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Human Health Risk Assessment at IR Program Site 16 (Former Creosote Dip Tank and Fire Fighting Training Area)

Naval Construction Battalion Center North Kingston, Rhode Island

Contract No. N62472-92-D-1296
Delivery Order No. 0097

Prepared for

Department of the Navy
Engineering Field Activity Northeast
10 Industrial Highway, Mail Stop No. 82
Lester, Pennsylvania 19113-2090

Prepared by

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NCBC Davisville, North Kingstown, Rhode Island

EA CTO Manager: James A. Shultz, C.P.G.

In compliance with EA's Quality Procedures for review of deliverables outlined in the Quality Management Plan, this final deliverable has been reviewed for quality by the undersigned Senior Technical Reviewer(s). The information presented in this report/deliverable has been prepared in accordance with the approved Implementation Plan for the Contract Task Order (CTO) and reflects a proper presentation of the data and/or the conclusions drawn and/or the analyses or design completed during the conduct of the work. This statement is based upon the standards identified in the CTO and/or the standard of care existing at the time of preparation.

Senior Technical Reviewer

Eileen Mahoney, Ph.D. (Date)
Senior Risk Assessor

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ACRONYMS AND ABBREVIATIONS

bgs	Below ground surface
bw	Body weight
CADI	Carcinogenic Average Daily Intake
CDC	Centers for Disease Control
CFR	Code of Federal Regulations
COPC	Constituents of Potential Concern
CTE	Central Tendency Exposure
dL	Deciliter(s)
EA	EA Engineering, Science, and Technology
EBS	Environmental Baseline Survey
EFANE	Engineering Field Activity Northeast
EPC	Exposure point concentration
ft	Foot/Feet
HEAST	Health Effects Assessment Summary Tables
HHRA	Human Health Risk Assessment
HI	Hazard Index
HQ	Hazard Quotient
Hr	Hour(s)
IEUBK	Integrated Exposure Uptake Biokinetic Model
IR	Installation Restoration
IRIS	Integrated Risk Information System
kg	Kilogram(s)
L	Liter(s)
LEAD99D	LEAD Version 0 99D
mg	Milligram(s)
NCADI	Non-Carcinogenic Average Daily Intake
NCBC	Naval Construction Battalion Center
NCEA	National Center for Environmental Assessment
NCP	National Oil and Hazardous Substances Pollution Contingency Plan
NOAEL	No-observed-adverse-effect-level
NRC	National Research Council

OAQPS	Office of Air Quality Planning and Standards
OSWER	Office of Solid Waste and Emergency Response
OU	Operable unit
PAH	Polycyclic Aromatic Hydrocarbons
ppm	Parts per million
PRG	Preliminary Remediation Goals
RAGS	Risk Assessment Guidance for Superfund
Rfc	Reference concentration
Rfd	Reference dose
RI	Remedial Investigation
RME	Reasonable Maximum Exposure
SF	Slope factor
Site 16	Program Site 16
SQL	Sample Quantitation Level
TCDD-TEQ	Dioxin-toxic equivalent concentration
TCE	Trichloroethene
TRW	Technical Review Workgroup for Lead
UCLM	Upper Confidence Limit of the Mean
µG	Microgram(s)
U.S. EPA	U.S. Environmental Protection Agency
VOC	Volatile organic compound(s)

EXECUTIVE SUMMARY

EA Engineering, Science, and Technology has been retained by the U.S. Navy Engineering Field Activity Northeast (EFANE) to provide risk assessment services for the former Naval Construction Battalion Center (NCBC) Davisville facility in North Kingstown, Rhode Island. This report presents the human health risk assessment (HHRA) for the Phase I Remedial Investigation (RI) for Installation Restoration (IR) Program Site 16 (Site 16).

The former NCBC Davisville facility is located in the Town of North Kingstown, Rhode Island, approximately 18 miles south of the state capital, Providence. NCBC Davisville is composed of three areas: the Main Center (Zones 1 through 4), the West Davisville storage area, and Camp Fogarty. NCBC Davisville was primarily used for training Naval seamen in construction operations, as well as for storage and freight yards for construction materials. Site 16 is the location of a former creosote dip tank area, where, during the 1960s, wood piles were dipped into tanks containing creosote and staged in the area to dry (Figure ES-1). In addition, a former fire fighter training area is reportedly located in the site. Currently the site is not used and consists of the remnants of these former activities. The site is primarily vegetated with the exception of a paved area in the central portion of the site.

