Water Solubility (mg/L):	2.6E-03 (@ 25°C)
Vapor Pressure (mm Hg):	1.03×10^{-10} (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	1.44×10^{-7}

Dibenz(a,h)anthracene [DA]

Atomic Weight (g/mol):	278.35
Melting Point:	262°C
Boiling Point:	269-270°C
Specific Density:	1.282
Water Solubility (mg/L):	5.0E-07
Vapor Pressure (mm Hg):	1.0×10^{-10} (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	7.3×10^{-8}

Fluoranthene

Atomic Weight (g/mol):	202.26
Melting Point:	111°C
Boiling Point:	375°C
Specific Density:	no data
Water Solubility (mg/L):	soluble in sea water at 22°C
Vapor Pressure (mm Hg):	0.01 (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	6.5×10^{-6}

Indeno(1,2,3-cd)pyrene [IP]

Atomic Weight (g/mol):	276.3
Melting Point:	163.6°C
Boiling Point:	530°C
Specific Density:	no data
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	$\sim 10.0 \times 10^{-10}$ (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	6.95×10^{-8}

Phenanthrene

Atomic Weight (g/mol):	178.2
Melting Point:	100°C
Boiling Point:	340°C
Specific Density:	0.900 (@ 4°C)
Water Solubility (mg/L):	virtually insoluble
Vapor Pressure (mm Hg):	9.6×10^{-4} (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	2.26×10^{-4}

Pyrene

Atomic Weight (g/mol):	202.3
Melting Point:	156°C
Boiling Point:	393°C
Specific Density:	no data
Water Solubility (mg/L):	virtually insoluble
Vapor Pressure (mm Hg):	2.5×10^{-6} (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	5.1×10^{-6}

B. Chemical Transformation

Air: PAHs can undergo photooxidation and can react in the atmosphere with pollutants such as ozone, nitrogen oxides, sulfur dioxide, and peroxyacetylnitrate (ATSDR, 1994b). Reaction with ozone or peroxyacetylnitrate yields diones; nitrogen oxide reactions yield nitro and dinitro PAHs. Sulfuric acids have also been formed from reaction with sulfur dioxide (ATSDR, 1994b). Photochemical oxidation of a number of PAHs has been reported with the formation of nitrated PAHs, quinones, phenols, and dihydrodiols (ATSDR, 1994b).

Water: The most important processes contributing to the degradation of PAHs in water are photooxidation, chemical oxidation, and biodegradation by aquatic organisms (ATSDR, 1994b). In the photooxidation of PAHs, the most common reactions result in the formation of peroxides, quinones, and diones (ATSDR, 1994b). In one study algae were found to transform BAP to oxides, peroxides, and dihydrodiols.

Soil: Microbial metabolism is the major process for degradation of PAHs in soil environments (ATSDR, 1994b). Metabolism of PAHs by bacteria and fungi includes formation of dihydrodiols.

2. Toxicological Properties

A. Metabolism

The lipophilicity of PAHs enables them to readily penetrate cellular membranes and remain in the body indefinitely. However, the metabolism of PAHs renders them more water-soluble and more excretable (ATSDR, 1994b). The metabolic process involves several possible pathways with varying degrees of enzyme activities. The activities and affinities of the enzymes in a given tissue determine which metabolic route will prevail (ATSDR, 1994b). The structural similarity of PAHs contributes to the similarities that exist in their biotransformation, thus, BAP metabolism, which has been extensively reviewed, will be used as a model for PAH metabolism.

BAP is metabolized initially into several arene oxides, which may be rearranged to phenols, undergo hydration to form trans-dihydrodiols, or be further metabolized to yield various quinone isomers. The phenols, quinones, and dihydrodiols can all be conjugated with glucuronides and sulfate esters; the quinones also form glutathione conjugates (ATSDR, 1994b). The route by which PAHs enter the body may determine their fate and organ specificity. For example, an inhaled compound may bypass the liver and reach peripheral tissues in concentrations higher than one would see after oral exposures (ATSDR, 1994b).

The excretion of BAP following low-level inhalation exposure is rapid and high in rats, however, elimination is low in dogs and monkeys. The excretion half-lives of BAP metabolites in the feces and urine of rats exposed once to 4.8 mg/m^3 were 22 and 28 hours, respectively (ATSDR, 1994b).

B. Acute Toxicity

There have been no reports of death in humans following exposure to any of the PAHs (ATSDR, 1994b). However, BAP is fatal to mice following ingestion, and death in animals has been reported following parenteral exposure to a number of PAHs. The intraperitoneal LD50s in mice for pyrene, anthracene, and BAP are 514, >430, and 232 mg/kg, respectively (ATSDR, 1994b).

Hepatic effects have been observed in animals following acute oral exposure to various PAHs. Rats administered 51.4 mg/kg/day acenaphthene or 180 mg/kg/day fluorene resulted in statistically significant increases in liver weight compared to controls, which may have indicated an effect on cell regeneration, although rates of cell proliferation were not determined. These and other hepatic effects are not considered serious, but their incidence and severity have been correlated with carcinogenic potency of particular PAHs (ATSDR, 1994b). Rats administered of 15.4 mg/kg/day acenaphthene, 51.4 mg/kg/day BAP, or 514 mg/kg/day pyrene, anthracene, or phenanthrene had no effects on the liver (ATSDR, 1994b).

Adverse skin effects associated with dermally administered PAHs have been reported following acute exposures.

C. Subacute and Chronic Toxicity

PAHs are contained in cigarette smoke, and chronic smoking is a well-established risk factor in the development of atherosclerosis (ATSDR, 1994b). Arterial smooth muscle cell proliferation, collagen synthesis, lipid accumulation, and cellular necrosis are all involved in atherosclerosis. BAP has been demonstrated to affect the aforementioned processes in studies with bovine, rabbits, and humans (ATSDR, 1994b). Adverse skin effects associated with dermally administered BAP have been reported following intermediate-duration exposures.

Adverse hematological effects have been observed in mice following oral PAH exposure to 120 mg BAP/kg/day for 180 days. The effects, which were associated with death, included a small spleen, marked cellular depletion, prominent hemosiderosis, and follicles with large lymphocytes (ATSDR, 1994b).

D. Carcinogenicity

Available data from animal studies indicate that certain PAHs (i.e., benz(a)anthracene, BAP, and dibenz(a,h)anthracene) are carcinogenic to animals by the oral route (ATSDR, 1994b). The results of

dermal studies indicate that benz(a)anthracene, BAP, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene are tumorigenic in mice following dermal exposure (ATSDR, 1994b). BAP has consistently been demonstrated to be one of the most potent of the carcinogenic PAHs and therefore, it is the most studied. Available literature on PAHs are primarily related to BAP. A quantitative cancer risk estimate (i.e., cancer slope factor or CSF) has been developed for BAP only.

In the past, estimates of cancer risks for other carcinogenic PAHs were calculated using a BAP one-to-one equivalency approach which overestimated the carcinogenic potency of most carcinogenic PAHs. Research on relative potencies for PAHs and on the development of a Toxicity Equivalency Factor (TEF) methodology is currently being undertaken by the Agency (USEPA, 2004b). In the interim, USEPA has provided a series of relative potency values to serve as temporary guidance for risk evaluation of PAHs.

USEPA has performed weight-of-evidence evaluations of several of the PAHs discussed herein. The carcinogenicity classifications currently verified by USEPA's Carcinogenicity Risk Assessment Verification Endeavor Work Group identifies benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, BAP, chrysene and indeno(1,2,3-cd)pyrene as probable human carcinogens (Group B2) with sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans. USEPA identifies acenaphthene, anthracene, benzo(g,h,i)perylene, fluoranthene, fluorene, phenanthrene and pyrene as not classifiable (Group D) as to human carcinogenicity due to inadequate or lack of evidence.

E. Mutagenicity

Results of genetic toxicity studies involving BAP indicate it produces damage in prokaryotes, eukaryotes, and mammalian cells *in vitro* and produces a wide range of genotoxic effects (gene mutations in somatic cells, chromosome damage in germinal and somatic cells, DNA adduct formation, sister chromatid exchange, and neoplastic cell transformation) (ATSDR, 1994b).

The results of genotoxicity for acenaphthene, acenaphthylene, and fluorene are consistently negative, however, none of these compounds have been extensively tested (ATSDR, 1994b). The majority of data for anthracene and pyrene were also negative. Studies involving phenanthrene have found a lack of genotoxicity, possibly related to its metabolism. There is ample evidence suggesting fluoranthene may cause gene mutations in bacteria, and human lymphoblasts and sister chromatid exchange in Chinese hamster cells (ATSDR, 1994b). The weight of evidence from studies conducted with benz(a)anthracene, dibenz(a,h)anthracene, and chrysene indicates that these three agents are

genotoxic (ATSDR, 1994b). Benzo(b)fluoranthene, benzo(k)fluoranthene, and indeno(1,2,3-cd)pyrene are known to exhibit mutagenic activity in bacteria and some animal tests (ATSDR, 1994b).

F. Teratogenicity/Reproductive Effects

Reproductive effects resulting from PAH exposure have been observed in both animal and human studies. Results of these studies demonstrate the reproductive toxicity of BAP, and that adverse reproductive effects may occur in humans exposed to BAP (ATSDR, 1994b). Developmental effects have been observed in animals following PAH exposure. The relevance of findings in animal studies with regard to human exposure is that BAP may produce adverse effects in the unborn and offspring of women exposed to BAP (ATSDR, 1994b). Other PAHs such as anthracene, chrysene, and dibenz(a,h)anthracene have also been tested for developmental effects, with dibenz(a,h)anthracene producing fetolethal effects in rats, and chrysene producing liver tumors in mouse progeny (ATSDR, 1994b).

G. Other Health Effects

Pathological changes in the respiratory mucosa of the trachea, as well as other adverse respiratory effects, have been observed in animal studies after PAH exposure. Anthracene has been associated with gastrointestinal toxicity in humans (ATSDR, 1994b). The carcinogenic PAHs as a group have an immunosuppressive effect (ATSDR, 1994b). BAP has been shown to markedly inhibit the immune system, especially T-cell dependent antibody production by lymphocytes (ATSDR, 1994b).

H. Epidemiological Evidence

Epidemiological data from occupational studies of workers exposed to mixtures of PAHs, as a result of their involvement in such processes as coke production, roofing, oil refining, or coal gasification, indicate carcinogenic effects to humans (ATSDR, 1994b). However, it is not clear whether PAHs are the causative agent.

I. Toxicity Data

Acenaphthene has not been evaluated for carcinogenicity by the USEPA. Benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene, have been placed into Group B2, probable human carcinogen based on sufficient carcinogenicity in animal studies. Anthracene, benzo(g,h,i)perylene, fluoranthene, phenanthrene and pyrene have been placed into Group D, not classifiable as a human carcinogen. The chronic cancer slope factors for the oral and inhalation routes, and the chronic reference doses for the oral and inhalation routes are presented in the table below. Toxicity values were taken from either USEPA, 2004a or USEPA, 2004b.

Chemical Name	*CSForal* $(mg/kg\text{-}d)^{-1}$	*CSF inhalation* $(mg/kg\text{-}d)^{-1}$	*RfD oral* *mg/kg-day*	*RfD Inhalation* *mg/kg-day*
acenaphthene	--	--	0.06	--
benzo(a)anthracene	0.73	--	--	--
benzo(a)pyrene	7.3	3.1	--	--
benzo(b)fluoranthene	0.73	--	--	--
benzo(k)fluoranthene	0.073	--	--	--
dibenz(a,h)anthracene	7.3	--	--	--
indeno(1,2,3-cd)pyrene	0.73	--	--	--
anthracene	--	--	0.3	--
benzo(g,h,i)perylene	--	--	0.03	--
fluoranthene	--	--	0.04	--
phenanthrene	--	--	0.03	--
pyrene	--	--	0.03	--

2.8 PHENOL

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	94.144
Boiling Point:	181.84°C
Melting Point:	40.85°C
Specific Density:	1.07 (@ 20°C)
Water Solubility (mg/L):	86,600 (@ 25°C)
Vapor Pressure (mm Hg):	0.41 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	3.97×10^{-7} (@ 25°C)

Reference: ATSDR, 1998. Toxicological Profile for Phenol.

B. Chemical Transformation

Air: The major removal process for phenol in the atmosphere is reaction with hydroxyl radicals.

Water: Phenol is readily biodegradable in natural waters. In an aqueous, oxygenated solution exposed to artificial light, phenol photolyzed to hydroquinone, catechol, 2,2'-, 2,4'-, and 4,4'-dihydroxy-biphenyl (Montgomery and Welkom, 1990).

Soil: Data indicates that phenol will biodegrade in soil under both aerobic and anaerobic conditions (ATSDR, 1998).

2. Toxicological Properties

A. Metabolism

Four urinary metabolites of phenol have been identified in mammals: phenyl glucuronide, phenyl sulfate, 1,4-dihydroxybenzene glucuronide, and 1,4-dihydroxybenzene sulfate (ATSDR, 1998). In addition to the above mentioned metabolites, it is has been suggested that carbon dioxide can be a significant metabolite of phenol in rabbits when given an oral dose approaching lethal levels (0.3 to 0.5 mg/kg) (ATSDR, 1998).

B. Acute Toxicity

Reported deaths associated with phenol are due to intentional oral exposure (suicides) and accidents involving exposure of a large fraction of the skin surface (>25%) to concentrated phenol solutions. The minimal lethal oral dose in humans was estimated to be ~140 mg/kg, which is similar to the lethal oral dose for a variety of animal species (ATSDR, 1998).

In humans, application of concentrated solutions of phenol to the skin (>*in vivo* 5%) results in inflammation and necrosis at the site of application. The results of animal studies suggest that exposure to as little as 0.5 mg/cm^2 is sufficient to induce skin irritation. Cardiac arrythmias have been reported in individuals undergoing chemical face peels involving application of concentrated solutions of phenol (50%) in combination with hexachlorophene and croton oil to extensive areas of the face. Cardiac arrythmias have been demonstrated in rabbits exposed to 300 mg/kg or ~67 mg/cm^2 of skin (ATSDR, 1998).

Renal toxicity has been reported in laboratory animals for all routes of exposure (inhalation, dermal, oral), but has not been reported in humans. Descriptions of the lesions in the available studies have been poor and it is unclear whether the pathological characteristics vary with route of administration.

C. Subacute and Chronic Toxicity

Elevated concentrations of hepatic enzymes in serum, suggestive of liver injury, were reported in an individual who had been exposed repeatedly to phenol vapor. It was uncertain whether or not the elevated enzymes resulted from liver injury, and whether the symptoms were caused by the phenol exposure.

Hepatotoxicity, characterized histologically by centrilobular necrosis, and elevated hepatic enzymes in serum were reported in separate studies in which laboratory animals were exposed to phenol in air at concentrations > 26 ppm (ATSDR, 1998). Numerous other serious systemic effects have been reported in animals, however, the relevance of these effects to human health is unclear. Serious injuries to the lungs, myocardial necrosis, and hepatic and renal injury were reported in one study in

which guinea pigs and rabbits were exposed repeatedly to 26 ppm phenol in air for 74 to 88 days (ATSDR, 1998).

D. Carcinogenicity

No evidence was found for carcinogenicity of phenol in humans. Phenol has been tested in animals for carcinogenicity by the oral and dermal routes, but results are equivocal. Given the mixed results of carcinogenicity studies, it is difficult to draw conclusions regarding the human carcinogenic potential of phenol. The animal data do not provide sufficient evidence to classify phenol as a carcinogen in humans (ATSDR, 1998). USEPA has not classified phenol as a human carcinogen (Group D) due to inadequate or no evidence of carcinogenicity in animals.

E. Mutagenicity

Phenol has been evaluated for genotoxicity in a variety of test systems. Both negative and positive results for gene mutations have been reported in bacterial tests. Positive results were reported for several *in vitro* mammalian systems. Because of the positive results reported in mammalian tests, phenol may have mutagenic potential (ATSDR, 1998).

F. Teratogenicity/Reproductive Effects

No human evidence was located. Teratogenic effects were observed in rats and mice at oral dose levels that resulted in maternal toxicity (120 mg/kg/day). As a result, phenol cannot be classified as a pure teratogen (ATSDR, 1998).

G. Other Health Effects

Signs of gastrointestinal irritation, including mouth sores and diarrhea, have been reported in humans exposed to drinking water containing 5 to 126 ppm phenol (ATSDR, 1998). Neurological effects such as muscle tremors, loss of coordination, paralysis, and convulsions are indicative of phenol poisoning in laboratory animals (ATSDR, 1998). Descriptions of similar effects in humans were not found.

H. Epidemiological Evidence

No data available.

I. Toxicity Data

USEPA has placed phenol into Group D, not classifiable as a human carcinogen. The chronic reference dose is 0.6 mg/kg-day for the oral route (USEPA, 2004a).

2.9 PYRIDINE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	79.10
Boiling Point:	115.2°C
Melting Point:	-41.6°C
Specific Density:	0.98272 (@ 20°C)
Water Solubility (mg/L):	miscible in water
Vapor Pressure (mm Hg):	20.8 (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	1.1 x 10^{-13}(@ 25°C)

Reference: Hazardous Substances Databank (HSDB, 2004).

B. Chemical Transformation

Air: If released to air, pyridine will exist solely as a vapor in the ambient atmosphere. Vapor-phase pyridine will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 43 days, calculated from its rate constant of 3.7 x 10^{-13} cu cm/molecule-sec at 25 degrees C (HSDB, 2004).

Water: Pyridine was biodegraded in about 8 days in a river die-away test, suggesting biodegradation occurs rapidly in natural waters. Pyridine may undergo indirect photolysis in sunlit surface waters (HSDB, 2004).

Soil: Pyridine was shown to undergo complete biodegradation in soils within 66 to 170 and 32 to 66 days under aerobic and anaerobic conditions, respectively (HSDB, 2004).

2. Toxicological Properties

A. Metabolism

A study in humans, rats, and guinea pigs indicates that pyridine can be absorbed by these species via the oral route. Estimates of the extent of inhalation and dermal routes and calculations of the rates of absorption via all three routes would be useful in helping to compare relative potential risks due to the presence of pyridine in various environmental media (HSDB, 2004).

B. Acute Toxicity

A woman was decontaminating a spill for 15 to 20 minutes. Symptoms did not occur for 10 hours and intensified until the third day. Symptoms included speech disorders and "rather diffused cortical affliction" which receded after thiamine therapy. Upper respiratory symptoms were not present at all. May cause CNS depression, irritation of skin and respiratory tract. Large doses may produce GI disturbances, kidney and liver damage (HSDB, 2004).

C. Subacute and Chronic Toxicity

Most of the effects observed in humans have been caused by repeated or intermittent exposure to the vapor. Clinical symptoms and signs of intoxication include GI disturbance with diarrhea, abdominal pain, nausea, weakness, headache, insomnia and nervousness. Exposure less than those required to produce overt clinical signs may cause varying degrees of liver damage with central lobular fatty degeneration, congestion and cellular infiltration; repeated low-level exposures cause cirrhosis. The kidney is less sensitive to pyridine-induced damage than is the liver. In general, pyridine and its derivatives cause local irritation on contact with the skin, mucous membranes, and cornea (HSDB, 2004).

D. Carcinogenicity

This substance/agent has not undergone a complete evaluation and determination under USEPA's IRIS program for evidence of human carcinogenic potential (HSDB, 2004).

E. Mutagenicity

There is limited information on the mutagenicity of pyridine (HSDB, 2004).

F. Teratogenicity/Reproductive Effects

No studies were located regarding reproductive effects in humans or animals after oral exposure to pyridine (HSDB, 2004).

G. Other Health Effects

May cause smarting of the skin and first degree burns on short exposure, and secondary burns on long exposure (HSDB, 2004).

H. Epidemiological Evidence

After vapor inhalation in humans, central nervous system depression has occurred. Small oral doses (2 to 3 mL) has produced mild anorexia, nausea, fatigue, and mental depression. Ingestion of several ounces has produced severe vomiting, diarrhea, hyperpyrexia, delirium, and death. Autopsy revealed pulmonary edema and membrane tracheobronchitis (HSDB, 2004).

I. Toxicity

Pyridine has not been evaluated for carcinogenicity by the USEPA. The chronic reference dose is 0.001 mg/kg-day for the oral route, (USEPA, 2004a).

3.0 PESTICIDES/PCBs

3.1 4,4'-DDD, 4,4'-DDE & 4,4'-DDT

1. Constituent Properties

A. Physical and Chemical Properties

4,4'-DDD

Atomic Weight (g/mol):	320.05
Boiling Point:	350°C
Melting Point:	109°C
Specific Density:	1.385
Water Solubility (mg/L):	0.09 (@ 25°C)
Vapor Pressure (mm Hg):	1.35×10^{-6} (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	4.0×10^{-6}

4,4'-DDE

Atomic Weight (g/mol):	318.03
Boiling Point:	336°C
Melting Point:	89°C
Specific Density:	no data
Water Solubility (mg/L):	0.12 (@ 25°C)
Vapor Pressure (mm Hg):	6.0×10^{-6} (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	2.1×10^{-5}

4,4'-DDT

Atomic Weight (g/mol):	354.49
Boiling Point:	decomposes
Melting Point:	109°C
Specific Density:	0.99
Water Solubility (mg/L):	0.025 (@ 25°C)
Vapor Pressure (mm Hg):	1.6×10^{-7} (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	8.3×10^{-6}

Reference: ATSDR, 2002b. Toxicological Profile for 4,4'-DDD, 4,4'-DDE, 4,4'-DDT.