The HHRA for Site 16 is conducted in accordance with U.S. Environmental Protection Agency (EPA) *Risk Assessment Guidance for Superfund (RAGS), Volume I: Human Health Evaluation Manual (Part A) (Interim Final)* (U.S. EPA 1989a) and U.S. EPA RAGS, *Volume 1 – Human Health Evaluation Manual (Part D), Standardized Planning, Reporting and Review of Superfund Risk Assessments Interim* (U.S. EPA 1998a). The risk assessment follows the methodologies presented in the *Final Work Plan, Remedial Investigation of IR Program Site 16 (Former Creosote Dip Tank and Fire Fighting Area), Naval Construction Battalion Center, North Kingstown, RI* (EA 2000) and comments provided by EPA Region I (U.S. EPA 2000).

A human health site conceptual model is developed and provided in Figure ES-2 and Table ES-1. Media of concern include surface soil, total soil (combined surface and subsurface soil), seep water, and ground water. Based on the shallow and intermittent nature of the seeps at Site 16, sediment is not addressed as a medium of concern, and, instead, seep water is evaluated. Seep water samples are not filtered, and thus their evaluation is representative of both seep water and sediment exposure. The site conceptual model also depicts the receptors and exposure pathways evaluated in this HHRA.

A risk-based screening assessment (hazard identification) is conducted to eliminate chemicals that were not detected at levels capable of producing unacceptable risk. Risk-based screening is conducted by comparing maximum detected chemical concentrations for each medium to risk-based screening concentrations. Any analyte in any medium for which the maximum measured concentration exceeded the risk-based screening concentration is retained as a constituent of potential concern (COPC) and is assessed further in the HHRA. The EPA Region IX soil preliminary remediation goals (PRGs) (U.S. EPA 2001a) are used for screening purposes.

One-tenth of the PRG for non-carcinogens is used to account for potential additivity of toxic effects.

An exposure assessment was conducted to calculate Exposure Point Concentrations (EPCs) representative for each COPC. The exposure assessment utilized exposure parameters presented in the *Final Work Plan, Remedial Investigation of IR Program Site 16 (Former Creosote Dip Tank and Fire Fighting Area), Naval Construction Battalion Center, North Kingstown, RI* (EA 2000) and comments provided by U.S. EPA Region I (U.S. EPA 2000). Reasonable Maximum Exposure (RME) and Central Tendency Exposure (CTE) scenarios were evaluated.

The toxicity assessment and risk characterization section focused on non-carcinogenic COPCs, carcinogenic COPCs, and lead. Risks were evaluated for each receptor on a cumulative basis across all pathways and media. A breakdown for each medium was also provided in Tables 10.1 through 10.5.

Non-Carcinogenic Risks

Non-carcinogenic risks exceed the U.S. EPA threshold of 1.0 for residents (adult and child) and construction workers; non-carcinogenic risks do not exceed 1.0 for recreational users (adult and child) and commercial workers. The risks exceeding the threshold are driven entirely by the ground-water exposure pathways.

For RME and CTE, cumulative Hazard Indexes (His) are calculated as less than the 1.0 threshold for surface soil (commercial workers, adult recreational user, and child recreational user) and total soil (construction workers, child residents, and adult residents). For RME and CTE, cumulative HIs are also less than the 1.0 threshold for seep water (adult recreational user, child recreational user) exposure. Thus, non-carcinogenic risk for all receptors to soil and seep water is acceptable.

For the RME, cumulative HIs for ground water (construction workers, child residents, and adult residents) are calculated above the 1.0 threshold. The ground-water CTE HI for the construction worker is less than 1.0. COPCs with hazard quotients (HQs) above 1.0 in ground water are manganese and trichloroethene. A breakdown by target organ indicates that the HQs for the central nervous system, liver, and kidney exceed the 1.0 threshold indicating unacceptable non-carcinogenic risks for residential and construction worker exposure to ground water.

Carcinogenic Risks

Cumulative cancer risks for residential receptors exceeded U.S. EPA's "acceptable risk range" of 10^{-6} to 10^{-4} for the RME scenario only, driven by the ground-water exposure pathways. Combined adult and child resident risks for ground water are 2.1×10^{-3} and 6.1×10^{-5} for RME and CTE, respectively. Cumulative cancer risks for total soil are within U.S. EPA's "acceptable risk range" of 10^{-6} to 10^{-4}. The combined adult and child resident risks for total soil are 3.8×10^{-5} and 2.0×10^{-6} for RME and CTE, respectively. The only residential COPCs with risks greater than

10^{-5} are trichloroethene and vinyl chloride in ground water. Therefore, there are no concerns for cancer effects for residential exposure to total soil.

There are no cancer risks that exceeded U.S. EPA's "acceptable risk range" of 10^{-6} to 10^{-4} for recreational users. The only COPCs with risks greater than 10^{-6} in surface soil are dioxin, arsenic, benzo(a)pyrene, and dibenz(a,h)anthracene. RME cumulative risks for the adult and child recreational users are 6.4×10^{-6} and 7.4×10^{-6}, respectively. Therefore, there are no concerns for cancer effects for recreational users.

There are no cancer risks that exceeded U.S. EPA's "acceptable risk range" of 10^{-6} to 10^{-4} for industrial receptors. Both the construction and commercial workers had cumulative risks exceeding 10^{-6}. Risks are calculated as 7.3×10^{-6} for RME and 2.7×10^{-7} for CTE for construction workers. Risks are calculated as 6.2×10^{-6} for RME and 4.3×10^{-7} for CTE for commercial workers. In both cases, cumulative risks are below 10^{-5}. Therefore, there are no concerns for cancer effects for construction or commercial workers.

Lead Risks

In addition, lead is evaluated for total soil, ground water, and seep water. Blood lead levels are the indicator of excess lead exposure in humans. Modeled blood level results are compared to the established cutoff value or acceptable threshold of 10 micrograms (µg) lead/dL (deciliter). This blood level is considered protective of human health for children. Lead modeling is performed for residential, recreational, and worker scenarios. Blood lead level modeling results are below 10 µg lead/dL blood for the child resident scenario, indicating that there is no concern for adverse effects from site exposure to the detected lead in soil and ground water. This scenario provides the most conservative estimate of risks for lead and is, therefore, protective of recreational and worker exposure scenarios. However, although a recreational user model is not available, the residential model is run to estimate recreational risks as a conservative measure. The potential risks from recreational exposure are modeled using conservative residential assumptions for soil and conservative recreational exposure assumptions for seep water, indicating no unacceptable risks. Likewise the construction worker scenario is evaluated for exposure to total lead in soil; the results indicate no unacceptable risks.

Conclusions

Non-carcinogenic risks, carcinogenic risks, and potential lead exposure risks were all evaluated. Potential risks associated with ground-water exposure for the residential (adult and child) scenario and the construction worker scenario were unacceptable for non-carcinogenic risk and were unacceptable for the residential scenario for carcinogenic risk. These risks were driven by manganese and trichloroethene in ground water.

Potential risks for all other evaluated media for all evaluated receptors were acceptable. Therefore, there are no concerns for human exposure to site soil and seep water. Blood-lead level modeling indicated that exposure to lead is not of concern at the site for any of the evaluated receptors in any of the evaluated media.

1. INTRODUCTION

EA Engineering, Science, and Technology has been retained by the U.S. Navy Engineering Field Activity Northeast (EFANE) to provide risk assessment services for the former Naval Construction Battalion Center (NCBC) Davisville facility in North Kingstown, Rhode Island. This appendix presents the human health risk assessment (HHRA) for the Phase I Remedial Investigation (RI) for Installation Restoration (IR) Program Site 16 (Site 16).

1.1 FACILITY HISTORY

The former NCBC Davisville facility is located in the Town of North Kingstown, Rhode Island, approximately 18 miles south of the state capital, Providence. NCBC Davisville is composed of three areas: the Main Center (Zones 1 through 4), the West Davisville storage area, and Camp Fogarty. NCBC Davisville was primarily used for training Naval seamen in construction operations, as well as for storage and freight yards for construction materials. NCBC Davisville was comprised primarily of warehouse space and freight yards, most of which are currently empty. The Navy acquired the property in 1939. NCBC Davisville closed on 1 April 1994. Site 16 (formerly designated Environmental Baseline Study [EBS] Review Item 28 and then Study Area 16) is located in the eastern portion of Zone 3 at NCBC Davisville. A detailed description of the Base history can be found in the Final Basewide EBS (EA 1995).