B. Chemical Transformation

Air: In the atmosphere, about 50% of DDT is adsorbed to particulates and the other half exists in the vapor phase. In the vapor phase, DDT reacts with photochemically produced hydroxyl radicals. Both DDE and DDD have high vapor pressures than DDT, therefore a small percentage of these analytes will adsorb to particulates. Direct photolysis may also occur (ATSDR, 2002b).

Water: These compound may be converted by both photodegradation and biodegradation. Direct photolysis primarily occurs in surface water and is dependent on the clarity of water. Biodegradation of DDT in water is reported to be a minor

mechanism and biodegradation of DDD and DDE is slower than that of DDT (ATSDR, 2002b).

Soil: The fate of DDE and DDT can be either one of the following: volatilization, photooxidation or biodegradation. Under aerobic conditions, DDT converts to DDE and under anaerobic conditions, DDT converts to DDD (ATSDR, 2002b).

2. Toxicological Properties

A. Metabolism

DDT is initially metabolized in the liver to DDE and DDD. DDT undergoes reductive dechlorination to DDD. This is further degraded in the kidney to readily excreted DDA. DDE is degraded at a slower rate by dehydro-chlorination. Further metabolism of DDE in the kidney produces the metabolite DDA. Absorbed DDT is primarily excreted in urine, mostly as conjugated DDA with minor amounts excreted in the feces (ATSDR, 2002b).

B. Acute Toxicity

Only one case of fatal poinsoning after accidental oral exposure to DDT was reported. One ounce of 5% DDT in kerosene was ingested by a 1-year-old child. Clinical symptoms included coughing and vomiting followed by tremors and convulsions. The child then became comatose and died. Doses as high as 285 mg/kg had been accidentally ingested by humans with no fatal results (ATSDR, 2002b).

Acute, high exposures of DDT in humans primarily affect the nervous system. DDT, at a low dosage of 6 mg/kg, produced no illnesses in humans but perspiration, headache and nausea were reported. Convulsions in humans have been reported at doses of 16 mg/kg and higher. Higher concentrations of 250 to 1,500 mg DDT orally administered to humans exhibit sensitivity of the lower part of the face, dizziness, cold sweats, tremors, headache, fatigue, malaise, confusion and vomiting. Within 24 hours, all volunteers had recovered completely (ATSDR, 2002b). Rats exposed to a single dose ranging from 50 to 600 mg/kg/day of DDT had reported effects of tremors, abrupt and involuntary contractions of skeletal muscles, hyperexcitability and convulsions (ATSDR, 2002b).

It has been noted that dermal contact with DDT and DDE produced minor skin irritations. Inhalation exposure to DDT and DDE is expected to be minimal since their large particle size restricts inhalation absorption. Instead, they are deposited on the upper respiratory tract where they are swallowed. After inhalation, people complained of irritation to the eyes, nose and throat (ATSDR, 2002b).

C. Subacute and Chronic Toxicity

Chronic exposure to DDT in small amounts would not cause any permanent effects in humans. In a study where workers were exposed to 14 to 42 mg/day, no evidence of hyperexcitability were observed in the workers and neurological examinations were reported to be normal. However in chronic oral exposure, animal studies reported neurological and hepatic effects. These symptoms included increased liver weights to cellular necrosis and hyperactivity and tremors. Based on induction profiles obtained in rats, DDT and related compounds are considered primarily as phenobarbital-type inducers (ATSDR, 2002b).

D. Carcinogenicity

There are several animal studies reporting that DDE and DDT are cancer causing agents. A study where DDT was fed to rats for 2 years caused liver tumors at all dose levels ranging from 10 to 800 ppm and in mice, DDT administered chronically in the diet produced liver tumors at doses of 19 to 34 mg/kg/day for 30 to 78 weeks (USEPA, 1996a; ATSDR, 2002b). Twenty-five animal carcinogenicity assays have been reviewed for DDT. Both hepatocellular adenomas and carcinomas were observed in six mouse liver tumor studies. Both benign and malignant lung tumors were observed in two studies wherein mice were exposed both in utero and throughout their lifetime. Doses producing increased tumor incidence ranged from 0.15 to 37.5 mg/kg/day (USEPA, 1996a). USEPA has classified DDT as B2; probable human carcinogen based on observation of tumors (generally of the liver) in seven studies in various mouse strains and three studies in rats. DDT is structurally similar to other probable carcinogens, such as DDD and DDE.

A study where administered DDE in feed at doses of 148 and 261 ppm to mice for 78 weeks reported an increase in incidence of hepatocellular carcinomas in males and females in comparison with controls. Increased weight loss and mortality was observed in females. Another study where DDE (250 ppm) was administered to mice in feed for lifetime (130 weeks) reported a significant increase in incidence of hepatomas observed in both males and females in comparison with controls. In females, 98% of the 55 surviving exposed animals developed hepatomas, compared to 1% of the surviving controls. Thyroid tumors was observed in female rats who were fed DDE at doses of 242 ppm and 462 ppm for 78 weeks. USEPA has classified DDE as B2; probable human carcinogen based on increased incidence of liver tumors including carcinomas in two strains of mice and in hamsters and of thyroid tumors in female rats by diet (USEPA, 1988a).

Mice fed DDD for 130 weeks at 250 ppm had a statistically significant increase in incidence of lung tumors seen in both sexes compared with controls. In males, a statistically significant increase in incidence of liver tumors was also seen.

One study where mice were given DDD doses of 350 or 630 ppm for 5 weeks, 375 or 750 ppm for 11 weeks, and 425 or 850 ppm for the next 62 weeks reported an increased incidence of hepatocellular carcinomas seen in both sexes by comparison to controls. Another study also fed DDD at 1,647 and 3,294 ppm TWA for males and 850 and 1,700 ppm TWA for females for 78 weeks to rats. Males were fed 1,400 or 2,800 ppm for 23 weeks followed by 1,750 or 3,500 ppm for 55 weeks. Females were fed 850 or 1,700 ppm for the entire 78 weeks. After an additional 35 weeks, an increased incidence of thyroid tumors (follicular cell adenomas and carcinomas) was observed in males. USEPA has classified DDD as B2; probable human carcinogen based on an increased incidence of lung tumors in male and female mice, liver tumors in male mice and thyroid tumors in male rats. DDD is structurally similar to, and is a known metabolite of DDT, a probable human carcinogen (USEPA, 1988b).

E. Mutagenicity

There are human studies to suggest that DDT may cause chromosomal aberrations. Blood cultures of occupationally exposed workers, exhibited an increase in chromatid lesions. Another study reported an increase of sister chromatid exchanges and chromosomal aberrations in peripheral lymphocytes compared to a control group (ATSDR, 2002b). Positive effects were found with DDD in mammalian cytogenetic assays and a host-mediated assay (USEPA, 1988b). DDE was mutagenic in mouse lymphoma (L5178Y) cells and chinese hamster (V79) cells, but not in Salmonella (USEPA, 1988a). DDT has produced both negative and positive responses in tests for genotoxicity. Positive responses have been noted in V79 mutation assays, for chromosome aberrations in cultured human lymphocytes, and for sister chromatid exchanges in V79 and CHO cells. In one study, DDT was reported to interact directly with DNA; this result was not confirmed in the absence of a metabolizing system (USEPA, 1996a).

F. Teratogenicity/Reproductive Effects

After acute and chronic exposures, animal studies reported adverse reproductive effects. These included a decrease in fertility, stillbirths and increase in fetal mortality. DDE and DDT are embryotoxic and fetotoxic. After acute oral exposure of animals to DDT resulted in decreased fetal body and organ weights, increases in resorption and prematurity. Chronic oral exposure resulted in mortality, slowed development and premature puberty (ATSDR, 2002b).

G. Other Health Effects

These compounds may cause immunotoxicity. The effects reported include decreases in antibodys and plaque-forming cells, increases in gamma globulin and serum immunoglobulin, alterations in the spleen and increased growth of the leprosy bacterium. The immunotoxicity of

these compounds is increased by low protein diet and physical/emotional stress (ATSDR, 2002b).

H. Epidemiological Evidence

There are several acute oral exposure studies to humans documenting DDE and DDT are irritants to the eyes, nose, and throat and may cause symptoms such as sweating, nausea, headache, tremors and convulsions (ATSDR, 2002b).

I. Toxicity Data

USEPA has classified these compounds as probable human carcinogens based on sufficient evidence in animal studies. The chronic oral cancer slope factors are 0.24 (mg/kg/day)-1 , 0.34 (mg/kg/day)-1 and 0.34 (mg/kg/day)-1 for DDD, DDE, and DDT, respectively. A chronic inhalation cancer slope factor of 0.34 (mg/kg/day)-1 is reported for DDT. A chronic oral reference dose for DDT is 0.0005 mg/kg-day. The aforementioned toxicity values were was taken from USEPA, 2004a.

3.2 ALDRIN AND DIELDRIN

1. Constituent Properties

A. Physical and Chemical Properties

Aldrin

Atomic Weight (g/mol):	364.91
Melting Point:	104-105.5°C
Boiling Point:	decomposes
Specific Density:	1.6 (@ 20°C)
Water Solubility (mg/L):	0.011 (@ 20°C)
Vapor Pressure (mm Hg):	1.2×10^{-4} (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	4.9×10^{-5} (@ 25°C)

Dieldrin

Atomic Weight (g/mol):	308.93
Melting Point:	175°C
Boiling Point:	330°C
Specific Density:	1.75 (@ 20°C)
Water Solubility (mg/L):	0.18
Vapor Pressure (mm Hg):	1.78×10^{-7} (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	1.51×10^{-5}

Reference: ATSDR, 2002c. Toxicological Profile for Aldrin/Dieldrin.

B. Chemical Transformation

Air: Aldrin will exist solely in the vapor-phase in the ambient atmosphere. Vapor-phase aldrin will be degraded in the atmosphere by reaction with photochemically-produced

hydroxyl radicals; the half-life for this reaction in air is estimated to be 6 hours. Dieldrin will adsorb to particulate matter in air (ATSDR, 2002c).

Water: If released into water, aldrin is expected to adsorb to suspended solids and sediment. Hydrolysis is not expected to occur due to the lack of hydrolyzable functional groups (HSDB, 2004). Dieldrin is photochemically isomerized by sunlight to photodieldrin (ATSDR, 2002c).

Soil: Aldrin was classified as moderately persistent with a half-life in soil ranging from 20 to 100 days. In soil, aldrin is converted to dieldrin by epoxidation, which occurs in aerobic and biologically-active soils (HSDB, 2004). Dieldrin in soils may also be photodecomposed to photodieldrin. Although this compound is resistant to biodegradation it has been reported that dieldrin maybe subjected to aerobic degradation producing aldrin diol (ATSDR, 2002c).

2. Toxicological Properties

A. Metabolism

The dominant reaction of aldrin is epoxidation at double bond to form dieldrin. This oxidation occurs both photochemically and biologically (HSDB, 2004). Several metabolic studies indicate that dieldrin is absorbed from the gastrointestinal tract and is transported via the hepatic portal vein. Two major metabolic routes of dieldrin predominate: (1) direct oxidation by cytochrome oxidases to give 9-hydroxydieldrin, and (2) the opening of the epoxide ring by epoxide hydrases, resulting in 6,7-trans-dihydroxydihydroaldrin (ATSDR, 2002c).

B. Acute Toxicity

Acute toxicity has resulted in malaise, headache, nausea, vomiting, dizziness, and tremors. More serious symptoms include clonic and tonic convulsions, sometimes without premonitory symptoms. Convulsive episodes may alternate with periods of severe central nervous depression. Death from respiratory arrest may occur during coma (HSDB, 2004). Acute high-level exposure of humans to dieldrin has been observed to cause central nervous system excitation culminating in convulsions. The other effect observed in humans after acute high-level exposure is renal toxicity. Adverse effects on the kidneys were observed in rats and dogs exposed to dieldrin. These studies reported degenerative changes in the epithelial cells of the kidney, lymphocyte and macrophage infiltration, vascular congestion in the renal cortex, and hyaline casts in the renal tubules (ATSDR, 2002c).

C. Subacute and Chronic Toxicity

Longer term exposure of humans in occupational settings has been associated with occasional cases of central nervous system intoxication

resulting in convulsions. Following relatively long-term exposure to constant levels of dieldrin, a steady state of body levels is achieved. Other symptoms reported by workers exposed to dieldrin included headaches, dizziness, hyperirritability, anorexia, muscle twitching and jerking. For the most part, studies in animals support the observation of these toxic effects in humans. In addition, studies in animals indicate that other toxic effects may be associated with exposure to sufficiently high levels of dieldrin. These include hepatic degeneration and immunosuppression (ATSDR, 2002c).

D. Carcinogenicity

There are several studies in mice that strongly indicate that aldrin and dieldrin is a promoter of tumors. Mice exposed to aldrin and dieldrin reported hepatocellular tumors and in other studies, dieldrin was expected to induce pulmonary, lymphoid, thyroid and other tumors (ATSDR, 2002c). Based on the conclusion that there is sufficient animal evidence of carcinogenicity, USEPA has classified aldrin and dieldrin as B2, probable human carcinogen.

E. Mutagenicity

Aldrin was not mutagenic to bacteria and did not induce breakage of plasmid DNA. Also Aldrin did not induce dominant lethal mutations in mice (HSDB, 2004). Significant increases in chromosomal aberrations have been reported in cultured human lung cells. Similar results were observed in bone marrow cells in mice treated with dieldrin. They are other animal studies that suggest dieldrin to be mutagenic (ATSDR, 2002c).

F. Teratogenicity/Reproductive Effects

Aldrin had no effect on reproductive functions in male and female offsprings of female rats which had been feed aldrin during pregnancy (HSDB, 2004). Decreased fertility was reported in several studies in which dieldrin was administered to maternal or paternal animals. Hamsters and mice exposed to very high dosages of dieldrin were reported to have external malformations. Decreased postnatal survival was reported in a number of animal studies (ATSDR, 2002c).

G. Other Health Effects

Respiratory effects have been noted in workers manufacturing dieldrin. An increase in pulmonary diseases and pneumonia were reported in these workers (ATSDR, 2002c).

H. Epidemiological Evidence

Workers exposed to dusts of aldrin complained of headache, dizziness, nausea, and vomiting. No evidence of liver injury was found in these individuals. Only minor erythema is observed from skin contact. Workers (mostly men) who have been engaged in the manufacture,

handling, and spraying of aldrin have been exposed to considerably higher concentrations and quantities of these insecticides than the general population of the United States. Among the exposed groups, only acute effects such as eye, skin, or respiratory irritation were reported, particularly following exposures to dusty formulations of the compound (HSDB, 2004). For dieldrin, epidemiological studies reported were mainly done on occupationally exposed individuals. These studies support the finding that the central nervous system is the main target organ resulting in convulsions (ATSDR, 2002c).

I. Toxicity

USEPA has classified aldrin and dieldrin as a probable human carcinogen (Group B2) due to sufficient evidence of carcinogenicity in animals. The chronic cancer slope factors for aldrin are 17 (mg/kg-day)-1 for both the oral and inhalation routes of exposure. The chronic cancer slope factors for dieldrin are 16 (mg/kg-day)-1 for both the oral and inhalation routes of exposure. The chronic oral reference doses are 3.0E-05 and 5.0E-05 mg/kg-day for aldrin and dieldrin, respectively. The toxicity values were taken from USEPA, 2004a.

3.3 POLYCHLORINATED BIPHENYLS (PCBs)

1. Constituent Properties

A. Physical and Chemical Properties

Aroclor-1254	
Atomic Weight (g/mol):	328
Melting Point:	no data
Boiling Point:	365-390°C
Specific Density:	1.54 (@ 25°C)
Water Solubility (mg/L):	0.012 (@ 20°C)
Vapor Pressure (mm Hg):	7.71×10^{-5} (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	2.0×10^{-3} (@ 25°C)
Aroclor-1260	
Atomic Weight (g/mol):	357.7
Melting Point:	no data
Boiling Point:	385-420°C
Specific Density:	1.62 (@ 20°C)
Water Solubility (mg/L):	0.0027
Vapor Pressure (mm Hg):	4.05×10^{-5} (@ 25°C)
Henry's Law Constant (atm-m^3/mol):	4.6×10^{-3} (@ 25°C)

Reference: ATSDR, 2000c. Toxicological Profile for Selected PCBs.

B. Chemical Transformation

Air: Vapor-phase reaction of PCBs with hydroxyl radicals may be the dominant transformation process (ATSDR, 2000c).

Water: Photolysis appears to be the only viable chemical degradation process in water (ATSDR, 2000c).

Soil: Degradation of PCBs by microorganisms occurs through oxidation and cleavage of the biphenyl ring that ultimately results in the formation of chlorinated benzoic acids (ATSDR, 2000c).

2. Toxicological Properties

A. Metabolism

PCBs are well aborbed after exposure by all routes and are distributed to and retained in adipose tissues. PCBs are metabolized by microsomal cytochrome P-450 to polar metabolites that can undergo conjugation with glutathione and/or glucuronic acid. The rate of metabolism of some PCB congeners depends on the degree of ring chlorination, the chlorine ring subsitution pattern or the pattern and levels of P-450 isozymes and other enzymes in the target tissue. Analysis of adipose tissue samples of volunteers revealed almost 60 individual PCB components. Examination of these results showed that <12 congeners accounted for ≈ 80% of the total PCBs. PCB congeners of low chlorine content are transformed into hydroxylated derivatives that are predominantly eliminated in urine. Highly chlorinated congeners with nonsusceptible subsitution patterns are either retained or excreted in the feces (ATSDR, 2000c).

B. Acute Toxicity

Nonspecific symptoms such as loss of appetite, nausea, epigastric distress and pain, and intolerance to fatty foods have been experienced by workers exposed to PCBs. Dietary exposure to generally low doses of PCBs for durations as short as several months produced characteristic gastritis in monkeys, which progressed to cysts, ulcers, and hemorrhages (ATSDR, 2000c).

Histologically documented liver damage is a consistent and prominent finding among PCB-exposed animals. However, studies of Aroclor-exposed workers only provide suggestive evidence for subclinical increases in serum enzymes that are indicators of possible liver microsomal enzyme induction or possible hepatocellular damage (e.g., GGPT, SGOT). Asymptomatic hepatomegaly, but not hepatic dysfunction, has been observed in PCB-exposed workers (ATSDR, 2000c).

Renal effects of PCBs in humans have not been reported. One acute-duration and several intermediate-duration oral studies have shown treatment-related mild to moderate kidney damage, particularly to the tubular epithelium, in rats at high doses of Aroclor-1242 (single

4,000 mg/kg gavage dose) and Aroclor-1254 (>10 mg/kg/day for 5 to 15 weeks). Based on the effects observed in animals treated with high doses of PCBs, the kidney may also be a target of PCB toxicity in humans (ATSDR, 2000c).

C. Subacute and Chronic Toxicity

Effects such as upper respiratory tract irritation, tightness of the chest, and possible impaired lung function have been reported in people occupationally exposed to various Aroclors for >5 years at mean concentrations of 0.007 to 11 mg/m^3. Other occupational studies reported more frequent or severe respiratory infections and chronic bronchitis (ATSDR, 2000c).

Several human and animal studies suggest PCBs can induce thyroid tocixity as well as a variety of changes in thyroid hormone levels. In one study the risk for goiter was significantly increased indicating the possibility of excess thyroid disease in an adult population. Other more limited observations in adults include reports of increased thyroid gland volume. Studies that examined PCB exposure and thyroid hormone status in children and adults reported findings of both positive and negative correlations between PCB exposure and circulating levels of thyroid stimulating hormone, thyroxine or tiriodothyronine hormones (ATSDR, 2000c).

Dermal lesions including skin irritation, chloracne, and pigmentation of nails and skin have been observed in humans following occupational exposure to relatively low levels of PCBs (0.1 mg/m^3 for average duration of 14.3 months) (ATSDR, 2000c).

D. Carcinogenicity

Existing epidemiological data do not indicate a consistent tumorigenic effect among people exposed to PCBs. Occupational studies suggest possible PCB-related carcinogenicity of the liver, gastrointestinal tract, hematopoietic system, and skin. Occupational exposure largely involves the inhalation route, although dermal contact is likely to significantly contribute to overall exposure. Oral carcinogenicity studies with rats and mice indicate that Aroclor-1254 is a hepatocarcinogen. Additionally, there is suggestive evidence that Aroclor-1254 induced gastric adenocarcinomas in rats. The highly chlorinated PCB mixtures appear to be more potent than the lower chlorinated PCB mixtures as indicated by higher incidences of malignant tumors (hepatocellular carcinoma) (ATSDR, 2000c). USEPA has classified PCBs as a probable human carcinogen (Group B2) with sufficient evidence in animal studies and inadequate evidence in humans.

E. Mutagenicity

There is limited information on the mutagenicity of PCBs. Animals orally and dermally exposed to PCBs reported no mutagenic activity in the studies (ATSDR, 2000c).

F. Teratogenicity/Reproductive Effects

Results from two studies in the United States in which exposure to PCBs was assumed to have been by consumption of contaminated fish suggest that exposure to PCBs causes developmental effects in humans. Lower birth weight was reported. Neurodevelopmental effects were manifested as motor deficits at birth, impaired psychomotor index during the first year of life, impaired visual recognition memory at 7 months of age, and deficits in short-term memory at 4 years of age. PCBs are associated with menstrual disturbances in women and effects on male fertility. Increased PCB levels have also been observed in women with late miscarriages. Reduction in the months of lactation was associated with increasing levels of PCBs in maternal breast milk. The reproductive observations in humans were also noted in animal studies, indicating that PCBs may be reproductive toxicants in humans (ATSDR, 2000c).