1.2 SITE 16

Site 16 is the location of a former creosote dip tank area, where, during the 1960s, wood piles were dipped into tanks containing creosote and staged in the area to dry (Figure ES-1). In addition, a former fire fighter training area is reportedly located in the site. In such an area, temporary structures would have been doused with flammable materials, ignited, and fire fighters would have had to put these fires out. Such an operation could have released chemicals to the soil and ground water beneath Site 16. Currently the land is not used and consists of the remnants of these former activities. The site is primarily vegetated with the exception of a paved area in the central portion of the site. An unnamed asphalt road circles the outer perimeter of the wooded area of Site 16. The surrounding areas have been allowed to grow and presently have a very dense cover of bushes and small trees. The soil and seep samples and about 40 percent of the ground-water samples were collected from the approximately 9-acre area bordered to the south by Davisville Road, to the west by Westcott Road, to the north by Allen Harbor, and to the east by Allens Harbor Road (Figure ES-1). The remainder of the ground-water samples were collected from monitoring wells located in adjacent areas west, south, and east as the RI area expanded.

2. HUMAN HEALTH RISK ASSESSMENT FOR SITE 16

The purpose of this HHRA is to determine if, under expected exposure conditions, chemicals detected in environmental media at Site 16 are at concentrations that may cause unacceptable risk to humans using the area.

The HHRA for Site 16 is conducted in accordance with U.S. Environmental Protection Agency (U.S. EPA) *Risk Assessment Guidance for Superfund (RAGS), Volume I: Human Health Evaluation Manual (Part A) (Interim Final)* (U.S. EPA 1989a) and U.S. EPA RAGS, *Volume 1 – Human Health Evaluation Manual (Part D), Standardized Planning, Reporting and Review of Superfund Risk Assessments Interim* (U.S. EPA 1998a). The risk assessment follows the methodologies presented in the *Final Work Plan, Remedial Investigation of IR Program Site 16 (Former Creosote Dip Tank and Fire Fighting Area), Naval Construction Battalion Center, North Kingstown, RI* (EA 2000) and comments provided by U.S. EPA Region I (U.S. EPA 2000).

The risk assessment methodology used in this HHRA involves a four-step process: hazard identification, exposure assessment, toxicity assessment, and risk characterization. A brief description of each step is provided below:

In the *hazard identification*, environmental monitoring data are evaluated, constituents of potential concern (COPCs) are selected for inclusion throughout the remainder of the risk assessment, and the rationale for their selection is documented.

In the *exposure assessment*, the human population, or groups of individuals potentially exposed to COPCs (i.e., potential human receptors) are characterized. From the many potential pathways of exposure, pathways applicable to potential receptors at the site are identified. The concentrations of COPCs in relevant media (e.g., soil, air) are converted into systemic doses, taking into account rates of contact (e.g., ingestion rates) and absorption rates of different COPCs. The magnitude, frequency, and duration of these exposures are then integrated to obtain estimates of daily doses over a specified period of time (e.g., lifetime, activity-specific duration).

In the *toxicity assessment*, the relationship between extent of exposure and extent of toxic injury or disease is estimated for each COPC. Chemical-specific toxicity values, such as cancer slope factors (SFs) and reference doses (RfDs) or reference concentrations (RfCs) for non-carcinogens are presented along with a discussion of their scientific basis and derivation. The toxicity assessment includes toxicological profiles for each COPC, which are provided in Attachment A. *Risk characterization* integrates the results of the toxicity assessment and the exposure assessment to derive quantitative estimates of human health risk, including both the risks of cancer and of non-carcinogenic effects. The major uncertainties and limitations associated with the estimates of risk and their potential ramifications are presented in this section.

2.1 HAZARD IDENTIFICATION

A hazard identification is conducted to determine which constituents are of potential concern at the site. In the hazard identification, site-specific data are analyzed and compared to risk-based screening values. Although the current and expected future use of Site 16 is industrial in nature, screening is presented for both residential and industrial exposure-based scenarios.

2.1.1 Identification of Potential Exposure Pathways

An evaluation of potential exposure pathways of concern at Site 16 is undertaken based on planned future use. The results of this analysis are provided in Table ES-1.