G. Other Health Effects

Occupationally-exposed workers reported subjective symptoms, such as headache, dizziness, depression, and fatigue indicating some neurotoxicity. Clinical observations suggests gastrointestinal damage in workers exposed to airborne PCBs. Reported gastrointestinal symptoms include anorexia, nausea, vomiting, abdominal pain, diarrhea and increase in loss of appetite (ATSDR, 2000c).

H. Epidemiological Evidence

There are several studies done on the human population that support the above information.

I. Toxicity Data

USEPA has classified aroclor-1254 and aroclor-1260 as B2, probable human carcinogen based on sufficient evidence in animal studies. The chronic cancer slope factors for PCBs are 2.0 (mg/kg-day)-1 for both the oral and inhalation routes of exposure. The chronic oral reference dose is 2.0E-05 mg/kg-day for aroclor-1254. All of the aforementioned toxicity values were taken from USEPA, 2004a.

4.0 METALS

4.1 ALUMINUM

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	26.98
Melting Point:	660°C
Boiling Point:	2,327°C
Specific Density:	2.701 (@ 25°C)
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	1 (@ 1284°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 1999b. Toxicological Profile for Aluminum.

B. Chemical Transformation

Air: Aluminum compounds cannot be oxidized and atmospheric transformation is not expected to occur. Atmospheric aluminum is removed by wet and dry deposition (ATSDR, 1999b).

Water: The hydrated aluminum ion undergoes hydrolysis resulting in the formation of hydroxyaluminum species (ATSDR, 1999b).

Soil: Weathering of soils results in rapid release of silicon and aluminum precipitates such as hydrated aluminum oxides i.e., gibbsite and boehmite (ATSDR, 1999b).

2. Toxicological Properties

A. Metabolism

Aluminum occurs normally in the body tissues of humans. About one half of the body burden is in the skeleton and about one quarter is in the lungs. Since aluminum is an element, it is not metabolized like other more complex chemicals. It is always found attached to other chemicals. In living organisms, aluminum is believed to exist in four different forms: as free ions, as low molecular weight complexes, as reversible macromolecular complexes, and as irreversible macromolecular complexes. Al3+ is easily bound to many substances in the organism and therefore its fate is determined by its affinity for these ligands, their amounts and their metabolisms. The low molecular weight complexes are often chelates and may be very stable. The nucleus and chromatin are often sites of aluminum binding in cells. Most aluminum leaves the body quickly in the feces (ATSDR, 1999b).

B. Acute Toxicity

Aluminum is not considered to be life-threatening to humans. Studies of individuals receiving high doses of oral aluminum in antacids have not reported any deaths (ATSDR, 1999b).

Acute oral exposure to high doses of aluminum has been reported to cause gastrointestinal effects including vomiting and abdominal cramps. Oral exposure to aluminum phosphide was reported to cause renal failure, significant proteinuria and anuria in humans who ingested it accidentally or in suicide attempts (ATSDR, 1999b).

Skin rashes were commonly found in individuals who drank water containing aluminum sulfate. Aluminum is widely used in antiperspirants without harmful effects to the skin or other organs, however, some individuals are sensitive to some antiperspirants and develop skin rashes, which may be due to aluminum (ATSDR, 1999b).

C. Subacute and Chronic Toxicity

Workers exposed to high levels of aluminum dusts have developed respiratory problems. There are reports of lung fibrosis and pneumothorax in workers in aluminum smelting, in processes involving metallic aluminum dust, and in the manufacture of alumina abrasives. Asthma symptoms have been reported in workers exposed to aluminum fumes in soldering and welding (ATSDR, 1999b).

Recent data indicate that adverse effects can result from long-term use of aluminum-containing medications in healthy individuals. Several cases have reported skeletal changes in adults and children. The aluminum in the antacids binds with dietary phosphorous and impairs gastrointestional absorption of phosphorous. The observed osteomalacia and rickets is related to the decreased phosphate body burden. Osteomalacia is also well documented for dialyzed uremic patients exposed to aluminum via dialysis fluid or orally administered to control hyperphophatemia. In cases of uremic patients, bone aluminum levels are markedly increased (ATSDR, 1999b).

Uremic patients are at risk for aluminum related-dimentia. Prolonged dialysis treatment has produced neurotoxicity syndrome which has been referred to as "dialysis dementia". The onset of neurotoxicity is rapid and is characterized by confusion, muscle twitching, grand mal seizures, coma and death. Although neurotoxicity has not been observed in healthy individuals (normal renal function), several animal studies have reported neurotoxic effects of aluminum, ranging from neurobehavioral and neurodevelopmental alterations (ATSDR, 1999b).

D. Carcinogenicity

Evidence for or against an association between exposure to aluminum and cancer in humans has not been established. Available carcinogenic animal studies do not indicate that aluminum is carcinogenic. USEPA

has not given aluminum a weight of evidence classification as to carcinogenicity.

E. Mutagenicity

Data is available to indicate that aluminum may interact with neuronal DNA to alter gene expressions and protein formation. It is possible that this is a mechanism by which aluminum affects the brains of patients with Alzheimer's disease (ATSDR, 1999b).

F. Teratogenicity/Reproductive Effects

Limited data are available on the teratogenic and reproductive effects of aluminum. Studies in humans and animals reported that aluminum does not affect the reproductive system. Developmental toxicity of aluminum includes neurodevelopmental changes and skeletal effects. The effects most commonly found in exposed weanlings and young mice included increases in grip strength and landing foot splay as well as, decreased thermal sensitivity. Gestational exposure to aluminum induced skeletal variations such as delayed ossification in rats and mice (ATSDR, 1999b).

G. Other Health Effects

Several children and one adult who had previous injections of vaccines or allergens containing aluminum showed hypersensitivity, however, hypersensitivity to aluminum appears to be rare in humans (ATSDR, 1999b).

H. Epidemiological Evidence

Several epidemiology and case-control studies have reported an association between oral exposure to aluminum and an increased incidence of Alzheimer's disease (ATSDR, 1999b).

I. Toxicity Data

USEPA has not classified aluminum as to its carcinogenicity. The chronic reference doses are 1.0 and 0.001 mg/kg-day for the oral and inhalation routes, respectively. The aforementioned toxicity values were taken from USEPA, 2004b.

4.2 ANTIMONY

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	121.75
Melting Point:	630°C
Boiling Point:	1,750°C
Specific Density:	6.688 (@ 20°C)
Water Solubility (mg/L):	insoluble

Vapor Pressure (mm Hg): 1 (@ 886°C)
Henry's Law Constant (atm-m^3/mol): no data
Reference: ATSDR, 1992d. Toxicological Profile for Antimony.

B. Chemical Transformation

Air: Antimony is oxidized to antimony trioxide by reaction with atmospheric oxidants (ATSDR, 1992d).

Water: The fate of antimony in waters is difficult to determine since the dissolved state is important and generally, total antimony in water is low. Antimony compounds may undergo photochemical reactions, but this fate has been reported to be insignificant. Antimony may be reduced and methylated by microorganisms and become mobilized (ATSDR, 1992d).

Soil: Antimony is known to form coprecipitates with hydrous iron, manganese, and aluminum oxides in soil and sediment. Methylated antimony compounds may be formed in waterlogged soil (ATSDR, 1992d).

2. Toxicological Properties

A. Metabolism

Since antimony is a metal, it does not readily undergo metabolism. The metabolism of antimony consists of covalent interactions with sulfhydryl groups and phosphate, as well as numerous reversible binding interactions with endogenous ligands (e.g., proteins) (ATSDR, 1992d).

B. Acute Toxicity

The respiratory tract is a target in humans following inhalation exposure to antimony. Pneumoconiosis, impaired pulmonary function (airway obstruction, bronchospasm, and hyperinflation) and respiratory irritation (coughing and wheezing) have been observed in factory workers exposed to antimony. Also, the toxicity of inhaled antimony compounds may be greater for smaller particle sizes. Respiratory irritation was not noted in workers exposed to antimony trisulphide for 8 months to 2 years (ATSDR, 1992d).

The heart is another target organ in humans. Alterations in EKG readings and increased blood pressure (greater than 150/90) have been reported in workers exposed to 2.15 mg antimony/m^3 as antimony trisulphide for 8 months to 2 years (ATSDR, 1992d).

C. Subacute and Chronic Toxicity

Humans chronically exposed to airborne antimony reported the targets to be the respiratory tract, gastrointestinal tract and skin. Respiratory effects included, chronic bronchitis, chronic emphysema, inactive tuberculosis and pleural adhesions. It is likely that, with sufficiently high or prolonged exposures, serious lung diseases would occur in humans.

Gastrointestinal effects after repeated prolonged exposure included abdominal pain, diarrhea, vomiting and ulcers. The dermatitis associated with exposure to airborne antimony was seen most often during the summer months and in workers exposed to high temperatures. It is probably the result of antimony being dissolved in sweat and penetrating the sweat glands. There are no reports of human and animal health effects following dermal or oral exposure to antimony (ATSDR, 1992d).

D. Carcinogenicity

Animal studies show that there is no evidence of carcinogenicity following oral, dermal or inhalation exposure to antimony. USEPA has not evaluated antimony as to its carcinogenicity potential.

E. Mutagenicity

There are no *in vivo* genotoxicity studies, however, *in vitro* studies using human leukocytes were positive for chromosome breakage. Results were mixed for *in vitro* studies using mammalian cells and positive for DNA damage (ATSDR, 1992d).

F. Teratogenicity/Reproductive Effects

An increased incidence of spontaneous abortions, compared to a control group, were reported in women working at an antimony metallurgical plant. The women were exposed to a mixture of antimony trioxide, antimony pentasulphide, and metallic antimony. Women also reported disturbances in their menstrual cycles when exposed to the same antimony compounds (ATSDR, 1992d).

G. Other Health Effects

Ocular irritation and conjuctivitis has been reported in humans following exposure to airborne antimony (ATSDR, 1992d).

H. Epidemiological Evidence

No data available.

I. Toxicity Data

USEPA has not classified antimony regarding its carcinogenic potential. A chronic oral reference dose is reported to be 0.0004 mg/kg-day for antimony. The aforementioned toxicity value was taken from USEPA, 2004a.

4.3 ARSENIC

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol): 74.92

Melting Point:	817°C
Boiling Point:	613°C
Specific Density:	5.727 (@ 20°C)
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	1 (@ 373°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 2000d. Toxicological Profile for Arsenic.

B. Chemical Transformation

Air: When released to the atmosphere primarily as arsenic trioxide or as arsines, these arsenic compounds undergo oxidation to the pentavalent state. Arsenic in the atmosphere is typically a mixture of trivalent and pentavalent forms. Photolysis is not an important fate for arsenic compounds in the atmosphere (ATSDR, 2000d).

Water: Arsenic in aquatic environments undergo complex series of transformation, including oxidation-reduction reactions, ligand exchange, precipitation and biotransformation. Arsenate is the predominant form of arsenic in the aquatic environment. Aquatic microorganisms could reduce arsenate to arsenite and a variety of methylated arsenicals (ATSDR, 2000d).

Soil: The arsenic cycle in soils is complex, with many biotic and abiotic processes controlling its overall fate. Arsenicals applied to soils may be methylated by microorganisms to arsines which are lost through volatilization and organic forms may be mineralized to inorganic forms. Organoarsenical pesticides (i.e., MMA, DMA) are metabolized by bacteria to alkylarsines, arsenate and MMA (ATSDR, 2000d).

2. Toxicological Properties

A. Metabolism

Most studies on the toxicokinetics of inorganic arsenic have been performed in animals, but there are a number of studies in humans as well. These studies reveal the following main points:

- Both arsenate and arsenite are well absorbed by the oral and inhalation routes. Absorption by the dermal route has not been studied, but is probably quite low.
- Once absorbed, arsenites are partially oxidized to arsenates and arsenates are partially reduced to arsenites, yielding a mixture of As^{+3} and As^{+5} in the blood.
- The As^{+3} form undergoes enzymic methylation in the liver to form monomethyl arsenic acid (MMA) and dimethyl arsenic acid (DMA). The rate and relative proportion of methylation production varies among species.

- Most arsenic is excreted in the urine as a mixture of As(+3), As(+5), MMA and DMA. Smaller amounts are excreted in the feces.

Less information is available for the organic arsenicals. Both MMA and DMA are readily absorbed but are rapidly excreted in the urine and feces. MMA may be methylated to DMA, but neither MMA nor DMA are demethylated to yield inorganic arsenic (ATSDR, 2000d).

B. Acute Toxicity

Inhalation of inorganic arsenic dusts (mainly containing arsenic trioxide) is irritating to the nose, throat, and lungs, and can lead to hoarseness, bronchitis, and rhinitis. The effects of organic arsenicals on the respiratory tract have not been well studied. There are no data by any route from human studies, but acute respiratory distress and lung injury have been reported in mice that inhaled very high levels of DMA (ATSDR, 2000d).

Nausea, vomiting and diarrhea are very common symptoms in humans following oral exposure to inorganic arsenicals, both after acute high dose exposure and after repeated exposure to lower doses. Inhalation exposure of rats to high doses of DMA can cause diarrhea and oral exposure of rabbits to MMA can cause intestinal irritation and weakening of the intestinal wall (ATSDR, 2000d).

C. Subacute and Chronic Toxicity

Chronic functional impairment of respiration is not usually observed, even in workers exposed to high levels of arsenic trioxide in air. High oral doses of inorganic arsenic can lead to marked cardiac arrhythmias and altered cardiograms in humans. Chronic oral exposure to lower levels of inorganic arsenic can also result in serious damage to the vascular system. The most extreme manifestation of this is "Blackfoot disease", a progressive loss of circulation in the fingers and toes that ultimately leads to gangrene. Blackfoot disease may not likely occur in other areas besides Taiwan, but less severe signs of vascular injury (Raynaud's disease, cyanosis of fingers and toes) have been noted in several other populations exposed to inorganic arsenic, both by inhalation and by the oral route (ATSDR, 2000d).

Signs of peripheral and/or central neuropathy are common in humans exposed to inorganic arsenicals by the oral route, and have also been observed in some workers exposed by the inhalation route. No studies were located regarding neurological effects in humans after exposure to organic arsenicals but pigs given repeated oral doses of roxarsone developed muscle tremors, paralysis, and seizures along with a degeneration of myelinated axons in the spinal cord (ATSDR, 2000d).

Anemia is often observed in humans exposed to arsenic by the oral route. Organic arsenicals have low hemotoxicity. Oral exposure of humans to inorganic arsenicals often produce a swollen and tender liver, most often

observed after chronic exposure. The principal lesion is a portal tract fibrosis and cirrhosis that results in portal hypertension. No information is available on hepatotoxic and renal effects of organic arsenicals in humans, although some mild effects on liver weight and signs of tubular damage have been noted in rats given repeated oral doses of roxarsone and in rabbits given repeated oral doses of MMA (ATSDR, 2000d).

Exposure to inorganic arsenic can manifest itself as a triad of dermatological effects, including hyperkeratinization of the skin, formation of multiple hyperkeratinized corns or warts, and hyperpigmentation of the skin with interspersed spots of hypopigmentation. These effects have been noted in numerous studies of intermediate or chronic oral exposure to inorganic arsenic and similar effects have been noted in workers exposed to inorganic arsenic primarily by the inhalation route. Direct dermal contact with inorganic arsenicals may cause irritation and contact dermatitis. Little information is available on the dermal or ocular effects of organic arsenicals (ATSDR, 2000d).

D. Carcinogenicity

There is clear evidence from studies in humans that exposure to inorganic arsenic may increase the risk of cancer. In workers exposed by the inhalation route, the predominant carcinogenic effect is increased risk of lung cancer. Most cases are seen in workers with chronic exposures, although several studies suggest that even short (1 year) exposures may also increase risk. When exposure occurs by the oral route, the main carcinogenic effect is increased risk of skin cancer. Other studies have suggested ingestion of arsenic may also increase the risk to internal tumors, mainly liver, kidney, bladder, and lungs (ATSDR, 2000d). Arsenic is classified by USEPA as a known human carcinogen (Group A) with sufficient evidence of carcinogenicity in humans.

E. Mutagenicity

Inorganic arsenicals appear to be inactive or weak mutagens but are able to produce chromosomal effects (aberrations, sister chromatid exchange) in most systems. Several tests indicate that DMA and roxarsome, both organic arsenicals, may be able to cause mutations and DNA strand breaks (ATSDR, 2000d).

F. Teratogenicity/Reproductive Effects

Inorganic arsenic has been characterized as a developmental toxicant. Animal studies reported that various fetal malformations (reduced fetal weight, fetal mortality and both skeletal and soft tissue defects) occur, with neural tube defects the predominant and consistent malformation reported in these studies. Animal studies for organic arsenics reported minor developmental effects (i.e., stunted growth, malformations and

mortality) compared to inorganic arsenics suggesting that organic arsenics are less fetotoxic than inorganic arsenicals (ATSDR, 2000d).

There is very limited information regarding reproductive toxicity of arsenics. Only one human study where after maternal ingestion of arsenic, a 30-week gestation live infant died 11 days later. In a three generation study in mice, it detected no significant effects other than decrease pups per litter. Another study where male mice orally exposed to MMA, reported a decrease in the number of females producing litters suggesting interference with sperm production (ATSDR, 2000d).

G. Other Health Effects

Workers exposed to arsenic dusts had reported conjuctivitis (known as pink eye), characterized by redness, swelling and pain, usually in combination with facial dermatitis. In several arsenic poisoning cases, facial edema was reported. The edema developed soon after initial exposure but then subsided (ATSDR, 2000d).

H. Epidemiological Evidence

There have been several epidemiological studies of humans to support the information provided.

I. Toxicity Data

USEPA has classified arsenic as a human carcinogen with sufficient evidence in human epidemiological studies. The chronic cancer slope factors are 1.5 (mg/kg-day)-1 and 15 (mg/kg-day)-1 for the oral and inhalation routes, respectively. The chronic oral reference dose is 0.0003 mg/kg-day. All of the aforementioned toxicity values were taken from USEPA, 2004a.

4.4 BARIUM

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	137.3
Melting Point:	710°C
Boiling Point:	1,600°C
Specific Density:	3.5 (@ 20°C)
Water Solubility (mg/L):	decomposes
Vapor Pressure (mm Hg):	10 (@ 1,049°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 1992e. Toxicological Profile for Barium.

B. Chemical Transformation

Air: In the atmosphere, barium is mostly likely present as particulates and is removed from the atmosphere by dry and wet deposition. Elemental barium is oxidized readily in moist air (ATSDR, 1992e).

Water: In aquatic media, barium is likely to precipitate out of solution as barium salt. It may also adsorb to particulate matter. Under natural conditions barium will form compounds in the 2+ oxidation state. Barium under highly alkaline conditions will hydrolyze (ATSDR, 1992e).

Soil: Barium reacts with metal oxides and hydroxides in soils and is subsequently adsorbed onto particulates (ATSDR, 1992e).

2. Toxicological Properties

A. Metabolism

Since barium is an element, it is not metabolized in the body, but it may be metabolically transported or incorporated into complexes or tissues (ATSDR, 1992e).

B. Acute Toxicity

Benign pneumoconiosis, also called baritosis has been observed in workers exposed occupationally by inhalation to barium. There are also case reports of individuals who developed respiratory weakness and paralysis following acute ingestion of barium salts. These results suggest that humans who are exposed orally or by inhalation to barium may be at increased risk for minor respiratory effects (ATSDR, 1992e; USEPA, 1999).

Case reports of humans suggest that gastrointestinal hemorrhage and gastrointestinal disturbances, including gastric pain, vomiting, and diarrhea, have been associated with acute oral exposure to barium (ATSDR, 1992e).

C. Subacute and Chronic Toxicity

Previous intermediate and chronic oral studies with experimental animals indicated that barium may induce hypertension and increased blood pressure (ATSDR, 1992e). The identification of hypertension as a health endpoint of concern is supported by findings of hypertensive effects in humans who ingested acutely high doses of barium compounds, in workers who inhaled dusts of barium ores and barium carbonate, in experimental animals given barium intravenously, and in rats exposed to barium in drinking water while on restricted diets. Based on these past findings, lower dose human studies were conducted to examine the potential effects on blood pressure in humans, and both blood pressure and kidney function in animals. Although the studies did not report any significant effects on blood pressure, they establish a NOAEL in humans of 0.21 mg Ba/kg-day. The animal data suggest that

the kidney may be a sensitive target for ingested barium from low level exposure. Mortality and nephropathy were reported in high dosed animals at 2,500 to 4,000 ppm. The nephropathy was characterized as tubule dilatation, renal tubule atrophy, tubule cell regeneration, and the presence of crystals primarily in the lumen of the renal tubules. Other adverse effects observed in the high-dose animals were attributed to debilitation of the animals (USEPA, 1999).

D. Carcinogenicity

USEPA has classified barium as Group D, not classifiable as to human carcinogenicity. This is based on oral exposure studies in rats and mice which did not find significant increases in tumor incidence following chronic exposure. In another rat study, statistically significant negative trends in the incidence of leukemia, adrenal tumors, and mammary gland tumors were observed. The design of the rat and mouse studies were determined to be adequate to assess carcinogenicity (USEPA, 1999).

E. Mutagenicity

There is a limited amount of information available on the genotoxicity of barium compounds. No *in vivo* studies have been conducted. Most *in vitro* studies have found that barium chloride and barium nitrate did not induce gene mutations in bacterial assays with or without metabolic activation (USEPA, 1999).

F. Teratogenicity/Reproductive Effects

Rats were reported to have disturbances in spermatogenesis, shortened estrous cycle, and alterations of the testes and ovaries. In the same study, offsprings were reported to be underdeveloped, lowered body weight, blood disorders, increased leukocyte count, disturbances to the liver, decreased lability of the nervous system and increased mortality (ATSDR, 1992e; USEPA, 1999).