2.1.2 Media of Concern

Media of concern include surface soil, total soil (combined surface and subsurface soil), seep water, and ground water. Based on the shallow and intermittent nature of the seeps at Site 16, sediment is not addressed as a medium of concern, and, instead, seep water is evaluated. Seep water samples are not filtered, and thus their evaluation is representative of both seep water and sediment exposure.

2.1.3 Exposure Pathways and Receptors of Concern

An exposure pathway describes a mechanism by which a population or individual may be exposed to chemicals present at a site. A completed exposure pathway requires the following four components:

(1) A source and mechanism of chemical release to the environment
(2) An environmental transport medium for the released chemical
(3) A point of potential human contact with the contaminated medium
(4) A human exposure route at the point of exposure.

All four components must exist for an exposure pathway to be complete and for exposure to occur. Incomplete exposure pathways do not result in actual human exposure and are not included in the exposure assessment and resulting risk characterization.

The future use of this site is planned to remain commercial/industrial in nature. However, a residential scenario, for both adult and child future residents, is included, as a conservative measure. Recreational use of this area is possible, thus both adult and child recreational users are assessed.

Under the commercial/industrial scenario, potential receptors are construction workers and commercial workers.

Under the future residential scenario, potential exposure to soil and ground water will be characterized. Total soil (both surface and subsurface soil) is evaluated as a conservative

measure to account for mixing of soil that may occur during redevelopment of the property. Incidental ingestion of, dermal contact with, and inhalation of particulates from total soil, as well as ingestion of and dermal contact with ground water and adult inhalation of volatiles while showering is assessed. The residential scenario includes both an adult and a child receptor. To evaluate lifetime cancer effects, the adult and child cancer risks are combined for each medium to reflect potential risks.

Recreational use of the area is possible, and thus incidental ingestion of and dermal contact with surface soil (0 to 2 ft below ground surface [bgs]) while playing and/or picnicking is assessed. In addition, although it is unlikely based on the depth of the seeps, incidental ingestion of and dermal contact with seep water during wading is also assessed as a conservative measure. The recreational scenario includes both an adult and a child receptor. They are evaluated as independent receptors.

Future construction workers are evaluated for incidental ingestion of, dermal contact with, and inhalation of particulates from total soil during excavation activities. It is assumed that construction workers would contact both surface and subsurface soils; therefore, exposure to total soil is evaluated. Construction workers are also evaluated for incidental ingestion of and dermal contact with ground water during excavation activities as well as inhalation of volatiles released from ground water.

Current and future commercial workers are evaluated for incidental ingestion of, dermal contact with, and inhalation of particulates from surface soil (0 to 2 ft bgs) while working. Commercial workers typically are not involved in digging scenarios where exposure to subsurface soil would occur. In the unlikely event that these exposures occur, the evaluation of the construction worker scenarios provides a conservative estimate of potential risks to site soils.

2.1.4 Risk-Based Screening

Risk-based screening is conducted by comparing maximum detected chemical concentrations for each medium to risk-based screening concentrations. Any analyte in any medium for which the maximum measured concentration exceeded the risk-based screening concentration is retained as a COPC.

Risk-based screening concentrations that are used in the selection of COPCs are medium-specific and are discussed below. For both surface and total soils, the U.S. EPA Region IX soil preliminary remediation goals (PRGs) (U.S. EPA 2001a) are used for screening purposes. For seep water and ground water, the U.S. EPA Region IX tap water PRGs are used for screening purposes. One-tenth of the PRG for non-carcinogens is used to account for potential additivity of toxic effects.

It should be noted that the use of tap water PRGs for seep water screening is highly conservative given that seep water exposure could not be as great as the tap water ingestion scenario upon which the PRGs are based. Likewise, it is unlikely that ground water at Site 16 will be used as a water supply/tap water due to the availability of supplied water.

Region IX soil PRGs are available for both industrial and residential scenarios. Both sets of PRGs are employed in the screening analysis to ensure that COPCs are consistent with and specific to the exposure scenarios and receptors identified at the site. Residential receptors and recreational users, child and adult, are evaluated for COPCs determined by comparison to residential soil PRGs. Industrial receptors, construction worker and commercial worker, are evaluated for COPCs determined by comparison to industrial PRGs.

An analyte is eliminated from the list of COPCs if it is an essential nutrient of low toxicity, and its reported maximum concentration is unlikely to be associated with adverse health impacts. COPCs excluded from further consideration on this basis are calcium, iron, magnesium, potassium, and sodium.

Common laboratory contaminants that were detected above their screening limits were included as COPCs as a conservative measure. Discussion of the uncertainty associated with their inclusion as COPCs is provided in the uncertainty analysis (Subsection 2.6.1).

Background data are available for ground water at the Base and are developed in the *Basewide Groundwater Inorganics Study Report, NCBC Davisville, RI* (Stone & Webster 1996). This report focused on the basewise ground-water operable unit (OU) and was conducted to establish a single set of background values for inorganics in ground water at the Main Center and West Davisville.

A comparison of the maximum detected site ground-water concentrations to these established inorganic background concentrations per U.S. Navy policy (U.S. Navy 2000) showed that arsenic detected at Site 16 was below background. In addition, arsenic in Site 16 ground-water was detected in only 2 of 42 samples, which is less than a 5 percent detection frequency. Federal U.S. EPA guidance indicates that chemicals detected at less than 5 percent detection frequency may be eliminated from further consideration (U.S. EPA 1989a). Based on the comparison to background and a less than 5 percent detection frequency, arsenic in ground water is not assessed quantitatively as a COPC. A qualitative discussion regarding potential arsenic ground-water risk is provided in the uncertainty analysis (Subsection 2.6.4).

2.1.5 COPCs Selected

Tables 2.1 through 2.6 present the occurrence, distribution, and selection of COPCs in media of concern at Site 16. The tables are media-specific and present the minimum and maximum detected concentrations, as well as the frequency of detection for each chemical detected. COPCs are presented in bold type.

2.1.5.1 COPCs in Soil

COPCs in surface soil at Site 16 under the residential screen (Table 2.1) are dioxin, aluminum, arsenic, manganese, thallium, benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene.

Surface soil COPCs at Site 16 based on industrial soil PRG screening (Table 2.2) are dioxin, arsenic, benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, and dibenz(a,h)anthracene.

For Site 16, COPCs in total soil under the residential screen (Table 2.3) are dioxin, aluminum, antimony, arsenic, barium, copper, lead, manganese, thallium, 2-methylnaphthlene, benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, dibenz(a,h)anthracene, indeno(1,2,3-cd)pyrene, and naphthalene.

Total (combined surface and subsurface) soil COPCs at Site 16 under the industrial screen (Table 2.4) are dioxin, arsenic, lead, 2-methylnaphthlene, benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, and dibenz(a,h)anthracene.

2.1.5.2 COPCs in Seep Water

COPCs in seep water at Site 16 based on a comparison to tap water PRGs (Table 2.5) are arsenic, barium, lead, manganese, 2-methylnaphthalene, naphthalene, alpha-HCH, dieldrin, heptachlor epoxide, dibenzofuran, and vinyl chloride.

2.1.5.3 COPCs in Ground Water

COPCs in ground water are selected based on comparison to tap water PRGs and to established site-wide inorganic ground-water values (Stone & Webster 1996). The background values are presented in the screening table for ground water (Table 2.6) and are provided in Table 2.7. COPCs in ground water at Site 16 based on a comparison to tap water PRGs (Table 2.6) are barium, lead, manganese, 1,4-dichlorobenzene, bis(2-ethylhexyl)phthlate, 1,1,2-trichloroethane, 1,1-dichloroethene, 1,2-dichloroethane, acetone, benzene, chloroform, cis 1,2-dichloroethene, total 1,2-dichloroethene, trichloroethene, and vinyl chloride.

Several volatile organic compounds (VOCs) were included as COPCs based on a comparison to the tap water screening values. However, three of these COPCs were detected at a frequency below 5 percent: dichlorobenzene, chloroform, and vinyl chloride. The uncertainty and associated high bias in risk calculation associated with assessing chemicals that are detected at such low frequencies is discussed further in the uncertainty analysis (Subsection 2.6.4).

2.2 EXPOSURE ASSESSMENT

An exposure assessment is conducted to estimate the magnitude of potential human exposures to COPCs in site media. In the exposure assessment, central tendency and reasonable maximum estimates of potential exposure are developed in accordance with U.S. EPA guidance. Conducting an exposure assessment involves analyzing releases of COPC; identifying all potential pathways of exposure; estimating central tendency and reasonable maximum potential exposure point concentrations (EPCs) for specific pathways, based both on environmental monitoring data and predictive chemical modeling results; and estimating potential chronic daily intakes for specific pathways. The results of this assessment are pathway-specific estimates of potential intakes for exposures to individual COPCs.