G. Other Health Effects

Case studies of humans having acute oral or inhalation exposure to barium have reported such effects as deep tendon reflexes, numbness and tingling of the mouth and neck, partial and complete paralysis, brain congestion and edema (ATSDR, 1992e).

H. Epidemiological Evidence

The available epidemiological studies suggest that barium has no effect on blood pressure (ATSDR, 1992e; USEPA, 1999).

I. Toxicity Data

USEPA has classified barium as not classifiable as to human carcinogenicity based on inadequate or no evidence in studies. The chronic oral RfD for barium is 0.07 mg/kg-day (USEPA, 2004a) and the chronic inhalation RfD for barium is 1.4E-04 mg/kg-day (USEPA, 1997).

4.5 BERYLLIUM

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	9.012
Melting Point:	1,292°C
Boiling Point:	2,970°C
Specific Density:	1.846 (@ 20°C)
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	1 (@ 1,520°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 2002d. Toxicological Profile for Beryllium.

B. Chemical Transformation

In the environment, chemical reactions can change the water-soluble beryllium into insoluble forms and vice-versa (ATSDR, 2002d).

Air: In the air, beryllium compounds exist primarily in particulate form. The predominant fate of airborne beryllium is by dry or wet deposition (ATSDR, 2002d).

Water: Most of the beryllium in the water exists as insoluble beryllium where it will settle to the bottom with sediment (ATSDR, 2002d).

Soil: In soils, beryllium will typically not dissolve and therefore, will remained bound to soil particulates (ATSDR, 2002d).

2. Toxicological Properties

A. Metabolism

Beryllium is mainly absorbed after inhalation exposure. Absorption after oral and dermal exposures is reported to be poor. Beryllium and its compounds are not biotransformed, but soluble beryllium salts are partially converted to less soluble forms in the lung. Beryllium clearance from the lungs is biphasic. Approximately 30% is cleared during the first phase, reporting half-life of 2.5 days. The second phase is slower with a half-life reported at 833 days. Absorbed beryllium is distributed in the body by the blood stream, with highest concentrations reported in the liver and skeleton. Elimination of beryllium is via feces and urine (ATSDR, 2002d).

B. Acute Toxicity

The respiratory tract in humans and animals is the primary target organ following beryllium inhalation exposure. Occupational exposure to high concentrations of beryllium resulted in acute beryllium disease (ACD). ACD is characterized by inflammation of the respiratory tract tissues,

nasopharyngitis, shortness of breath, labored breathing and chemical pneumonitis. ACD is usually resolved within a couple of months of exposure termination. Acute exposure to high concentration of beryllium may lead to death (ATSDR, 2002d).

Two types of dermal effects have been observed in workers after exposure to beryllium, an inflammatory reaction and an immune reaction. Workers exposed to airborne beryllium sulfate, beryllium fluoride or beryllium oxyfluoride reported inflammatory reactions to include rashes. Beryllium exposure may also cause delayed, hypersensitive reaction in the skin. This is characterized by skin granulomas which appear as a rash or nodules (ATSDR, 2002d).

C. Subacute and Chronic Toxicity

Chronic inhalation exposure to beryllium has been reported to result in chronic beryllium disease (CBD). CBD is an immune response to beryllium and can be classified into three stages: beryllium sensitization, subclinical CBD, and clinical CBD. Beryllium sensitization can progress to CBD but not all individuals develop CBD. Individuals with subclinical CBD are sensitized to beryllium and have histological evidence of lung granulomas. Individuals with clinical CBD have histological evidence of lung granulomas and respiratory symptoms, changes on chest radiographs and altered lung functions. Other systemic effects that have been observed in individuals with severe cases of CBD include damage to the right heart ventricle, hepatic necrosis, kidney stones, and weight loss. These systemic effects are considered secondary to CBD rather than a direct effect on the tissues (ATSDR, 2002d).

No human data were located regarding effects after chronic oral exposure to beryllium, however, in animal studies it was reported that the most sensitive effects appear to be ulcerative gastrointestinal lesions in dogs and beryllium rickets in rats (ATSDR, 2002d).

D. Carcinogenicity

Based on the limited evidence of carcinogenicity in humans exposed to airborne beryllium (lung cancer) and sufficient evidence of carcinogenicity in animals (lung cancer in rats and monkeys inhaling beryllium, lung tumors in rats exposed to beryllium via intratracheal instillation, and osteosarcomas in rabbits and possibly mice receiving intravenous or intramedullary injection), beryllium is reclassified from a B2 (inadequate human data) to a B1 probable human carcinogen (limited human data) (USEPA, 1998a).

E. Mutagenicity

Genotoxicity information regarding exposure to beryllium are contradictory. Both positive and negative results were reported for forward and reverse mutation bacterial assays. Also, the results for

chromosomal aberrations in mammalian cell cultures were contradictory (ATSDR, 2002d).

F. Teratogenicity/Reproductive Effects

The limited studies performed on rats reported no effects on the reproductive system. However, a study where pregnant rats were exposed to beryllium during gestation, delivered pups that died soon after birth. In another study there was increased fetal mortality, fetal weight and abnormalities reported (ATSDR, 2002d).

G. Other Health Effects

No data available.

H. Epidemiological Evidence

There are a few studies reported that support beryllium exposure with effects on the respiratory system.

I. Toxicity Data

USEPA (1998a) has classified beryllium as a probably human carcinogen with limited evidence in human epidemiological studies. The chronic inhalation cancer slope factor is 8.4 (mg/kg-day)-1. The chronic reference doses are 0.002 and 5.7E-06 mg/kg-day for the oral and inhalation routes, respectively. All of the aforementioned toxicity values were taken from USEPA, 2004a.

4.6 CADMIUM

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	112.40
Melting Point:	321°C
Boiling Point:	765°C
Specific Density:	8.65 (@ 25°C)
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	1 (@ 394°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 1999c. Toxicological Profile for Cadmium.

B. Chemical Transformation

Air: Cadmium typically is in particulate form in the atmosphere. Common cadmium compounds found in the atmosphere include oxide, sulfate and chloride. These compounds are stable and not subject to photochemical reactions. Cadmium sulfide may photolyze to cadmium sulfate in aqueous aerosols. The

predominant fate of airborne cadmium is by dry or wet deposition (ATSDR, 1999c).

Water: In fresh water, cadmium is present primarily as Cd(+2) ion, Cd(OH), and CdCO complexes. Water insoluble compounds may be changed to water-soluble salts by interaction with acids or light and oxygen. In reducing environments, cadmium precipitates as cadmium sulfide (ATSDR, 1999c).

Soil: Transformation processes for cadmium in soils are dictated by sorption from and desorption to water, and include precipitation, dissolution, complexation and ion exchange (ATSDR, 1999c).

2. Toxicological Properties

A. Metabolism

Cadmium is widely distributed in the body, with the major portion of the body burden located in the liver and kidney. Cadmium is not known to undergo any direct metabolic conversions such as oxidation, reduction, or alkylation. The cadmium (+2) ion does bind to anionic groups (especially sulfhydryl groups) in proteins (especially albumin and metallothionein). Cadmium is circulated bound to these two proteins. Cadmium is excreted from the body via feces and urine (ATSDR, 1999c).

B. Acute Toxicity

High levels of exposure to cadmium by the inhalation or oral routes can cause death in humans and animals. It has been estimated that exposure via inhalation to 1 mg/m^3 for 8 hours could cause some deaths among exposed humans. The doses ingested in two known fatal cases were estimated to be 25 and 1,840 mg/kg. The cause of death is pulmonary edema following inhalation exposure and massive fluid imbalance and widespread gastrointestinal, liver, and other organ damage following oral exposure (ATSDR, 1999c).

Acute inhalation exposure to cadmium at concentrations above 5 mg/m^3 may cause destruction of lung epithelial cells, resulting in pulmonary edema, tracheobronchitis, and pneumonitis in both humans and animals. Nonoccupational exposure to cadmium is unlikely to be high enough to cause respiratory effects (ATSDR, 1999c).

The gastrointestinal tract is the largest target organ for high level, acute oral exposure to cadmium in both humans and animals. The main symptoms following ingestion of cadmium at doses above about 0.07 mg/kg in humans are nausea, vomiting, and abdominal pain. Gastrointestinal toxicity is not observed in humans or animals after lower levels of oral or inhalation exposure to cadmium, indicating that gastrointestinal effects are not likely to occur from environmental exposures to cadmium (ATSDR, 1999c).

C. Subacute and Chronic Toxicity

Long-term inhalation exposure at lower levels can lead to decreased lung function and emphysema. Some tolerance to cadmium-induced lung irritation develops in exposed humans. Another long-term effect after inhalation exposure to cadmium is damage to the olfactory function. Lung damage has also been seen following intermediate duration oral cadmium exposure in rats but the lung effects are likely to be related to liver or kidney damage and subsequent changes in metabolism (ATSDR, 1999c).

Both oral and inhalation exposure to cadmium can cause anemia in humans and animals. Prolonged exposure of humans to cadmium at levels causing renal dysfunction can lead to painful and debilitating bone disease after inhalation or oral exposure. Human and animal studies suggest that lower level chronic exposure to cadmium causes alterations in renal metabolism of vitamin D, which then may cause milder bone effects (osteoporosis). Cadmium accumulates in the liver following inhalation or oral exposure in humans, but there is little evidence for liver damage in humans exposed to cadmium (ATSDR, 1999c).

The kidney is the main target organ for cadmium toxicity following intermediate- or chronic-duration exposure by the inhalation or oral routes. Kidney damage, progressing from mild tubular lesions to necrosis has been reported in animal studies after administration of cadmium or cadmium salts (ATSDR, 1999c).

D. Carcinogenicity

The evidence that cadmium inhalation can cause lung cancer in humans is rather weak, but strong evidence exists that cadmium inhalation can cause lung cancer in rats. No studies were located providing evidence that humans or animals orally exposed to cadmium had increased incidences of cancer (ATSDR, 1999c). USEPA classifies cadmium as a probable human carcinogen (Group B1) with limited evidence of carcinogenicity in humans (USEPA, 1994b).

E. Mutagenicity

Studies of chromosomal aberrations in humans exposed to cadmium reported both positive and negative results. Animal studies, parenteral not oral exposure reported germ cell mutations (ATSDR, 1999c).

F. Teratogenicity/Reproductive Effects

Cadmium has been shown to be a developmental toxin by the inhalation, oral, and parenteral routes in animals. The most sensitive indicator of developmental toxicity appears to be impaired neurological development, end points such as locomotor activity and avoidance behavior decreased. It has also been reported that cadmium effects fetal development. Malformations of the skeleton such as fused lower limbs,

absence of limbs, delayed ossification of the sternum and ribs have been reported in animal studies (ATSDR, 1999c).

There is limited evidence that cadmium is a reproductive toxicant. Adverse effects have been reported in rats after both oral and inhalation exposures. The effects reported include testicular atrophy, necrosis, decreased fertility, decreased sperm count and motility (ATSDR, 1999c).

G. Other Health Effects

In examining effects of cadmium on the cardiovascular system, conflicting evidence is seen. In some studies on rats and rabbits, cadmium exposure was shown to increase blood pressure or to cause cardiac lesions. However, studies of exposed humans demonstrate results that are positive, negative, and show no association between cadmium exposure and hypertension. This suggests that if cadmium does affect blood pressure, the magnitude of the effect is small compared to other determinants of hypertension (ATSDR, 1999c).

H. Epidemiological Evidence

Several studies for renal toxicity of cadmium have been conducted on workers by inhalation and oral exposures. These studies confirmed kidney damage.

I. Toxicity Data

USEPA (1994b) has classified cadmium as a probably human carcinogen with limited evidence in human epidemiological studies. The chronic inhalation cancer slope factor is 6.3 (mg/kg-day)-1 (USEPA, 2004a). The chronic reference doses are 0.001 mg/kg-day (USEPA, 2004a) and 5.7E-05 mg/kg-day (USEPA, 2004b) for the oral and inhalation routes, respectively.

4.7 CHROMIUM

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	51.996
Boiling Point:	2,672°C
Melting Point:	1,857°C
Specific Density:	7.20 (@ 28°C)
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	1 (@ 1616°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 2000e. Toxicological Profile for Chromium.

B. Chemical Transformation

Air: Chromium is present in the atmosphere predominantly in particulate form and is subjected to dry and wet deposition. Typically chromium will remain in air for less than 10 days. Vanadium will reduce chromium (VI) to chromium (III). In the presence of manganese oxide, chromium (III) as a salt may be oxidized to chromium (VI) (ATSDR, 2000e).

Water: A small percentage of chromium can be present in both soluble and insoluble forms. Soluble chromium is present as chromium (VI) where chromium (III) is present as particulates. Particulate forms of chromium will deposit to the sediment in water columns. Chromium (VI) will be reduced to chromium (III) by organic matter in the water (ATSDR, 2000e).

Soil: Most of the chromium in soil doesn't dissolve and strongly adsorbs to particulates. A small percentage of chromium in soil may dissolve in water which may leach to groundwater. In the presence of low oxidizable organic substances such as oxygen, manganese dioxide, and moisture; chromium (III) is oxidized to chromium (VI). Under anaerobic conditions, chromium (VI) may be transformed to chromium (III) by sulfide and iron (ATSDR, 2000e).

2. Toxicological Properties

A. Metabolism

Chromium (III) compounds are essential to normal glucose, protein, and fat metabolism in mammals. In addition, chromium (III) is capable of forming complexes with nucleic acids and proteins. Chromium (VI) does not combine with nucleic acids and proteins unless it is first converted to chromium (III). In the lungs, chromium (VI) can be reduced to chromium (III) by ascorbate (ATSDR, 2000e).

B. Acute Toxicity

In general, chromium (VI) compounds are more toxic than chromium (III) compounds. Human death has resulted from accidental or intentional ingestion or dermal exposure to chromium (VI) compounds. One case involved a death of a man after he was immersed in a vat of a solution containing chromium (III) sulfate.

The respiratory tract is the major target following inhalation exposure to chromium (III) and chromium (VI) compounds in humans and animals. Human exposure to either chromium (III) or chromium (VI) compounds has resulted in perforations and ulceration of the nasal septum, bronchitis, pneumoconiosis, decreased pulmonary function, pneumonia, rhinorrhea, nasal itching and soreness, and epistaxis. Nasal irritation and atrophy and decreases in pulmonary function can occur at occupational exposure levels as low as 0.002 mg chromium (VI)/m^3. The effects of

chromium (III) and chromium (VI) on the respiratory system have been observed in animals (ATSDR, 2000e).

Workers in chromate plant exposed to high levels of atmospheric chromium (III) and chromium (VI) have developed gastric ulcers and gastritis as a result of swallowing chromium dust during mouth breathing. Inhaled chromium dust is also carried to the pharynx and swallowed. Stomach pain, duodenal ulcer, gastritis, and stomach cramps have also been reported in other studies of workers engaged in chromate production and electroplating but not in workers engaged in chromium (III) production. Abdominal pain, vomiting, and gastrointestinal hemorrhage have occurred in humans who eventually died after ingesting chromium (VI) as various chromium compounds. It is reasonable that gastrointestinal irritation can occur in humans exposed to chromium (VI) compounds at hazardous waste sites by inhalation, ingestion or dermal exposure (ATSDR, 2000e).

Chromium compounds produce effects on the skin and mucous membranes. These include irritation, burns, ulcers, and an allergic type of dermatitis. Acute dermal exposure to chromium (VI) compounds can cause skin burns. Although skin contact with chromate salts may cause rashes, the ulcers or sores (also called chrome holes) on the skin are a major problem because they can deeply penetrate the skin with prolonged exposure. In addition, irritation and ulceration of mouth structures and buccal mucosa can occur from exposure to chromium (VI) compounds. High incidences of inflammation of oral structures, keratosis of the lips, gingiva, and palate, gingivitis, and peridontis were observed in chromate production workers. Ocular effects can occur as a result of direct contact of eyes with chromium compounds. These include corneal vesication in a man who got a drop or a crystal of potassium dichromate in his eye and congestion of the conjunctiva, discharge, corneal scar, and burns in chromate production workers as a result of accidental splashes. It is possible that dermal effects could occur in humans exposed to chromium (VI) compounds at hazardous waste sites, particularly from dermal contact with contaminated soil (ATSDR, 2000e).

C. Subacute and Chronic Toxicity

The respiratory system and the skin are the primary target organs for occupational exposure to chromium and its compounds, as noted above. Epidemiological studies where populations residing in areas contaminated with chromium (VI) reported effects of oral ulcer, diarrhea, abdominal pain, indigestion, vomiting, constipation, nose and eye irritation, headache, fatigue, dizziness, and leukocytosis. (ATSDR, 2000e).

D. Carcinogenicity

The overall weight of evidence for carcinogenicity to humans outlined in humans, chromium (III) is most appropriately designated a Group D, not

classified as to its human carcinogenicity. Using the Proposed Guidelines for Carcinogen Risk Assessment (USEPA, 1996), USEPA has determined that there are inadequate data to determine the potential carcinogenicity of chromium (III) (USEPA, 1998b).

Under the current guidelines, USEPA has classified chromium (VI) as Group A, known human carcinogen by the inhalation route of exposure. Carcinogenicity by the oral route of exposure cannot be determined and is classified as Group D, not classified as to its human carcinogenicity. Under the guidelines, chromium (VI) would be characterized as a known human carcinogen by the inhalation route of exposure (USEPA, 1998c).

E. Mutagenicity

Several studies reported positive results for chromosomal aberrations in occupationally exposed individuals to chromium (ATSDR, 2000e).

F. Teratogenicity/Reproductive Effects

The only information regarding developmental effects of chromium exposure in humans is that female employees at dichromate manufacturing factories in Russia had greater incidences of complications during pregnancy and childbirth, toxicosis during pregnancy, and post natal hemorrhage than did controls. In animals, oral exposure to chromium (VI) appears to adversely affect the males and females reproductive system. Although there were no human studies located regarding developmental toxicity, there were several animal studies that reported developmental toxicity from chromium. Chromium (VI) compounds caused severe developmental effects in rats and mice after oral exposure. Effects reported in these animal studies included fetal mortality, decreased growth, reduced ossification, subdermal hemorrhagic patches and impaired development of the reproductive system (ATSDR, 2000e).

G. Other Health Effects

Liver effects, such as jaundice, increased bilirubin, increased levels of serum lactic dehydrogenate, and necrosis have been observed in humans after ingestion of lethal doses of potassium dichromate of chromium trioxide. Severe renal impairment, renal failure, and necrosis of renal tubules have been reported in cases of fatal or near fatal ingestion of chromium (VI) compounds by humans. Information regarding neurological effects after exposure to chromium or its compounds is limited. Dizziness, headache, and weakness were experienced by workers in a chrome plating plant were poor exhaust resulted in excessively high concentrations of chromium trioxide (ATSDR, 2000e).

H. Epidemiological Evidence

Epidemiology studies clearly indicate an increased respiratory cancer risk in chromate production workers. Increased risks of respiratory cancer are also reported in some studies of chrome pigment workers, chrome

plating workers, and ferrochromium workers. The epidemiology studies do not clearly implicate specific compounds, but do implicate chromium (VI) compounds. Chromium (VI) compounds are considered to be carcinogenic for humans (ATSDR, 2000e).

I. Toxicity Data

USEPA has determined that there are inadequate data to determine the potential carcinogenicity of chromium (III). A chronic oral reference dose is reported to be 1.5 mg/kg-day for chromium. The aforementioned toxicity value was taken from USEPA, 2004a.

USEPA has classified chromium (VI) as a known human carcinogen by the inhalation route of exposure. The chronic inhalation cancer slope factor is 42 (mg/kg-day)-1. The chronic oral reference dose is 0.003 mg/kg-day. The chronic inhalation reference doses are 2.3E-06 and 3.0E-05 mg/kg-day for chromium (VI) aerosols and particulates, respectively. The aforementioned toxicity values were taken from USEPA, 2004a.

4.8 COBALT

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	58.93
Melting Point:	1,495°C
Boiling Point:	2,870°C
Specific Density:	8.90 (@ 20)
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	1 (@ 1910°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 2004b. Toxicological Profile for Cobalt.

B. Chemical Transformation

Air: Cobalt released to the atmosphere is deposited onto soil or water surface by wet and dry deposition (ATSDR, 2004b).

Water: In water, cobalt largely partitions to sediment and suspended solids in the water column; however, the amount that is adsorbed to solids is highly variable (ATSDR, 2004b).

Soil: In general, cobalt has low mobility and strong adsorption in soils. Its mobility increases in moist, acidic soils (ATSDR, 2004b).

2. Toxicological Properties

A. Metabolism

Once cobalt enters the body, it goes into all tissues mainly into the liver, kidney and bones. The rest is absorbed into the blood and then into the tissues of the body. This metal is not metabolized; however, cobalt is essential in the body in that it is a component of Vitamin B12 which acts as a coenzyme in many enzymatic reactions. Unabsorbed cobalt leaves the body quickly in feces and absorbed cobalt is excreted from the body slowly, mainly in the urine (ATSDR, 2004b).

B. Acute Toxicity

Effects in humans following acute inhalation, oral and dermal exposures to cobalt have been reported. Occupational exposure of humans to cobalt have reported primarily respiratory effects, including decreased pulmonary function, asthma, interstitial lung disease, wheezing, and dyspnea. Animal studies have further identified respiratory tract hyperplasia, pulmonary fibrosis, and emphysema as sensitive effects of inhaled cobalt. Humans in the workplace have been shown to develop sensitivity to cobalt following inhalation exposures. Exposure to inhaled cobalt aerosols resulted in asthmatic attacks in sensitized individuals. This has been reported to be an allergic reaction within the lungs (ATSDR, 2004b).