2.2.1 Quantification of Potential Exposures

The first step of the exposure assessment is to quantify potential exposure concentrations. This involves the evaluation of site data and the quantification of exposure concentrations for central tendency and reasonable maximum exposure (RME) scenarios.

2.2.1.1 Data Quality Evaluation

Data for the HHRA were collected at Site 16 and are discussed in detail in the body of the RI. Inclusion or exclusion of data on the basis of analytical qualifiers was performed in accordance with U.S. EPA guidance (U.S. EPA 1989a).

Analytical results bearing the U qualifier (indicating that the analyte is not detected at the given Sample Quantitation Level [SQL]) are retained in the data set and considered non-detects. Where warranted for statistical purposes, each COPC is assigned a numerical value of one-half its SQL.

Analytical results bearing the J qualifier (indicating that the reported value is estimated because the analyte is detected at a concentration below the SQL or for other reasons) are retained at the measured concentration.

Common laboratory contaminants, including acetone, 2-butanone, methylene chloride, chloroform, toluene, phthalate esters, and uncommon laboratory contaminants are considered to be COPCs unless it is evident that their presence is not related to site-specific activities but is due to laboratory contamination.

2.2.1.2 Analysis of COPC Data

To assess human health risks, the statistical analyses of the COPC concentrations in each medium are performed. The methods used to analyze the data for each of these media are described below.

Reported concentrations are used to calculate the 95th percentile Upper Confidence Limit of the Mean (95UCLM) for COPCs in each medium (U.S. EPA 1992a). EPCs in site media are estimated as the 95UCLM values for purposes of estimating RMEs. In cases where the 95UCLM values exceed the maximum detected concentration, the maximum detected concentration is used. The first step in estimation of EPC is to determine whether medium-specific environmental data for a COPC are normally or log-normally distributed. For a log-normally distributed COPC, the following steps are carried out to calculate 95UCLM. Because transformation is a necessary step in calculating the UCLM for a log-normal distribution, the data are transformed by using the natural logarithm function (i.e., calculate ln(x), where x is the value from the data set). After transforming the data, 95UCLM for the data set is calculated by calculating the arithmetic mean of the transformed data; calculating standard deviation of the transformed data; determining H-statistic (Gilbert 1987); and calculating 95UCLM using the equation given below:

$$95\,UCLM = e^{(\bar{x} + 0.5\,s^2 + sH\,/\,\sqrt{n-1})} \qquad \text{(Equation 1)}$$

where:

95UCLM	=	95th percentile upper confidence limit on the mean
e	=	Constant (base of the natural logarithm; equal to 2.718)
x	=	Mean of the transformed data
s	=	Standard deviation of the transformed data
H	=	H-Statistic
n	=	Number of samples in the data set

If the statistical test supported the assumption that the data set for a COPC is normally distributed, the following steps are undertaken to calculate 95UCLM (U.S. EPA 1992a): (1) calculate the arithmetic mean of the untransformed data; (2) calculate standard deviation of the untransformed data; (3) determine the one-tailed t-statistic (Gilbert 1987); and (4) calculate 95UCLM using the equation given below:

$$95\,UCLM = (\bar{x} + t\,s\,/\,\sqrt{n}) \qquad \text{(Equation 2)}$$

where:

95UCLM	=	95th percentile upper confidence limit on the mean
x	=	Mean of the untransformed data
s	=	Standard deviation of the untransformed data
t	=	Student-t statistic
n	=	Number of samples in the data set.

2.2.2 Exposure Point Concentrations

Tables 3.1 through 3.6 present summary statistics (e.g., frequency of detection, range of detection, mean, and the 95UCLM) for each COPC per medium. These tables also present the selection of EPCs for use in the remainder of the risk assessment. The RME EPC value is utilized as the chemical-specific, medium-specific EPC in the exposure assessment for the RME assumptions. However, if the 95UCLM is greater than the maximum detected concentration, the maximum detected concentration value is used as the EPC and is listed in the table instead of the 95UCLM value, as per U.S. EPA guidance (U.S. EPA 1989a). The arithmetic average is used as the central tendency exposure (CTE) scenario per U.S. EPA guidance (U.S. EPA 1989a).