The most sensitive endpoint following oral exposure appears to be an increase in erythrocyte (polycythemia), hematocrit, and hemoglobin levels in both humans and animals. Following dermal exposure, the most commonly observed effect is dermatitis. Using patch test studies, it has been demonstrated that the dermatitis is most likely caused by an allergic reaction to cobalt with cobalt functioning as a hapten (ATSDR, 2004b).

C. Subacute and Chronic Toxicity

Lethal cardiomyopathy in humans was reported following repeated inhalation of airborne cobalt or ingestion of beer that contained cobalt. Occupational exposure to airborne cobalt is characterized by functional effects on the ventricles and enlargement of the heart, resulting in cardiomyopathy. Exposure of humans to beer containing cobalt as a foam stabilizer resulted in severe effects of the cardiovascular system, including cardiomyopathy and death. As well, gastrointestinal effects including nausea and vomiting and hepatic necrosis were reported (ATSDR, 2004b).

D. Carcinogenicity

Available studies of the carcinogenic effects of cobalt in workers have reported both positive and negative results. Lifetime occupational inhalation studies of cobalt reported increases in lung cancer mortality. As well, animal studies have reported increase in alveolar/bronchiolar

neoplasms, with lung tumors occurring with significantly positive trends. USEPA does not report a cancer classification for cobalt (ATSDR, 2004b).

E. Mutagenicity

Animal studies have reported mutagenic action in bacterial cell lines and mammalian cell lines when the animals were orally exposed (ATSDR, 2004b).

F. Teratogenicity/Reproductive Effects

Pregnant women treated with cobalt for hematocrit and hemoglobin levels reported no observable effects to the fetuses. However, animal studies reported stunted fetuses, decrease in the number of litters and average litter weights, and increased mortality (ATSDR, 2004). Following inhalation and oral exposure of male rats to cobalt, adverse effects on the testes were observed (degeneration, atrophy, decreased weight). An increase in the length of the estrous cycle was also reported in female mice following inhalation exposure (ATSDR, 2004b).

G. Other Health Effects

Neurotoxic effects of cobalt in humans after inhalation exposure has been reported. Two studies have reported memory deficits, optic atrophy, and nerve deafness in humans exposed to cobalt (ATSDR, 2004b).

H. Epidemiological Evidence

Epidemiological studies relating to cobalt exposure support the information provided above. Studies of persons exposed to cobalt occupationally are available, dietetically (beer drinkers) and medically (cobalt given to alleviate anemia) (ATSDR, 2004b).

I. Toxicity Data

USEPA has not classified cobalt as to human carcinogenicity. The chronic inhalation cancer slope factor is 9.8 (mg/kg-day)-1. The chronic reference doses are 0.02 and 5.7E-06 mg/kg-day for the oral and inhalation routes, respectively. All of the aforementioned toxicity values were taken from USEPA, 2004b.

4.9 COPPER

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	63.55
Melting Point:	1,083°C
Boiling Point:	2,959°C
Specific Density:	8.94 (@ 20°C)
Water Solubility (mg/L):	insoluble

Vapor Pressure (mm Hg): 1 (@ 1628°C)
Henry's Law Constant (atm-m^3/mol): no data
Reference: ATSDR, 2002e. Toxicological Profile for Copper.

B. Chemical Transformation

Air: Copper released to the atmosphere is deposited onto soil or water surface by wet and dry deposition. Copper may react with oxidants to produce forms of oxides in the atmosphere. The aged oxides may undergo sulfatization in the presence of SOx gases (ATSDR, 2002e).

Water: Much of the copper discharged to water is in particulate form and settles out, precipitates out or adsorbs to organic matter, hydrous iron and manganese oxides, and clay. The Cu(I) ion is unstable in aqueous solution, tending to disproportionate to Cu(II) and copper metal unless a stabilizing ligand is present (ATSDR, 2002e).

Soil: Most copper released to soils will be strongly adsorbed to organic material and minerals in the top layers of soil and does not move very far from its released site. Transformation is not expected to occur (ATSDR, 2002e).

2. Toxicological Properties

A. Metabolism

Copper is readily absorbed from the stomach and small intestines. The metabolism of copper consists mainly of its transfer to and from various organic ligands, most notably sulfhydryls and imidazole groups on amino acids and proteins. Excretion of copper is predominantly through the fecal (biliary) route (ATSDR, 2002e).

B. Acute Toxicity

There are several reports of humans dying following ingestion of large amounts of copper, ranging from 6 to 637 mg/kg-day. The deaths were attributed to shock and hepatic and/or renal complications. Deaths due to central nervous system depression and hepatic and renal failure were also reported in individuals ingesting "spiritual green water" containing 100 mg copper sulfate/L. Deaths in animals given >250 mg copper/kg/day in the diet have also been attributed to extensive hepatic centrilobular necrosis (ATSDR, 2002e).

The most common reported adverse health effect of copper is gastrointestinal distress. Vomiting, nausea, and abdominal pain was reported in humans immediately after ingesting beverages contaminated with copper. Animal studies have also reported gastrointestinal effects (hyperplasia of forestomach mucosa) following ingestion of copper sulfate in the diet. Copper is also irritating to the respiratory tract. Coughing, sneezing, runny nose, pulmonary fibrosis, and increased

vascularity of the nasal mucosa have been reported in workers exposure to copper dust (ATSDR, 2002e).

C. Subacute and Chronic Toxicity

The liver is a sensitive target organ of chronic toxicity. In adults, liver damage including necrosis, fibrosis, and abnormal biomarkers of liver damage have been reported in individuals ingesting lethal doses of copper sulfate. In infants and children, reported liver effects are usually manifested in one of three syndromes: Wilson's disease, Indian childhood cirrhosis, or idiopathic copper toxicosis. These syndromes are genetic disorders that result in an accumulation of copper in the liver, usually from ingesting copper-contaminated milk or water with high levels of copper. The clinical age onset is usually between 6 months and 5 years. The observed effects include pericellular fibrosis, abnormal biochemical markers of liver damage, and very high levels of copper in the liver. Two studies have established no effect levels of 0.14 and 0.315 mg copper/kg/day in adults and infants, respectively (ATSDR, 2002e).

D. Carcinogenicity

USEPA has not classified copper as a human carcinogen (Group D) due to no human data, inadequate animal data from assays of copper compounds, and equivocal mutagenicity data (USEPA, 1991e).

E. Mutagenicity

The available genotoxicity data suggest that copper is a clastogenic agent. Mixed results have been found in point mutation assays. Several studies have also shown that exposure to copper can result in DNA damage (ATSDR, 2002e).

F. Teratogenicity/Reproductive Effects

Animal studies suggest that copper does not have any effects on the reproductive system. Developmental studies by the oral route in rats and mice reported effects of decreased embryo and fetal growth (ATSDR, 2002e).

G. Other Health Effects

Copper has been noted to have immunotoxic potential. Reports on individuals developing dermatitis after dermal exposure to copper suggest that copper is an allergen. This is supported by a report of a woman developing dermatitis after insertion of a copper IUD. Immunologic effects were reported in mice following acute inhalation exposure. Impaired immune function was observed in mice exposed to copper chloride or copper sulfate (ATSDR, 2002e).

H. Epidemiological Evidence

Most studies are based on the toxicity of inhaled copper to workers, reporting respiratory effects. There are several cases of accidental or

intentional ingestion of copper. The most common reported effect is gastrointestinal upset (ATSDR, 2002e).

I. Toxicity Data

USEPA has not classified copper as to human carcinogenicity, due to inadequate evidence of carcinogenicity in studies. A chronic oral reference dose is reported to be 0.04 mg/kg-day for copper. The aforementioned toxicity value was taken from USEPA, 1997.

4.10 CYANIDE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	27.03
Melting Point:	-13.24°C
Boiling Point:	25.7°C
Specific Density:	0.6884 (@ 20°C)
Water Solubility (mg/L):	0.17
Vapor Pressure (mm Hg):	264 (@ 0°C)
Henry's Law Constant (atm-m^3/mol):	5.1×10^{-2}

Reference: ATSDR, 2004c. Toxicological Profile for Cyanide.

B. Chemical Transformation

Air: Cyanide reacts with photochemically-produced hydroxyl radicals.

Water: Volatilization and biodegradation are the predominant fate processes in water.

Soil: In soil, cyanide present at low concentrations would biodegrade under aerobic conditions with the initial formation of ammonia, which will be converted to nitrate in the presence of nitrifying bacteria. Under anaerobic conditions, cyanides will denitrify to gaseous nitrogen exposure (ATSDR, 2004c).

2. Toxicological Properties

A. Metabolism

No studies were located regarding metabolism of cyanide in humans, but the metabolism of cyanide has been studied in animals. The proposed metabolic pathways are:

i) the major pathway, conversion to thiocyanate by either rhodanese or 3-mercaptopyruvate;

ii) conversion to 2-aminothiazoline-4-carboxylic acid;

iii) incorporation into a 1-carbon metabolic pool; or

iv) combining with hydroxocobalamin to form cyanocobalamin (vitamin B_{12}).

B. Acute Toxicity

Nausea and vomiting were reported in workers exposed to cyanide occupationally. The gastrointestinal effects can be caused by central nervous system stimulation (nausea) or by direct contact (necrosis) with cyanide salts. Convulsions are typical symptoms of cyanide poisoning following inhalation, oral, or dermal exposure. Respiratory effects commonly occur after cyanide poisoning by any route of exposure. Various symptoms indicating respiratory effects were reported in humans exposed to cyanide in occupational settings. Upper respiratory irritation, cough, altered sense of smell, nasal congestion, epitaxis, hemoptysis, and dyspnea were among the clinical signs of cyanide toxicity. Cardiovascular effects are common after exposure to cyanide. Palpitations and hypotension were the main effects reported in those exposed via inhalation, or oral or dermal exposure (ATSDR, 2004c).

C. Subacute and Chronic Toxicity

Chronic exposure to cyanide in the working environment caused eye irritation in exposed individuals. Decreased body weight was seen in workers occupationally exposed to hydrogen cyanide (ATSDR, 2004c). The central nervous system is the major target of cyanide toxicity in humans and animals. Chronic exposure to lower cyanide concentration causes a variety of symptoms from fatigue, dizziness, headaches, ringing in the ears, paresthesias of extremities, and syncopes. Respiratory and cardiovascular effects were also noted in after chronic exposure to cyanide, displaying the same symptoms as acute exposure (ATSDR, 2004c).

D. Carcinogenicity

There are no studies available on the carcinogenic effects in human or animals by any route of exposure. USEPA has not classified cyanide as a human carcinogen (Group D) due to inadequate or no evidence of carcinogenicity in animal studies.

E. Mutagenicity

Most of the genotoxic studies reported negative results for cyanide. These results indicate that cyanide is not mutagenic (ATSDR, 2004c).

F. Teratogenicity/Reproductive Effects

There is very limited information on the reproductive effects of cyanide. After oral exposure, increase resorptions and increase in gonadal weights was reported for rats. Teratogenic and fetotoxic effects were reported for rats and hamsters exposed to cyanide. These effects included limb defects, growth retardation, microcephaly with open eyes, encephalocele and rib abnormalities (ATSDR, 2004c).

G. Other Health Effects

Chronic oral exposure to cyanide in humans who ingest cassava roots (cyanide naturally occurs in this plant) or by occupational exposure has been associated with thyroid effects. Several animal studies support this association (ATSDR, 2004c).

H. Epidemiological Evidence

Case histories for workers or individuals who ingested cyanogenic food support the findings that this compound has neurological and thyroid effects. Dose relationships of these effects are not known (ATSDR, 2004c).

I. Toxicity Data

USEPA has classified cyanide into Group D, not classifiable as a human carcinogen. The chronic reference dose is 0.02 mg/kg-day for the oral route of exposure. The toxicity value was taken from USEPA, 2004a.

4.11 IRON

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	55.85
Melting Point:	1,535°C
Boiling Point:	3,000°C
Specific Density:	7.86
Water Solubility (mg/L):	no data
Vapor Pressure (mm Hg):	no data
Henry's Law Constant (atm-m^3/mol):	no data

Reference: Hazardous Substances Databank (HSDB), 2004.

B. Chemical Transformation

Air: No information was found on the atmospheric fate of iron, however, its mostly likely present as particulates and is removed from the atmosphere by dry and wet deposition.

Water: The principal compounds of iron are ferrous (+2) and ferric (+3). In general, ferrous and ferric forms are mutually interconvertible. Ferrous compounds are more stable than ferric when ionized, less stable when covalent A large proportion of iron salts are water soluble (Patty's, 1994).

Soil: No data available.

2. Toxicological Properties

A. Metabolism

Absorption of iron is considered a two-step process. The first step, chelate complex containing ferrous iron is taken up from the intestinal lumen by the mucosal cells. The second step involves iron from the mucosal cells is transformed across the cell membrane to serum, where it is bound to transferrin (located in the liver). It binds iron and thus protects the cells from potential toxic effect of free iron ions. It also transports iron to bone marrow and other storage tissues. The human body contains 3 to 5 g of iron. Two-thirds of this amount are bound to hemoglobin which is found almost entirely in blood and the remaining is bound to storage proteins: ferritin and hemosiderin. These storage proteins are mainly found in the liver, bone marrow and spleen. Non-absorbed iron is eliminated from the body via feces (Friberg et al., 1986).

B. Acute Toxicity

Acute oral exposure to iron may result in iron intoxication. Intoxication is most common in children in the age group of 1 to 5 due to their eating ferrous sulfate tablets, however there is one reported case of an adult. In this case a worker fell into a vat of FeCl3. The first symptom is gastrointestinal irritation and damage which includes vomiting. Due to ulceration of the gastrointestinal mucosa, vomitus frequently becomes bloody and stool may turn black. Central nervous system depression, as well as cardiovascular symptoms, such as pallor, tachycardia and hypotension may occur. Following initial intoxication phase, the patient may appear to recover, however within 12 to 48 hours, life-threatening symptoms can appear. These include gastrointestinal perforation, coma, convulsions, vasomotor collapse, cyanosis and pulmonary edema. Hepatorenal failure may also develop (Patty's, 1994; Friberg et al., 1986).

C. Subacute and Chronic Toxicity

Chronic oral exposure to iron has been reported to result in hemosiderosis or hemochromatosis. Hemosiderosis is a condition in which there is a generalized increase in the iron content in the body tissues, typically the liver and spleen. Hemochromatosis is marked by the accumulation of iron, as in Kupffer cells in the liver and reticuendothelial cells in the splean and bone marrow. This is accompanied by fibrotic changes in the affected organ. Clinical symptoms of hemochromatosis include skin pigmentation, diabetes mellitus, hepatomegaly with symptoms of hepatic dysfunction and signs of panhypopituitarism (Patty's, 1994; Friberg et al., 1986).

Pulmonary siderosis results from chronic inhalation of iron dust or fumes. Three cases of severe pulmonary changes related to iron oxide exposure from welding fumes reported symptoms of persistent cough

and shortness of breath (Friberg et al., 1986). This condition in mottling of lungs, is considered to be benign pneumoconiosis (HSDB, 2004).

Exposure to iron dust has been reported to produce "rust ring" of the cornea. This is often associated with sensation of irritation and hyperemia of conjunctiva (HSDB, 2004).

D. Carcinogenicity

USEPA has not given iron a weight of evidence classification as to carcinogenicity.

E. Mutagenicity

No data available.

F. Teratogenicity/Reproductive Effects

No data available.

G. Other Health Effects

It is a known fact that humans need iron and iron deficiency is probably the most prevalent deficiency state affecting the human population. Early symptoms of iron deficiency in humans is mild to moderate anemia which may lead to lowered working capacity. If the deficiency persists the anemia is exacerbated and clinical manifestations occur, including fatigue, headache, and anorexia. In more severe cases, abnormalities in epithelial tissues occur with sore mouth and tongue, and angular stomatitis (Friberg et al., 1986).

H. Epidemiological Evidence

No data available.

I. Toxicity Data

USEPA has not classified iron as to its carcinogenicity. The chronic oral reference dose is 0.3 mg/kg-day. The aforementioned toxicity value was taken from USEPA, 2004b.

4.12 LEAD

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	207
Melting Point:	327.4°C
Boiling Point:	1,740°C
Specific Density:	11.34 (@ 20°C)
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	1.77 (@ 1000°C)

Henry's Law Constant (atm-m^3/mol): no data
Reference: ATSDR, 1999d. Toxicological Profile for Lead.

B. Chemical Transformation

Air: In the atmosphere, inorganic lead exists primarily in particulate form. Lead particles are dispersed and ultimately removed by dry and wet deposition. Monitoring studies indicate that tetraalkyl lead react with hydroxyl ions to form ionic trialkyl and dialkyl species (ATSDR, 1999d).

Water: In most waters, the concentration of dissolved lead will be low since lead will form compounds with anions in the water such as hydroxides, carbonates, sulfates and phosphates that have low water solubilities and will precipitate out of the water column. Under anaerobic conditions, organic and inorganic lead may transform to tetraalkyl lead (ATSDR, 1999d).

Soil: Inorganic lead will tend to sorbed to soil partiles. Limited data indicate that tetraethyl and tetramethyl lead compounds are transformed to water-soluble lead (ATSDR, 1999d).

2. Toxicological Properties

A. Metabolism

Inorganic lead ion in the body is not known to be metabolized or biotransformed. Primarily, it is absorbed, distributed, and then excreted, often in conjugated form. About 99% of the amount of lead taken into the adult body will leave in the waste within a couple of weeks. However, only 32% of lead taken in by the body of a child will leave in the waste thus lead accumulates in body tissues, notably bone. Conversely, alkyl lead compounds are actively metabolized in the liver by oxidative dealkylation catalyzed by cytochrome P-450 (ATSDR, 1999d).

B. Acute Toxicity

Lead has been shown to affect virtually every organ and/or system in the body in both humans and animals. The most sensitive target organs appear to be the nervous system, the hematopoietic system, and the cardiovascular system (ATSDR, 1999d).

The evidence from occupational, clinical, and general population studies suggest that lead affects the cardiovascular system in humans, producing cardiac lesions and electrocardiographic abnormalities at high levels of exposure (600 to 4,000 $\mu g/m^3$) and increases in blood pressure, particularly in middle-aged men, at very low levels of exposure with no evident threshold through the lowest blood levels, 7 $\mu g/dL$ (ATSDR, 1999d).

Colic is a consistent early symptom of lead poisoning in occupationally exposed cases or in individuals, particularly children, acutely exposed to high levels of lead. It is characterized by a combination of the following

symptoms: abdominal pain, constipation, cramps, nausea, vomiting, anorexia, weight loss, and decreased appetite. These gastrointestinal effects typically occur in occupational workers at blood lead levels ranging from 100 to 200 μg/dL. Colic in children was observed at blood lead levels of greater than or equal to 60 μg/dL (ATSDR, 1999d).

Lead inhibits the activity of certain enzymes involved in heme biosynthesis. A marked interference with heme synthesis reduces the hemoglobin concentration in blood coupled with an increase in erythrocyte destruction (another result of increased lead levels), resulting in lead-induced anemia. USEPA estimated that the threshold blood lead level for a decrease in hemoglobin is 50 and 40 μg/dL for occupational workers and children, respectively (ATSDR, 1999d).

In humans, encephalopathy can occur at blood levels as low as 100 to 120 μg/dL in some adults and at blood levels as low as 80 to 100 g/dL in some children. This impairment can result in death or in permanent cognitive impairment, particularly in children (ATSDR, 1999d).

C. Subacute and Chronic Toxicity

Exposure to lead that resulted in blood levels of approximately 40 μg/dL to >100 g/dL has been associated with nephropathy in lead-exposed workers. Characteristics of chronic lead nephropathy include progressive interstitial fibrosis, dilation of tubules and atrophy or hyperplasia of the tubular epithelial cells. The chronic form is reported mainly in lead workers, whose primary exposure is via inhalation (ATSDR, 1999d).

D. Carcinogenicity

USEPA has concluded that the animal data are sufficient to demonstrate that lead and (inorganic lead) compounds, particularly soluble lead salts, are carcinogenic (ATSDR, 1999d). USEPA classified lead as a probable human carcinogen (Group B2) with sufficient evidence of carcinogenicity in animal studies.

E. Mutagenicity

Studies on humans do indicate that lead is mutagenic. It was reported that workers exposed to lead had a positive correlation between chromosomal aberrations and blood levels (ATSDR, 1999d).

F. Teratogenicity/Reproductive Effects

There is sufficient qualitative evidence to support the conclusion that at high exposure levels, lead has significant adverse effects on human reproduction. Men exposed to lead have reproductive effects that include asthenospermia, hypospermia, and teratospermia. These effects are related to blood levels. Women exposed to lead have reproductive effects that include spontaneous abortions and stillbirths. Because of prenatal exposure to lead, fetuses have been reported to have low birth weights and adverse neurobehavioral affects (ATSDR, 1999d).

G. Other Health Effects

Occupational workers exposed to high levels of lead have been reported to exhibit a bluish-tinged line in the gums, called the "lead-line". In addition, case reports of high occupational exposure have reported the occurrence of muscle weakness, cramps and joint pains. Epidemiological cases of children exposed to lead, have reported decrease in growth. There are some rat studies supportive of these cases where after oral lead exposure it was reported that lead impaired normal bone growth (ATSDR, 1999d).

H. Epidemiological Evidence

Many epidemiological studies have been performed that support the toxicity of lead on the human population.

I. Toxicity Data

No data available.

4.13 MANGANESE

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	54.94
Melting Point:	1,244°C
Boiling Point:	1,962°C
Specific Density:	7.21
Water Solubility (mg/L):	decomposes
Vapor Pressure (mm Hg):	1 (@ 1292)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 2000f. Toxicological Profile for Manganese.

B. Chemical Transformation

Air: In the atmosphere, manganese reacts with sulphur dioxide and nitrogen dioxide (ATSDR, 2000f).

Water: Manganese in water may undergo oxidation at high pH or EH and is also subject to microbial activity. The microbial metabolism of manganese is a function of pH, temperature and other factors (ATSDR, 2000f).

Soil: The oxidation state of manganese in soils may be altered by microbial activity. It was observed that manganese (II) in suspensions of silt or clay loams was oxidized by microorganisms, leading to the precipitation of manganese minerals. Other studies have shown that bacteria and

microflora can increase the mobility by increasing dissolution of manganese in subsurface environments (ATSDR, 2000f).

2. Toxicological Properties

A. Metabolism

Manganese (Mn) is capable of existing in several oxidation states and limited suggest that manganese may undergo changes in oxidation state in the body. Circumstantial support comes from the observation that the oxidation state of manganese ion in several enzymes appears to be Mn(III), while most Mn intake from the environment is either Mn(II) or Mn(VI). In humans, absorbed Mn is removed from the blood by the liver where it conjugates with bile and excreted in the intestine. Biliary secretion is the main pathway by which manganese reaches the intestines where most of the element is excreted in the feces (ATSDR, 2000f).

B. Acute Toxicity

Studies in humans and animals indicate that manganese has very low acute toxicity by any route. Acute inhalation exposure to high concentrations of manganese dust can cause inflammatory response in the lungs, which can lead to impaired lung function as seen with any inhalable particulate matter. Large oral doses of manganese salts given by gavage resulted in death for animals but oral exposures via food or drinking water have not been found to cause acute toxicity effects (ATSDR, 2000f).

C. Subacute and Chronic Toxicity

While manganese is clearly an essential element, it has also been demonstrated to be the causative agent in a syndrome of neurologic and psychiatric disorders. Among the primary effects associated with Mn toxicity from inhalation exposure in humans are signs and symptoms of CNS toxicity, called manganism. Although the course and degree of Mn intoxication can vary greatly among individuals, manganism is generally considered to consist of two or three phases. The first is the psychiatric aspect, which includes disturbances such as excessive weeping and laughing, sleep disturbance, irritability, apathy, and anorexia. These symptoms can occur independently of the second phase, neurological signs. The latter may include gait disturbances, dysarthria, clumsiness, muscle cramps, tremor, and mask-like facial expression. In addition, there may be a final stage of Mn intoxication involving symptoms of irreversible dystonia and hyperflexion of muscles that may not appear until many years after the onset of exposure (ATSDR, 2000f; USEPA, 1996b).

In contrast to inhaled manganese, ingested manganese has rarely been associated with toxicity. There is one study done regarding manganese toxicity in humans via the oral route through drinking water. Manganese poisoning was reported with symptoms including lethargy, increased

muscle tonus, tremor and mental disturbances. The most severe symptoms were observed in elderly people, while children appeared to be unaffected (ATSDR, 2000f; USEPA, 1996b).

D. Carcinogenicity

USEPA has not classified manganese as a probable human carcinogen (Group D) based on inadequate human and animal data (USEPA, 1996b).

E. Mutagenicity

One study on occupationally exposed welders reported manganese may cause chromosomal aberrations, however the welders were exposed to other compounds that have these tendencies and therefore the observed genotoxic effect could not attributed solely to manganese. Mutagenicity studies in both mammalian and bacteria strains are equivocal. *In vitro* and *in vivo* assays also gave both positive and negative results (ATSDR, 2000f).

F. Teratogenicity/Reproductive Effects

A primary endpoint of Mn toxicity has been male reproductive dysfunction, often manifesting in symptoms such as loss of libido, impotence, and similar complaints. Some neuropathological evidence suggests that the hypothalamus is a site of Mn accumulation; thus, disturbance of the hypothalamic- pituitary-gonadal axis hormones might be expected (USEPA, 1996b). Although effects on libido and sexual performance are partly neurologic in origin, recent human and animal studies indicate manganese is damaging to the testes (ATSDR, 2000f).

The developmental effects of Mn have been investigated primarily from the viewpoint of the nutritional role of this element and therefore have generally involved oral exposure. Some studies indicate that neonates of various species have a greater body burden of Mn than mature individuals have, possibly because neonates do not develop the ability to eliminate Mn, and thereby maintain Mn homeostasis until some time after birth. Moreover, some evidence suggests that the neonate's inability to maintain Mn homeostasis is due to a limitation in the elimination of Mn rather than in its gastrointestinal absorption, which would suggest a potentially greater vulnerability of young individuals to excessive Mn exposure regardless of the route of exposure (USEPA, 1996b). Several studies have demonstrated neurochemical alterations in young rats and mice exposed postnatally to Mn by routes other than inhalation. There is one inhalation study where female mice were exposed to MnO2 7 hours/day, 5 days/week for 16 weeks prior to conception and for 17 days following conception (i.e., gestational days 1 to 18). For the first 12 weeks, the air concentration was 49.1 mg Mn/cu.m; all later exposures were at 85.3 mg Mn/cu.m. To separate prenatal and postnatal exposure effects, a cross-fostering design was used. Although mothers exposed to MnO2 prior to conception produced significantly worse pups per litter,

prenatally exposed offspring showed reduced scores on various activity measures (open field, roto-rod, and exploration) and retarded growth that persisted into adulthood. A decrease in roto-rod performance was also observed in the offspring of nonexposed mice that were fostered to Mn-exposed females during lactation. Thus, balance and coordination were affected by either gestational or postpartum exposure to MnO2 (USEPA, 1996b).

G. Other Health Effects

The respiratory system is another primary target for Mn toxicity; numerous reports of Mn pneumonitis and other effects on the respiratory system have appeared in the literature, dating back to 1921. In a cross-sectional study of workers exposed to mixed Mn oxides and salts, it was found that significantly greater prevalences of coughs during the cold season, dyspnea during exercise, and recent episodes of acute bronchitis were reported in the exposed group on a self-administered questionnaire. However, objectively measured lung function parameters were only slightly altered and only in Mn-exposed (USEPA, 1996b). Significant increases in past history of pneumonia, eye problems, clogged nose, nose colds, throat swelling and soreness, and other symptoms were noted among students in the school 100 metres from a ferromanganese plant. Those living closest to the plant reported more throat symptoms and past history of pneumonia than did students living farther away. Pulmonary function tests revealed statistically significant decreases in maximum expiratory flow, forced vital capacity, forced expiratory volume at 1 second, and the FVC:FEV- 1 ratio in the students attending the school closer to the plant, with some measures suggesting a relationship between performance and distance of residence from the plant (ATSDR, 2000f; USEPA, 1996b).

Manganese is a ubiquitous element that is essential for normal physiologic functioning in all animal species. Several disease states in humans have been associated with both deficiencies and excess intakes of manganese. These include epilepsy, exocrine pancreatic insufficiency, multiple sclerosis, cataracts, and osteoporosis. Experiments in several species have shown a deficiency in dietary manganese to result such as disorders in lipid and carbohydrate metabolism, impaired growth and reproductive function, and ataxia and skeletal abnormalities in neonates (ATSDR, 2000f; USEPA, 1996b).

H. Epidemiological Evidence

There are several studies and/or case reports in humans that support the above information (ATSDR, 2000f).

I. Toxicity Data

USEPA has not classified manganese as a probable human carcinogen due to inadequate data. The chronic reference doses are 0.02 and

1.43E-05 mg/kg-day for the oral and inhalation routes, respectively. All of the aforementioned toxicity values were taken from USEPA, 2004a.

4.14 MERCURY

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	200.59
Melting Point:	-38.87°C
Boiling Point:	356.72°C
Specific Density:	13.53 (@ 20°C)
Water Solubility (mg/L):	0.28
Vapor Pressure (mm Hg):	0.002 (@ 20°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 1999e. Toxicological Profile for Mercury.

B. Chemical Transformation

Air: The primary form of atmospheric mercury, metallic mercury vapor is oxidized by ozone to other mercury forms and is removed from the atmosphere by precipitation. The overall residence time of elemental mercury have been reported to be 6 days to 2 years. The main atmospheric transformation process of organomercurials is reported to be photolysis (ATSDR, 1999e).

Water: The most important transformation process of mercury in water is biotranformation. Any form of mercury entering waters can be microbially coverted to methylmercuric ions. Volatile elemental mercury may be formed through the demethylation of methylmercury or the reduction of inorganic mercury. Abiotic reduction of inorganic mercury to metallic mercury in aqeuous solutions can also occur, particularly in the presence of soluble humic substances. This reduction is enhanced by light and can occur under both aerobic and anaerobic conditions (ATSDR, 1999e).

Soil: Mercury compounds in soils may undergo the same fate process as those seen in waters. The formation and degradation of organic mercurials occurs through biotransformation and may also undergo abiotic processes. Mercuric mercury usually forms various complexes with chloride and hydroxide ions in soils (ATSDR, 1999e).

2. Toxicological Properties

A. Metabolism

Metallic mercury vapor is inhaled through the lungs and rapidly enters the bloodstream. The dissolved vapor undergoes rapid oxidation, primarily in the red blood cells, to its inorganic divalent form by the hydrogen peroxide-catalase pathway. The oxidation of metallic mercury may also occur in the brain, liver, lungs and other tissue to some extent. Unoxidized metallic mercury can still be transported to the brain since the oxidization process is slow compared to circulation time from the lungs to the brain. Unoxidized metallic mercury in the brain can be oxidized and become trapped in the brain tissues. There is evidence from animal studies that divalent inorganic mercury cation is reduced by mammalian tissues to metallic mercury after its oxidation (ATSDR, 1999e).

Once absorbed, organic mercury can be converted to inorganic mercury in tissues, specifically the divalent cation. Inorganic mercury production from methylmercury paralleled the hydroxy radical production, as well as phenylmercury. The promotion and inhibition of the hydroxyl radical formation and the hydroxyl radical scavenger, affected inorganic mercury production (ATSDR, 1999e).

Elimination of metallic mercury occurs through the urine, feces and expired air, while inorganic mercury is excreted through the urine and feces. The body burden half-life of these mercuries has been reported to be 1 to 2 months. Excretion of organic mercury is predominantly through the fecal (biliary) route. Elimination rates of organic mercury is dependant on dose, route of exposure and duration of exposure (ATSDR, 1999e).

B. Acute Toxicity

The inhalation of metallic mercury and organic mercury vapors, and the ingestion of organic and inorganic mercury can be fatal to humans and experimental animals. Lethal doses after acute oral exposure to inorganic mercury have been estimated to be 29 to 50 mg mercury/kg. Deaths were attributed to renal failure, cardiovascular collapse and severe gastrointestinal damage. Lethal doses for organic mercury have been estimated to be 10 to 60 mg mercury/kg, deaths attributed to central nervous system toxicity (ATSDR, 1999e).

Respiratory, cardiovascular, gastrointestinal, hematological, neurological and renal effects have been observed in both humans and animals after acute-duration inhalation exposure to metallic mercury. Tremors, irritability, and decreased motor functions and reflexes were common neurological symptoms following high-level acute duration exposures to metallic mercury vapors. Short-term exposure to high levels of metallic mercury vapor can also damage the lining of the mouth and irritate the

respiratory tract, causing tightness of the breath, burning sensation in the lungs and coughing. Other effects from mercury vapor exposure include nausea, vomiting, diarrhea, increase in blood pressure or heart rate, skin rashes, and eye irritation (ATSDR, 1999e).

Acute oral exposures to organic and inorganic mercury has been reported to result in neurological and renal effects. Other effects from ingestion of inorganic mercury include gastrointestinal effects, producing symptoms of nausea, diarrhea, or severe ulcers. Effects on the heart have also been observed after accidental ingestion of mercuric chloride, symptoms include rapid heart rate and increased blood pressure (ATSDR, 1999e).

Neurological, renal, and gastrointestinal effects have been reported after acute dermal exposure to organic and inorganic mercury compounds. Acute dermal exposure to organic and inorganic mercury compounds have also been reported to result in dermatitis. Symptoms include rashes and blisters on the skin (ATSDR, 1999e).

C. Subacute and Chronic Toxicity

Substantial evidence from occupational exposures to mercury compounds indicate that chronic inhalation of metallic mercury vapors results in neurotoxicity. Chronic oral exposure to mercurous chloride resulted in dimentia and irritability. Neurological effects from organic mercury oral exposure is provided by a study where humans ingested methylmercury-contaminated fish. Chronic occupational exposure to alkyl mercury compounds also resulted in neurological changes in humans. Other effects from occupational exposure to metallic mercury vapors have been reported to result in adverse cardiovascular, gastrointestinal, renal, ocular, and immunological effects (ATSDR, 1999e). The mercury levels reported to be associated with preclinical and symptomatic neurological dysfunction are generally lower than those found to affect other bodily functions (i.e., kidney, pulmonary).

D. Carcinogenicity

USEPA has not classified mercury as a probable human carcinogen (Group D) based on inadequate human and animal data. Epidemiologic studies failed to show a correlation between exposure to elemental mercury vapor and carcinogenicity; the findings in these studies were confounded by possible or known concurrent exposures to other chemicals, including human carcinogens, as well as lifestyle factors (USEPA, 1995d).

E. Mutagenicity

Cytogenetic monitoring studies of workers occupationally exposed to mercury by inhalation provide very limited evidence that mercury adversely affects the number or structure of chromosomes in human somatic cells. One study compared four men exposed to elemental mercury vapor with an unexposed group and found a statistically

significant increase in the incidence of chromosome aberrations in the WBCs from whole blood. Another found an increase in aneuploidy after exposure to low concentrations of vapor, but results could not be repeated in later studies. In contrast, one study did not find increases in structural chromosomal aberrations of lymphocytes of exposed workers. Similarly, another study found no increase in the incidence or size of micronuclei and no correlation between micronuclei and blood or urinary mercury levels of chloralkali workers. A statistically significant correlation was observed between cumulative exposure to mercury and micronuclei induction in T lymphocytes in exposed workers, suggesting a genotoxic effect. Findings from the genotoxicity tests are severely limited and provide equivocal evidence that mercury adversely affects the number or structure of chromosomes in human somatic cells (USEPA, 1995d).

F. Teratogenicity/Reproductive Effects

Occupational exposure to metallic mercury has not been shown to result in significant effects on male fertily, however, increase in the rate of spontaneous abortions may occur. After an acute dose of mercuric chloride, spontaneous abortion resulted for a female. Abortions and decreased mean litter size have been reported in animals studies following oral exposure to organic mercury. Adverse effects on spermatogenesis and on histopathology of the testes have been reported in several studies in animals exposed to methylmercury (ATSDR, 1999e).

Occupational exposure to metallic mercury in males did not result in significant developmental effects. The results from an inhalation developmental animal study suggest that metallic mercury vapors may cause a higher incidence of fetal malformations, resorptions, and deaths. Prenatal exposure to methylmercury during the early stages of pregnancy resulted in neurological damage. Severe neurological impairment was noted in a child whose mother ingested contaminated bread in utero. The child was still affected 6 years later. Several animal studies exposed to methylmercury reported disruptions in the development of the nervous system and in the immune system. Inorganic mercury exposure caused significant increase in the incidence of resorptions in hamsters (ATSDR, 1999e).

G. Other Health Effects

Probably the most widely recognized form of hypersensitivity to mercury poisoning is the uncommon syndrome known as acrodynia, also called erythredema polyneuropathy or pink disease. Acrodynia may result after exposure through inhalation of mercury vapors, ingestion of organic mercury or mercury salts, or dermal application of mercury-containing ointments. Infantile acrodynia was first described in 1828 and was typically considered that this syndrome occurred exclusively in children, but recent reported cases in teenagers and adults have shown these

groups to be susceptible. Acrodynia is often characterized by severe leg cramps, irritability, erythema and subsequent peeling of the hands, nose, and soles of the feet. Itching, swelling, fever, tachycardia, elevated blood pressure, excessive salivation and perspiration, rashes, fretfulness, sleeplessness, and weakness maybe also be present. Acrodynia has generally been associated with short-term exposures and with urine levels of 50 μg/L or more (ATSDR, 1999e; USEPA, 1995d).

H. Epidemiological Evidence

Occupational studies conducted on workers exposed to metallic mercury vapors, concluded that mercury exposure causes neurological effects (ATSDR, 1999e).

I. Toxicity Data

USEPA has not classified mercury as a probable human carcinogen due to inadequate data. A chronic inhalation reference dose is reported to be 8.6E-05 mg/kg-day for mercury. Due to the lack of an oral RfD, typically the oral RfD for mercuric chloride is substituted. A chronic oral reference dose is reported to be 0.0003 mg/kg-day for mercuric chloride. Methyl mercury has a chronic oral reference dose of 0.0001 mg/kg-day. The aforementioned toxicity values were taken from USEPA, 2004a.

4.15 NICKEL

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	58.69
Melting Point:	1,455°C
Boiling Point:	2,730°C
Specific Density:	8.91 (@ 20°C)
Water Solubility (mg/L):	1.13 (@ 37°C)
Vapor Pressure (mm Hg):	1 (@ 1810°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 2003a. Toxicological Profile for Nickel.

B. Chemical Transformation

Air: In the atmosphere, nickel strongly adsorbs to particulates. The predominant fate of airborne nickel is by dry or wet deposition (ATSDR, 2003a).

Water: In natural waters, nickel predominantly exists as the hexahydrate. Under anaerobic conditions, would reduce free aqueous nickel concentrations to low levels. Precipitation can remove soluble nickel from water. Nickel may also be removed by coprecipitation with hydrous iron and manganese oxides.

Nickel removed by these processes will settle to the sediment (ATSDR, 2003a).

Soil: In soils, nickel will typically not dissolve and therefore, strongly bounds to soil particulates. Under acidic conditions, nickel may leach to groundwater (ATSDR, 2003a).

2. Toxicological Properties

A. Metabolism

The metabolism of nickel consists of ligand exchange reactions. In human serum, nickel binds to albumin, L-histidine, and a-2-macroglobulin. Absorbed nickel is excreted in the urine regardless of the route of exposure (ATSDR, 2003a).

B. Acute Toxicity

The primary causes of death in workers exposed to nickel were nonmalignant respiratory disease and nasal and lung cancers. A worker died due to respiratory tract injury following a 90-minute exposure to very high concentration (382 mg/m^3) of metallic nickel. Histological changes noted were alveolar wall damage and edema in the alveolar space. A child who accidentally ingested nickel sulfate (570 mg nickel/day) died from cardiac arrest. In lethality studies in animals, soluble nickel compounds were more toxic than the insoluble nickel compounds. Clinical signs observed prior to death from oral exposure included lethargy, ataxia, irregular breathing, cool body temperature, salivation, squinting and loose stools (ATSDR, 2003a).

Acute inhalation exposure to nickel results in respiratory tract effects. Alveolitis, chronic lung inflammation, alveolar macrophage hyperplasia, and atrophy of the nasal olfactory epithelium have been observed in animal studies. Adverse gastrointestinal and neurological effects were observed in workers who ingested drinking water contaminated (7.1 to 35.7 mg/kg) with nickel. Symptoms include nausea, cramps, diarrhea, vomiting, giddiness and weariness. Loss of vision for 2 hours was found in one man following ingestion of nickel sulfate at a concentration of 0.05 mg/kg. Lethargy, ataxia, and prostration were seen following oral exposure of rats for 90 days to concentrations of >1.2 mg/kg-day (ATSDR, 2003a).

Contact dermatitis, resulting from dermal exposure to nickel, is the most prevalent effect of nickel in the general population. The most common reaction is a skin rash at the site of contact. Less frequently, some people who are sensitive to nickel may have asthma attacks. Once an individual is sensitized, even minimal contact with nickel by any route of exposure will elicit a reaction (ATSDR, 2003a).

C. Subacute and Chronic Toxicity

As with acute-duration exposures, chronic inhalation exposure to nickel and nickel compounds resulted in chronic active lung inflammation. A 2-year exposure to nickel sulfate resulted in chronic lung inflammation, bronchialization and atrophy of the olfactory epithelium. A similar study reported chronic lung inflammation, alveolar epithelium hyperplasia, fibrosis and rapid and shallow breathing. Several animal studies have assessed the toxicity of nickel following chronic oral exposure to nickel. Significant decreases in body weight and organ weight (liver, kidney, pituitary) were consistently observed in rats exposed to 8.6 mg/kg/day and higher to several nickel compounds. Other systemic effects included changes in blood glucose and minimal convoluted tubular damage in the kidneys (ATSDR, 2003a).

D. Carcinogenicity

Carcinogenicity of nickel in workers has been well documented. Predominant forms of cancer were lung and nasal. Increases in the incidence of lung tumors have also been observed in animal studies. USEPA has determined nickel refinery dust and nickel subsulfide to be probable human carcinogens (Group A). Other nickel compounds have not been classified by USEPA as to human carcinogenicity (ATSDR, 2003a).

E. Mutagenicity

Epidemiological studies have reported a higher incidence of chromosomal aberrations in nickel workers compared to controls. Both *in vitro* and *in vivo* animal studies suggest nickel to be mutagenic on cellular DNA (ATSDR, 2003a).

F. Teratogenicity/Reproductive Effects

An increase in the abortion rate and structural malformations in infants were reported in women who worked in a nickel hydrometallurgy refining plant. A number of oral exposure animal studies suggest that nickel can result in testicular and epididymal damage. Decreases in sperm motility and count, and sperm abnormalities were also reported. Decreased fetal body weight was observed in offspring of rats exposed to nickel via inhalation. Developmental effects such as increased pup mortality, decreased pup survival, and decreased pup weights were reported in oral exposure animal studies to nickel (ATSDR, 2003a).

G. Other Health Effects

No data available.

H. Epidemiological Evidence

Information of the toxicity of nickel in humans comes from occupational studies, primarily nickel refinery workers, and studies/reports of allergic contact dermatitis in nickel-sensitized individuals. Neoplastic and

nonneoplastic lung and nasal effects have been found in occupational studies. Nickel sensitivity has been observed in workers and the general population (ATSDR, 2003a).

I. Toxicity Data

USEPA has not evaluated nickel for potential human carcinogenicity. However, nickel refinery dust and specific nickel compounds - nickel carbonyl and nickel subsulfide - have been evaluated. A chronic oral reference dose is reported to be 0.02 mg/kg-day for nickel. The aforementioned toxicity value was taken from USEPA, 2004a.

4.16 SELENIUM

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	78.96
Boiling Point:	685°C
Melting Point:	221°C
Specific Density:	4.81 (@ 20°C)
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	1 (@ 356°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 2003b. Toxicological Profile for Selenium.

B. Chemical Transformation

Selenium in the environment is influenced by its oxidation state and the differences in behavior depends on its different chemical compounds. The oxidation state of selenium in the environment is dependent on ambient conditions, particularly on pH, pE, and biological activity (ATSDR, 2003b).

Air: Hydrogen selenide is highly reactive in air and rapidly decomposes to elemental selenium and water, but the other compounds of selenium persist in air. Selenium in the atmosphere is removed by dry and wet deposition (ATSDR, 2003b).

Water: The formation of various metal selenides is favored by acidic and reducing conditions. Aquatic organisms can convert selenium to both inert and soluble forms (ATSDR, 2003b).

Soil: In soils, elemental selenium and inorganic selenium compounds such as sodium selenite can be methylated by microorganisms and subsequently volatilized to the atmosphere (ATSDR, 2003b).

2. Toxicological Properties

A. Metabolism

For selenium to become incorporated into selenium-specific proteins through contranslational mechanism requires selenium be in the form of selenide. All forms of selenium can be transformed to selenide, however, the rates of transformation vary for each parent compound. For example, selenate is not converted to selenide as readily as selenite. Excess selenium is methylated and exhaled or excreted in the urine. The metabolite dimethyl selenide is exhaled and trimethylselenonium ion is the major urinary metabolite of selenium (ATSDR, 2003b).

B. Acute Toxicity

Systemic effects in humans and animals following oral and inhalation exposure to selenium compounds are similar. The primary target organ in humans and in animals following acute exposure by inhalation or oral routes is the lung, with effects also occurring on the cardiovascular, hepatic, and renal systems and lesser effects on all but the muscular/skeletal system (ATSDR, 2003b).

Following acute inhalation or oral exposure to selenium compounds, the primary sign of selenium toxicity in both humans and animals is respiratory distress. Clinical signs in humans include bronchial spasms, severe bronchitis, and bronchial pneumonia, and signs in animals have included labored breathing and bronchial pneumonia. Following acute inhalation or oral exposure to selenium compounds, some cardiovascular signs such as elevated pulse rate and tachycardia have been reported in humans (ATSDR, 2003b).

In animals, the liver is affected following both inhalation and oral exposure to several different selenium compounds. Hepatocellular degeneration, cirrhosis, and changes in the liver enzymes have been reported in several animal studies (ATSDR, 2003b). Mild kidney effects have also been reported in animal studies. These effects include hydropic degeneration, interstitial nephritis, and renal papilla degeneration (ATSDR, 2003b).

C. Subacute and Chronic Toxicity

Several occupational studies of chronic inhalation exposure reported primarily respiratory effects to include irritation of the nose, respiratory tract, and lungs, bronchial spasms, and coughing. Following intermediate and chronic oral exposure to selenium compounds produces selenosis, the primary effects in humans of which are dermal and neurological. The dermal manifestations following chronic oral exposure to predominantly organic selenium compounds found in food include loss of hair, deformation and loss of nails, and discoloration and excessive decay of teeth. Following chronic oral exposure to selenium compounds in the diet, neurological manifestations in humans have been reported to

include numbness, paralysis, and occasionally hemiplegia (ATSDR, 2003b).

Some evidence for adverse effects on the endocrine system has also been reported following chronic exposure to elevated levels of dietary selenium in humans and animals. Both human and animals studies have reported decreases in thyroid hormone levels. Two of the most common effects in animals noted were reduced growth rate of young animals and loss of weight by older animals. There is evidence that suggest these effects may be due to the interactions of selenium with hormones that regulate normal growth and body weight (ATSDR, 2003b).

D. Carcinogenicity

Studies in animals and humans have shown that selenium compounds don't cause cancer. Studies of cancer in humans suggest that lower-than-normal selenium levels in the diet might increase risk of cancer, however, levels of selenium in the diet higher than normal have not shown to reduce the risk of cancer, just the possibility of selenium poisoning (ATSDR, 2003b). USEPA has not identified selenium as a probable human carcinogen (Group D) due to inadequate human data and inadequate evidence of carcinogenicity in animal studies. However, USEPA has determined that one specific form of selenium, called selenium sulfide, is a probable human carcinogen. Selenium sulfide is a very different chemical from the organic and inorganic selenium compounds found in the environment and exposure to this compound is considered minimal to none.

E. Mutagenicity

Genotoxicity studies indicate that inorganic compounds of selenium have both genotoxic and antigenotoxic effects (ATSDR, 2003b).

F. Teratogenicity/Reproductive Effects

There is no evidence that selenium produces reproductive effects in humans, however, animal studies have reported abnormal sperm counts and testicular hypertrophy in males, and affect the estrous and menstrual cycles in females. Developmental studies by the oral route of administration reported fetal toxicity and reduced growth in experimental animals, but only at doses that produced signs of severe maternal toxicity (ATSDR, 2003b).

G. Other Health Effects

Humans exposed to high levels of selenium have been reported to have a noticeable garlic odor of the breath, probably due to excretion of dimethylselenide in expired air (ATSDR, 2003b). Acute oral exposure to high concentrations of selenium produces nausea, vomiting, and diarrhea in humans. Workers exposed to high concentrations of selenium dust reported stomach pain and headaches (ATSDR, 2003b).

H. Epidemiological Evidence

A few epidemiological studies in humans correlated toxicity to selenium blood levels (ATSDR, 2003b).

I. Toxicity Data

USEPA has classified selenium to be a non-carcinogenic compound. A chronic oral reference dose is reported to be 0.005 mg/kg-day for selenium. The aforementioned toxicity value was taken from USEPA, 2004a.

4.17 SILVER

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	107.87
Melting Point:	961.9°C
Boiling Point:	2,212°C
Specific Density:	10.50 (@ 20°C)
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	100 (@ 1865°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 1990b. Toxicological Profile for Silver.

B. Chemical Transformation

Air: Silver particulates released to the atmosphere are likely to become coated with silver oxide, silver sulfide and silver carbonate as the particles. Dry and wet deposition of silver particles is the predominant fate (ATSDR, 1990b).

Water: In water, silver may form complex ions with chlorides, ammonium, and sulfates to form soluble organic compounds or may become adsorbed onto humic complexes and suspended particles (ATSDR, 1990b).

Soil: Silver tends to form complexes with inorganic chemicals and humic substances in soils (ATSDR, 1990b).

2. Toxicological Properties

A. Metabolism

The deposition of silver in tissues is the result of precipitation of insoluble silver salts. These insoluble salts appear to be transformed into soluble silver sulfide albuminates, to bind to or form complexes with amino or carboxyl groups in RNA, DNA, and proteins, or to be reduced to metallic silver by ascorbic acid or catecholamines. The metallic silver is then

oxidized by tissue and bound as black silver sulfide. Excretion of silver usually occurs within a week through the feces (ATSDR, 1990b).

B. Acute Toxicity

Exposure to dust containing high levels of silver compounds such as silver nitrate or silver oxide has been reported to cause upper and lower respiratory irritation. In one case, inhalation of silver during work with molten silver ingots produced respiratory failure the day after exposure. Evidence that silver can act as an irritant is provided by the fact that ultrastructural damage was seen in the tracheal epithelium in animal studies following inhalation exposure. Dermal contact with silver compounds has been found to cause mild allergic reactions, such as rash, swelling, and inflammation (ATSDR, 1990b).

C. Subacute and Chronic Toxicity

The critical effect in humans ingesting silver is argyria, a medically benign but permanent bluish-gray discoloration of the skin. Argyria results from the deposition of silver in the dermis and also from silver-induced production of melanin. Although silver has been shown to be uniformly deposited in exposed and unexposed areas, the increased pigmentation becomes more pronounced in areas exposed to sunlight due to photoactivated reduction of the metal. Although the deposition of silver is permanent, it is not associated with any adverse health effects. No pathologic changes or inflammatory reactions have been shown to result from silver deposition. Silver compounds have been employed for medical uses for centuries. In the nineteenth and early twentieth centuries, silver arsphenamine was used in the treatment of syphilis; more recently it has been used as an astringent in topical preparations. While argyria occurred more commonly before the development of antibiotics, it is now a rare occurrence (USEPA, 1996c).

D. Carcinogenicity

USEPA does not classify silver as a suspect or probable human carcinogen (Group D) due to inadequate or no evidence of carcinogenicity in animal studies. No evidence of cancer in humans has been reported despite frequent therapeutic use of the compound over the years. In animals, local sarcomas have been induced after implantation of foils and discs of silver. However, the interpretation of these findings has been questioned due to the phenomenon of solid-state carcinogenesis in which even insoluble solids such as plastic have been shown to result in local fibrosarcomas (USEPA, 1996c).

E. Mutagenicity

No evidence of the mutagenicity of silver was shown in two available studies. One study evaluated silver nitrate for the possible induction of back-mutations from streptomycin dependence to nondependence in Eschericha coli. Silver nitrate was considered nonmutagenic in this assay.

The other study screened silver chloride with other chemicals for mutagenic effects using a method called the rec-assay. Silver chloride was considered nonmutagenic in this assay (USEPA, 1996c).

F. Teratogenicity/Reproductive Effects

Limited information on the reproductive and teratogenic effects of silver on animals is available. It is reported that this compound orally administered to rats induce adverse reproductive effects. Animals injected with silver demonstrated an effect on testicular tissue and sperm morphology. Neonatal rats exposed to silver in drinking water had reduced volume of certain brain regions (ATSDR, 1990b).

G. Other Health Effects

Abdominal pain has been reported by workers exposed to silver compounds (silver nitrate and silver oxide) after inhalation exposure. The pain was described by the workers as "burning in quality and relieved by antacids". Exposure levels were estimated to be between 0.039 and 0.378 mg silver/m^3.

H. Epidemiological Evidence

Several cases concerning individuals ingesting metallic and other silver compounds has been reported. These cases all reported argyria, discoloration of the skin (ATSDR, 1990b; USEPA, 1996c).

I. Toxicity Data

USEPA has not classified silver as to human carcinogenicity, due to inadequate evidence of carcinogenicity in studies. A chronic oral reference dose is reported to be 0.005 mg/kg-day for silver. The aforementioned toxicity value was taken from USEPA, 2004a.

4.18 THALLIUM

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	204.38
Melting Point:	303.5°C
Boiling Point:	1,457°C
Specific Density:	11.85 (@ 20°C)
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	10 (@ 1000°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 1992f. Toxicological Profile for Thallium.

B. Chemical Transformation

Air: Thallium may exist as an oxide, hydroxide, sulfate or as a sulfide. Metallic thallium oxidizes slowly in air. Thallium released to the atmosphere is deposited onto soil or water surface by wet and dry deposition (ATSDR, 1992f).

Water: In water, thallium exists primarily as a monovalent ion, however it may be present as a trivalent thallium in very oxidizing waters. Thallium ion forms complexes in solution with halogens, oxygen and sulfur (ATSDR, 1992f).

Soil: Thallium may partition from water to soils (ATSDR, 1992f).

2. Toxicological Properties

A. Metabolism

No information was found on the metabolism of thallium. Thallium is readily absorbed and rapidly is distributed throughout the body, especially the kidneys and liver. Removal of thallium from the body is slow. Most of the thallium leaves your body in urine and to a lesser extent in feces (ATSDR, 1992f).

B. Acute Toxicity

Mortality data of exposed humans and results of studies in animals suggest that thallium may cause death at high concentrations. Neurological damage was a consistent feature among humans; however, death was regularly attributed to cardiac or respiratory failure. The estimated lethal dose for the average adult is 14 to 15 mg/kg (1 g) (ATSDR, 1992f).

Acute oral exposure to thallium may develop respiratory effects including alveolar damage, hyaline membrane formation and pulmonary edema. Humans exposed to this compound orally demonstrated cardiovascular effects including myocardial damage and electrocardiographic changes. The liver may also be effected by thallium toxicity. Necrosis, fatty changes and altered serum enzyme levels were reported in humans (ATSDR, 1992f).

C. Subacute and Chronic Toxicity

In non-fatal occupational cases of moderate or long term exposure, early symptoms of thallium usually include fatigue, limb pain, metallic taste in the mouth and loss of hair. Later, peripheral neuritis, proteinuria and joint pains occur (ATSDR, 1992f).

D. Carcinogenicity

No studies were found on the carcinogenicity of thallium. USEPA has not classified thallium as to its carcinogenicity.

E. Mutagenicity

Animal *in vitro* and *in vivo* studies were positive for DNA damage (ATSDR, 1992f).

F. Teratogenicity/Reproductive Effects

Subchronic oral studies on rats reported that the testes may be susceptible. In these animals studies, it was reported that thallium may cross the placenta and act as a developmental and neurological toxicant (ATSDR, 1992f).

G. Other Health Effects

There is some evidence that thallium may effect the kidneys. Tubular necrosis has been reported in some cases following ingestion. Animal studies have reported accumulation of debris in the lumen of the convoluted tubules and progressive changes in the mitochrondia of the tubule cell after parenteral exposure to thallium (ATSDR, 1992f).

H. Epidemiological Evidence

Human case reports demonstrated that thallium affects the central and peripheral nervous systems. Ataxia, tremor, multiple cranial palsies, numbness of toes and fingers, "burning feet" sensation and muscle cramps are the symptoms reported in these case studies. In one case, death of an individual was reported following exposure to an estimated single thallium oral dose of between 54 to 110 mg/kg (ATSDR, 1992f).

I. Toxicity Data

USEPA has not classified thallium as to its carcinogenicity. A chronic oral reference dose is reported to be 7E-05 mg/kg-day for thallium. The aforementioned toxicity value was taken from USEPA, 2004b.

4.19 VANADIUM

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	50.94
Melting Point:	1,917°C
Boiling Point:	3,380°C
Specific Density:	6.11 (@ 20°C)
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	no data
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 1992g. Toxicological Profile for Vanadium.

B. Chemical Transformation

Transformation occurs primarily between the various inorganic compounds during its movement through the environment i.e., in water, the trivalent state of vanadium is converted to the pentavalent state (ATSDR, 1992g).

2. Toxicological Properties

A. Metabolism

Because vanadium is a metal, it is not expected to be metabolized. However, there is an interconversion of 2 oxidation states of vanadium, vanadyl (V+4) and vanadate (V+5). Vanadate is considered more potent than vanadyl since it is reactive with several enzymes and is a potent inhibitor. Vanadyl leaves the body quicker than vanadate (ATSDR, 1992g).

B. Acute Toxicity

The available information on occupationally exposed individuals identify the respiratory system as the target organ following acute inhalation exposure. Symptoms include mild respiratory distress, coughing, wheezing, chest pains, runny nose, and sore throat. Once the exposure was stopped, the symptoms disappeared (ATSDR, 1992g).

C. Subacute and Chronic Toxicity

Workers chronically exposed to vanadium dusts in factories had slight to moderate eye irritations in addition to respiratory distress. The other significant finding in some workers was a green discoloration of the tongue attributed to direct deposition of vanadium (ATSDR, 1992g).

D. Carcinogenicity

There are no studies reporting vanadium as a cancer causing agent. Workers exposed to vanadium dusts did not show any increase in cancer deaths. Animals exposed to vanadium orally or by inhalation did not have any increases in tumors (ATSDR, 1992g).

E. Mutagenicity

In vitro studies reported positive effects for gene mutations and DNA synthesis using bacteria, yeast and mouse cells in culture. Human leukocytes have been reported to show DNA strand breaks from exposure to vanadate (ATSDR, 1992g).

F. Teratogenicity/Reproductive Effects

There is one well-conducted study on rats exposed to vanadium. No adverse effects were noted on the reproductive system. Other animals studies suggest that vanadium may be toxic to offspring. The effects included, increases in visible hemorrhages, altered lung collagen

metabolism, decrease in fetal length and weight, and skeletal abnormalities (ATSDR, 1992g).

G. Other Health Effects

Volunteers given vanadium in capsules as ammonia vanadyl tartrate experienced abdominal cramping and diarrhea. Workers exposed to vanadium dusts via ingestion reported nausea and vomiting (ATSDR, 1992g).

H. Epidemiological Evidence

There are several studies reported on workers who have inhaled vanadium, that support the information provided above.

I. Toxicity Data

Vanadium has not been classified by USEPA as to its carcinogenicity. The chronic oral RfD for vanadium is 0.001 mg/kg-day. The aforementioned toxicity value was taken from USEPA, 2004b.

4.20 ZINC

1. Constituent Properties

A. Physical and Chemical Properties

Atomic Weight (g/mol):	65.38
Melting Point:	419.5°C
Boiling Point:	908°C
Specific Density:	7.14 (@ 25°C)
Water Solubility (mg/L):	insoluble
Vapor Pressure (mm Hg):	1 (@ 487°C)
Henry's Law Constant (atm-m^3/mol):	no data

Reference: ATSDR, 2003c. Toxicological Profile for Zinc.

B. Chemical Transformation

Air: In the atmosphere, zinc exists primarily in an oxidized form bound to aerosols. The predominant fate of airborne zinc is by dry or wet deposition (ATSDR, 2003c).

Water: In the aquatic environment, zinc partitions to sediments and suspended solids in waters through sorption onto hydrous iron and manganese oxides, clay minerals and organic material. A small fraction of zinc will exist in the aquatic phase as soluble inorganic zinc compounds. Soluble inorganic zinc compounds hydrolyze in solution, forming zinc hydroxide precipitates (ATSDR, 2003c).

Soil: In soils, zinc will typically not dissolve and therefore, strongly bounds to soil particulates (ATSDR, 2003c).

2. Toxicological Properties

A. Metabolism

Plasma provides a metabolically active transport compartment for zinc. Zinc is most often complexed to organic ligands rather than free in solution as metallic ion. Zinc is found in diffusible or non-diffusible forms in the blood. In the diffusible form, zinc may bind to albumin and amino acids where zinc can be transported across tissue membranes to bind to proteins. In the non-diffusible form, zinc is tightly bound to alpha2-macroglobulin present in the liver (ATSDR, 2003c).

B. Acute Toxicity

Acute inhalation exposure to zinc oxide, and to a lesser extent zinc metal and other zinc compounds will result in metal fume fever. Metal fume fever displays symptoms such as chest pains, cough, dyspnea, reduced lung volumes, nausea, chills, malaise, and leukocytosis. Symptoms generally appear within a few hours and are reversible 1 to 4 days following cessation of exposure. Gastrointestinal effects may result after oral exposure to zinc. The symptoms include nausea, vomiting, abdominal cramps and diarrhea. Levels resulting in these effects generally range from 2 to 8 mg zinc/kg/day. In most cases, dermal exposure to zinc doesn't result in noticeable effects, however, zinc chloride or zinc salts can result in severe skin irritancy. This is characterized by parakeratosis, hyperkeratosis, and inflammatory changes in the epidermis and superficial dermis (ATSDR, 2003c).

C. Subacute and Chronic Toxicity

Anemia has been reported in individuals following ingestion of high doses of zinc supplements. Animal studies support the finding of anemia and also report renal and pancreas toxicity effects. Chronic dermal exposure to zinc oxide dust has been reported to result in pustular lesions. No exposure-related effects on lung function were reported for a group of welders chronically exposed to zinc (ATSDR, 2003c).

D. Carcinogenicity

There are no reports on the possible carcinogenicity of zinc and compounds in humans. Case studies have been used to evaluate the effects of zinc administered for therapeutic reasons. There are reports which compare zinc levels in normal and cancerous tissue. Studies of occupational exposure to zinc compounds have also been conducted, but have limited value because they do not correlate exposure with cancer risk. USEPA has not classified zinc as to human carcinogenicity (Group D) due to inadequate evidence in humans and animals (USEPA, 1992).

E. Mutagenicity

There are sufficient *in vivo* data establishing the clastogenicity of zinc, however, data regarding mutagenicity of zinc are conflicting. Several *in vitro* microbial gene mutation assays were negative and evidence from gene mutation assays in mammalian cells were mixed. An increase in the occurrence of chromosomal aberrations was reported *in vitro* in human lymphocytes, and *in vivo* in rats and mice. Responses in mutagenicity assays are thought to depend on the form (e.g., inorganic or organic salt) of the zinc tested. For example, inorganic salts tend to dissociate and the zinc becomes bound with culture media constituents. Salts that dissociate less readily tend to be transported into the cell and are postulated to cause a positive response (ATSDR, 2003c; USEPA, 1992).

F. Teratogenicity/Reproductive Effects

Rats orally exposed to zinc showed dysfunction in the reproductive system and increased fetal resorptions. One case history reported an increase in stillbirths to women who received high doses of zinc supplements during their last trimester (ATSDR, 2003c).

G. Other Health Effects

Consuming too little zinc is at least as important a health problem as consuming too much zinc. Without enough zinc, humans may experience loss of appetite, decreased sense of taste and smell, decreased immune function, slow wound healing, and skin sores. Too little zinc may also cause poorly developed sex organs and retarded growth in young men. Infants may develop birth defects if the mother doesn't receive enough zinc during pregnancy (ATSDR, 2003c).

H. Epidemiological Evidence

There are a few human studies that have reported metal fume fever after inhalation exposure to zinc oxide. Oral studies have shown that zinc results in anemia, pancreatic damage and immunotoxicity (ATSDR, 2003c).

I. Toxicity Data

USEPA has not classified zinc as to human carcinogenicity, due to inadequate evidence of carcinogenicity in studies. A chronic oral reference dose is reported to be 0.3 mg/kg-day for zinc. The aforementioned toxicity value was taken from USEPA, 2004a.

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APPENDIX Q.5

IEBUK MODELLING OUTPUT

LEAD MODEL FOR WINDOWS Version 1.0

Model Version: 1.0 Build 261
User Name: Project # 3698
Date: 2/18/2005
Site Name: Hamptonburgh, New York
Operable Unit: Groundwater Only
Run Mode: Site Risk Assessment

Water Data
Site-specific groundwater lead concentration.

The time step used in this model run: 1 - Every 4 Hours (6 times a day).

****** Air ******

Indoor Air Pb Concentration: 30.000 percent of outdoor.
Other Air Parameters:

Age	Time Outdoors (hours)	Ventilation Rate (m^3/day)	Lung Absorption (%)	Outdoor Air Pb Conc (ug Pb/m^3)
.5-1	1.000	2.000	32.000	0.100
1-2	2.000	3.000	32.000	0.100
2-3	3.000	5.000	32.000	0.100
3-4	4.000	5.000	32.000	0.100
4-5	4.000	5.000	32.000	0.100
5-6	4.000	7.000	32.000	0.100
6-7	4.000	7.000	32.000	0.100

****** Diet ******

Age	Diet Intake(ug/day)
.5-1	5.530
1-2	5.780
2-3	6.490
3-4	6.240
4-5	6.010
5-6	6.340
6-7	7.000

****** Drinking Water ******

Water Consumption:

Age	Water (L/day)
.5-1	0.200
1-2	0.500
2-3	0.520
3-4	0.530
4-5	0.550
5-6	0.580
6-7	0.590

Drinking Water Concentration: 69.900 ug Pb/L

****** Soil & Dust ******

Multiple Source Analysis Used
Average multiple source concentration: 150.000 ug/g

Mass fraction of outdoor soil to indoor dust conversion factor: 0.700
Outdoor airborne lead to indoor household dust lead concentration: 100.000
Use alternate indoor dust Pb sources? No

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
.5-1	200.000	150.000
1-2	200.000	150.000
2-3	200.000	150.000
3-4	200.000	150.000
4-5	200.000	150.000
5-6	200.000	150.000
6-7	200.000	150.000

****** Alternate Intake ******

Age	Alternate (ug Pb/day)
.5-1	0.000
1-2	0.000
2-3	0.000
3-4	0.000
4-5	0.000
5-6	0.000
6-7	0.000

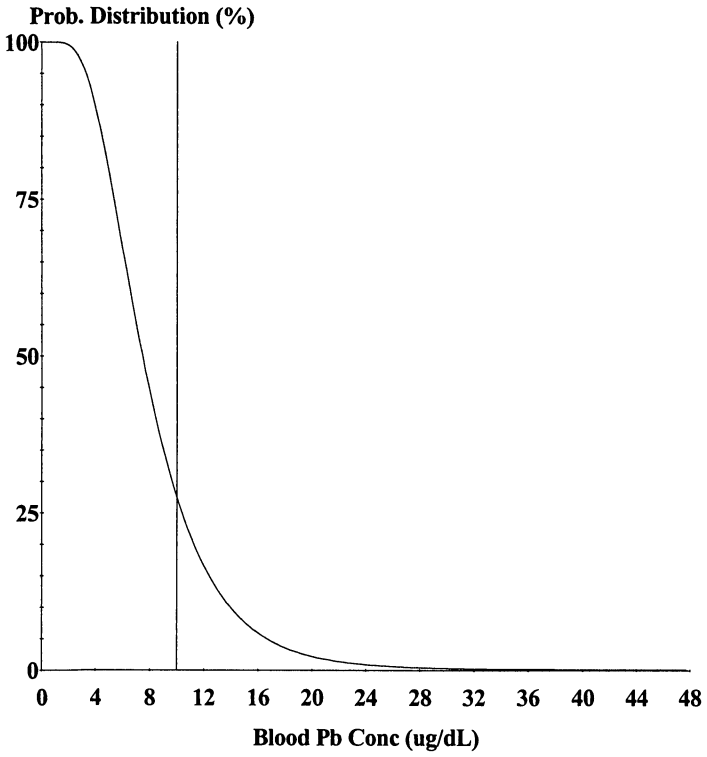
****** Maternal Contribution: Infant Model ******

Maternal Blood Concentration: 2.500 ug Pb/dL

****
CALCULATED BLOOD LEAD AND LEAD UPTAKES:
****

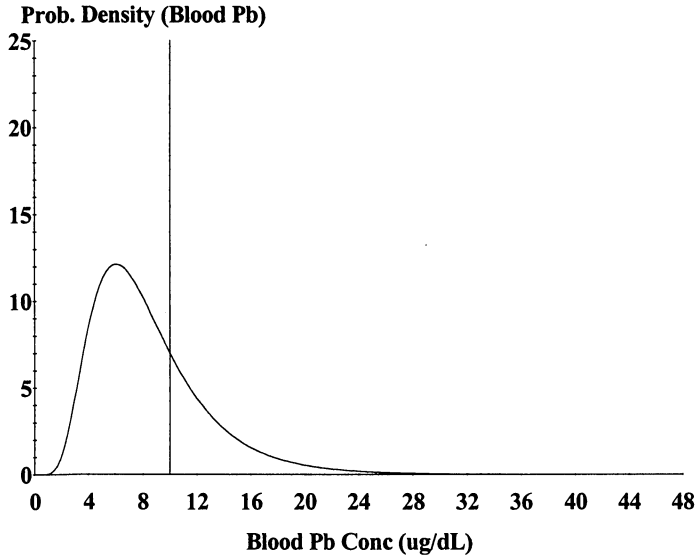
Year	Air (ug/day)	Diet (ug/day)	Alternate (ug/day)	Water (ug/day)
.5-1	0.021	2.399	0.000	6.064
1-2	0.034	2.363	0.000	14.290
2-3	0.062	2.708	0.000	15.169
3-4	0.067	2.662	0.000	15.807
4-5	0.067	2.629	0.000	16.816
5-6	0.093	2.804	0.000	17.928
6-7	0.093	3.119	0.000	18.379

Year	Soil+Dust (ug/day)	Total (ug/day)	Blood (ug/dL)
.5-1	3.816	12.301	6.6
1-2	5.713	22.400	8.9
2-3	5.831	23.770	8.7
3-4	5.962	24.498	8.4
4-5	4.527	24.039	8.0
5-6	4.119	24.944	7.6
6-7	3.920	25.512	7.1



Cutoff = 10.000 ug/dl
Geo Mean = 7.805
GSD = 1.600
% Above = 29.903

Age Range = 0 to 84 months
Time Step = Every 4 Hours
Run Mode = Site Risk Assessment



Cutoff = 10.000 ug/dl
Geo Mean = 7.805
GSD = 1.600
% Above = 29.903
% Below = 70.097

Age Range = 0 to 84 months
Time Step = Every 4 Hours
Run Mode = Site Risk Assessment

LEAD MODEL FOR WINDOWS Version 1.0

Model Version: 1.0 Build 261
User Name: Project # 3698
Date: 2/18/2005
Site Name: Hamptonburgh, New York
Operable Unit: Groundwater + Sediment
Run Mode: Site Risk Assessment

Water Data
Site-specific groundwater lead concentration.
GSD, Cutoff and Age Type
Default value.
GSD, Cutoff and Age Type
Default value.
GI Values + Bioavailability Data
Soil default value utilized for sediment.
Alternate Source Data
Site-specific sediment lead concentration.

The time step used in this model run: 1 - Every 4 Hours (6 times a day).

****** Air ******

Indoor Air Pb Concentration: 30.000 percent of outdoor.
Other Air Parameters:

Age	Time Outdoors (hours)	Ventilation Rate (m^3/day)	Lung Absorption (%)	Outdoor Air Pb Conc (ug Pb/m^3)
.5-1	1.000	2.000	32.000	0.100
1-2	2.000	3.000	32.000	0.100
2-3	3.000	5.000	32.000	0.100
3-4	4.000	5.000	32.000	0.100
4-5	4.000	5.000	32.000	0.100
5-6	4.000	7.000	32.000	0.100
6-7	4.000	7.000	32.000	0.100

****** Diet ******

Age	Diet Intake(ug/day)
.5-1	5.530
1-2	5.780
2-3	6.490
3-4	6.240
4-5	6.010
5-6	6.340
6-7	7.000

****** Drinking Water ******

Water Consumption:

Age	Water (L/day)
.5-1	0.200
1-2	0.500
2-3	0.520
3-4	0.530
4-5	0.550
5-6	0.580
6-7	0.590

Drinking Water Concentration: 69.900 ug Pb/L

****** Soil & Dust ******

Multiple Source Analysis Used
Average multiple source concentration: 150.000 ug/g

Mass fraction of outdoor soil to indoor dust conversion factor: 0.700
Outdoor airborne lead to indoor household dust lead concentration: 100.000
Use alternate indoor dust Pb sources? No

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
.5-1	200.000	150.000
1-2	200.000	150.000
2-3	200.000	150.000
3-4	200.000	150.000
4-5	200.000	150.000
5-6	200.000	150.000
6-7	200.000	150.000

****** Alternate Intake ******

Age	Alternate (ug Pb/day)
.5-1	1.276
1-2	1.276
2-3	1.276
3-4	1.276
4-5	1.276
5-6	1.276
6-7	1.276

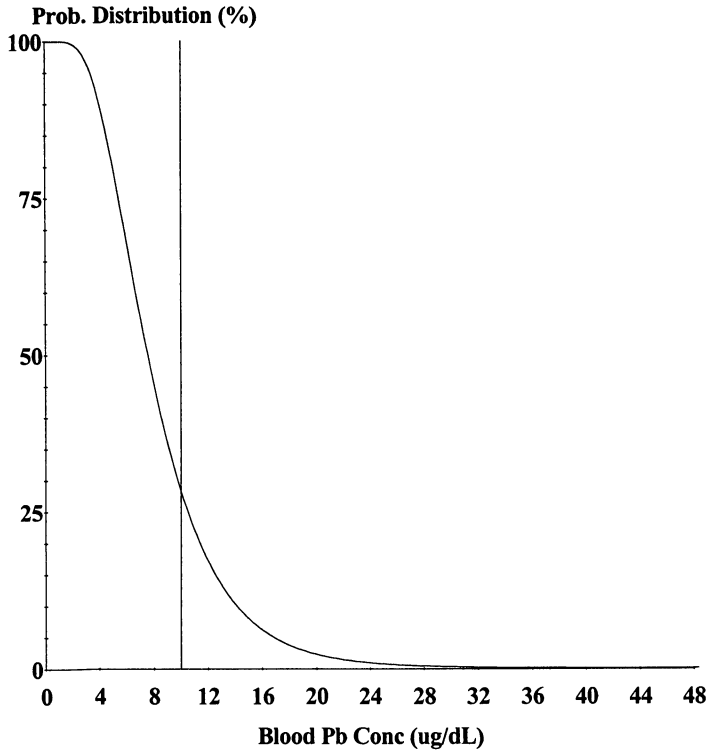
****** Maternal Contribution: Infant Model ******

Maternal Blood Concentration: 2.500 ug Pb/dL

****
CALCULATED BLOOD LEAD AND LEAD UPTAKES:
****

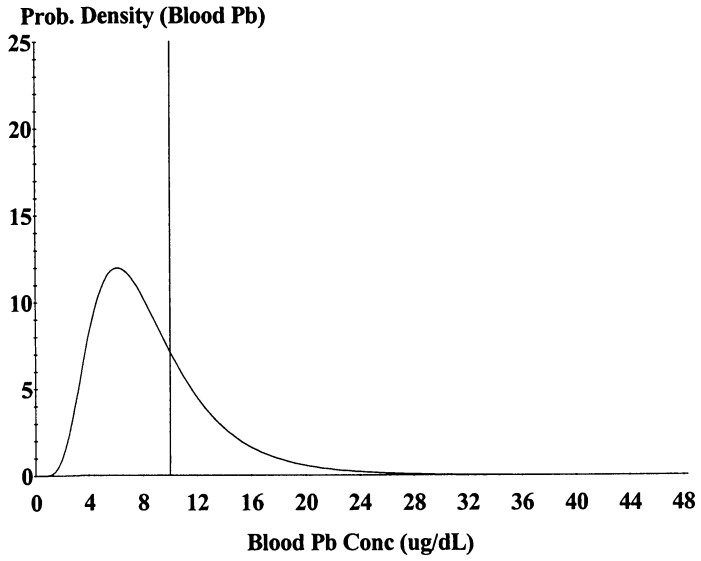
Year	Air (ug/day)	Diet (ug/day)	Alternate (ug/day)	Water (ug/day)
.5-1	0.021	2.391	0.331	6.044
1-2	0.034	2.358	0.312	14.256
2-3	0.062	2.703	0.319	15.137
3-4	0.067	2.657	0.326	15.777
4-5	0.067	2.624	0.334	16.788
5-6	0.093	2.799	0.338	17.901
6-7	0.093	3.115	0.341	18.353

Year	Soil+Dust (ug/day)	Total (ug/day)	Blood (ug/dL)
.5-1	3.803	12.590	6.7
1-2	5.699	22.659	9.0
2-3	5.819	24.039	8.8
3-4	5.951	24.778	8.5
4-5	4.520	24.333	8.0
5-6	4.113	25.245	7.7
6-7	3.915	25.817	7.2



Cutoff = 10.000 ug/dl
Geo Mean = 7.906
GSD = 1.600
% Above = 30.852

Age Range = 0 to 84 months
Time Step = Every 4 Hours
Run Mode = Site Risk Assessment



Cutoff = 10.000 ug/dl
Geo Mean = 7.906
GSD = 1.600
% Above = 30.852
% Below = 69.148

Age Range = 0 to 84 months
Time Step = Every 4 Hours
Run Mode = Site Risk Assessment

LEAD MODEL FOR WINDOWS Version 1.0

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Model Version: 1.0 Build 261
User Name: Project # 3698
Date: 2/18/2005
Site Name: Hamptonburgh, New York
Operable Unit: Sediment Only
Run Mode: Site Risk Assessment

Water Data
Site-specific groundwater lead concentration.
GSD, Cutoff and Age Type
Default value.
GSD, Cutoff and Age Type
Default value.
GI Values + Bioavailability Data
Soil default value utilized for sediment.
Alternate Source Data
Site-specific sediment lead concentration.
GSD, Cutoff and Age Type
Default value.
GSD, Cutoff and Age Type
Default value.

==========

The time step used in this model run: 1 - Every 4 Hours (6 times a day).

****** Air ******

Indoor Air Pb Concentration: 30.000 percent of outdoor.
Other Air Parameters:

Age	Time Outdoors (hours)	Ventilation Rate (m^3/day)	Lung Absorption (%)	Outdoor Air Pb Conc (ug Pb/m^3)
.5-1	1.000	2.000	32.000	0.100
1-2	2.000	3.000	32.000	0.100
2-3	3.000	5.000	32.000	0.100
3-4	4.000	5.000	32.000	0.100
4-5	4.000	5.000	32.000	0.100
5-6	4.000	7.000	32.000	0.100
6-7	4.000	7.000	32.000	0.100

****** Diet ******

Age	Diet Intake(ug/day)
.5-1	5.530
1-2	5.780
2-3	6.490
3-4	6.240
4-5	6.010
5-6	6.340
6-7	7.000

****** Drinking Water ******

Water Consumption:

Age	Water (L/day)
.5-1	0.200
1-2	0.500
2-3	0.520
3-4	0.530
4-5	0.550
5-6	0.580

6-7 0.590

Drinking Water Concentration: 4.000 ug Pb/L

****** Soil & Dust ******

Multiple Source Analysis Used
Average multiple source concentration: 150.000 ug/g

Mass fraction of outdoor soil to indoor dust conversion factor: 0.700
Outdoor airborne lead to indoor household dust lead concentration: 100.000
Use alternate indoor dust Pb sources? No

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
.5-1	200.000	150.000
1-2	200.000	150.000
2-3	200.000	150.000
3-4	200.000	150.000
4-5	200.000	150.000
5-6	200.000	150.000
6-7	200.000	150.000

****** Alternate Intake ******

Age	Alternate (ug Pb/day)
.5-1	1.276
1-2	1.276
2-3	1.276
3-4	1.276
4-5	1.276
5-6	1.276
6-7	1.276

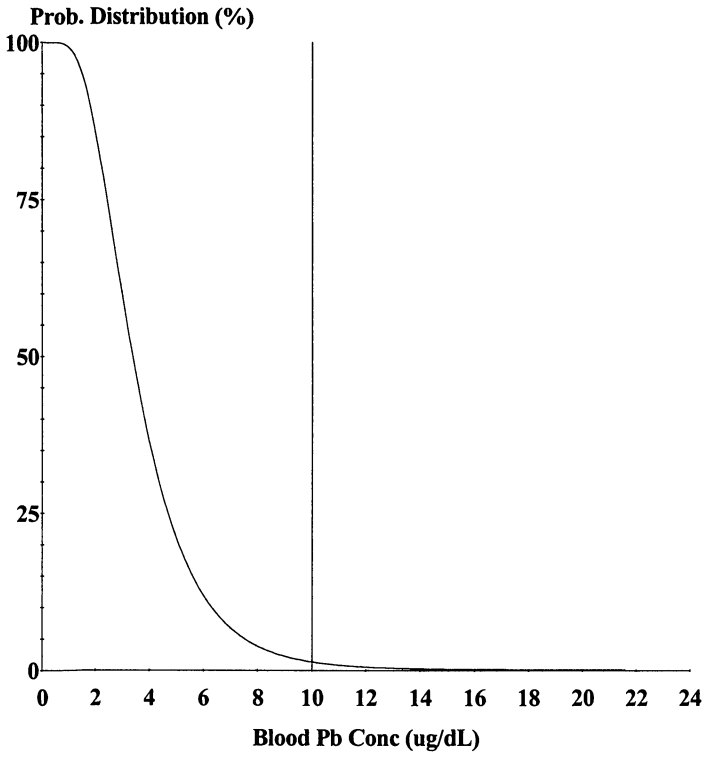
****** Maternal Contribution: Infant Model ******

Maternal Blood Concentration: 2.500 ug Pb/dL

****
CALCULATED BLOOD LEAD AND LEAD UPTAKES:
****

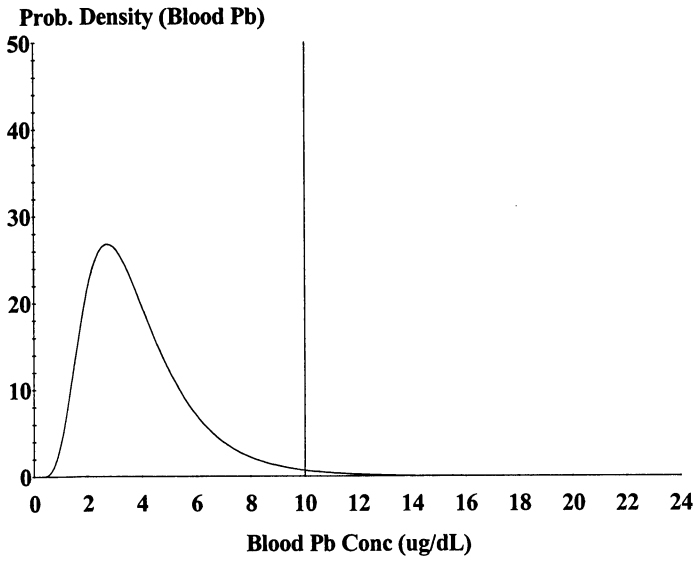
Year	Air (ug/day)	Diet (ug/day)	Alternate (ug/day)	Water (ug/day)
.5-1	0.021	2.543	0.352	0.368
1-2	0.034	2.640	0.350	0.913
2-3	0.062	2.994	0.353	0.960
3-4	0.067	2.913	0.357	0.990
4-5	0.067	2.857	0.364	1.046
5-6	0.093	3.035	0.366	1.111
6-7	0.093	3.361	0.368	1.133

Year	Soil+Dust (ug/day)	Total (ug/day)	Blood (ug/dL)
.5-1	4.046	7.330	4.0
1-2	6.381	10.318	4.3
2-3	6.446	10.815	4.0
3-4	6.522	10.848	3.8
4-5	4.921	9.255	3.2
5-6	4.459	9.064	2.8
6-7	4.224	9.179	2.6



Cutoff = 10.000 ug/dl
Geo Mean = 3.530
GSD = 1.600
% Above = 1.337

Age Range = 0 to 84 months
Time Step = Every 4 Hours
Run Mode = Site Risk Assessment



Cutoff = 10.000 ug/dl
Geo Mean = 3.530
GSD = 1.600
% Above = 1.337
% Below = 98.663

Age Range = 0 to 84 months
Time Step = Every 4 Hours
Run Mode = Site Risk Assessment