2.2.3 Exposure Equations

The next step in this exposure assessment is to estimate COPC intakes for each of the pathways considered in the assessment. In this exposure assessment, we have provided two different measures of intake, depending on the nature of the effect being evaluated. When evaluating longer-term (i.e., subchronic and chronic) exposures to chemicals that produce adverse non-carcinogenic effects, intakes are averaged over the period of exposure (i.e., the averaging time [AT]) (U.S. EPA 1989a). This measure of intake is referred to as the non-carcinogenic average daily intake (NCADI) and is a less than lifetime exposure. For chemicals that produce

carcinogenic effects, intakes are averaged over an entire lifetime and are referred to as the lifetime carcinogenic average daily intake (CADI) (U.S. EPA 1989a).

The generic equation to calculate intakes is given below:

$$(L)ADI = \frac{C \times IF \times EF \times ED \times RAF}{BW \times AT} \times CF \qquad \text{(Equation 3)}$$

where:

(L)ADI	=	(Lifetime) Average daily dose (mg/kg-day)
C	=	Concentration in a specific medium (mg/L or mg/kg)
IF	=	Intake factor[1] (mg/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
RAF	=	Relative absorption factor (unitless)
BW	=	Body weight (kg)
AT	=	Averaging time (days)
CF	=	Conversion Factor (10^{-6} kg/mg)

The exposure pathways considered to be complete for Site 16 are presented in Table ES-1. The equations used to estimate exposures for each of the exposure pathways assessed for the site are presented in Attachment B.

2.2.4 Selection of Exposure Factor Values

All exposure factor values used in estimating intakes for the industrial scenario are described and referenced in Tables 4.1 through 4.11. The following guidance documents are used in defining exposure factor values for estimating intakes for exposure pathways evaluated at the site:

- *Risk Assessment Guidance for Superfund (RAGS) Volume I Human Health Evaluation Manual Part A*, U.S. EPA, December 1989 (U.S. EPA 1989a).
- *Office of Solid Waste and Emergency Response (OSWER) Directive 9285.6-03; Human Health Evaluation Manual, Supplemental Guidance: "Standard Default Exposure Factors,"* U.S. EPA, 1991 (U.S. EPA 1991).
- *Exposure Factors Handbook, Volume I, General Factors*, U.S. EPA, August 1997 (U.S. EPA 1997a).

[1] The intake factor is the product of all intake variables that, when multiplied by the concentration of the chemical of potential concern in a specific medium, results in an estimate of the chemical intake in mg/kg-day for that population and exposure pathway. Intake factors may include ingestion rate, inhalation rate, body surface area exposed to soil or water, dermal permeability constants, and soil adherence factors.

- *Dermal Exposure Assessment: Principles and Applications*, U.S. EPA 1992 (U.S. EPA 1992b).

- *Risk Assessment Guidance for Superfund, Volume 1: Human Health Evaluation Manual, Dermal Risk Assessment, Interim Guidance.* U.S. EPA, 1998 (U.S. EPA 1998b).

For all exposure pathways that have exposure factor values specified in RAGS Part A and in Office of Solid Waste and Emergency Response (OSWER) Directive 9285.6-03, those values are used in this risk assessment. For exposure values not specified in RAGS Part A or the OSWER Directive, the Region I Risk Update Number 2 (U.S. EPA 1994a) and the Exposure Factors Handbook (U.S. EPA 1997a) are used to determine appropriate exposure values, where available. U.S. EPA dermal guidance documents are also utilized (U.S. EPA 1992b, 1998b). All exposure factor values are provided in the Site 16 RI Work Plan (EA 2000) and are presented in the following sections.

2.2.4.1 Resident Adult

Exposure parameters for resident adult exposure are presented in Tables 4.1 (total soil) and 4.7 (ground water) and in the adult residential risk calculation tables in Attachment B. Body weight for the adult resident is assumed to be 70 kilograms (kg). Under RME conditions, future adult residents are assumed to have an exposure duration of 24 years for carcinogenic risks, based on additivity with child carcinogenic risks, and an exposure duration of 30 years for non-carcinogenic risks. An exposure frequency of 150 days/year for soil and 350 days/year for ground water is assumed. Skin surface area available for contact with soil is assumed to be 8,000 cm^2, based on the 95th percentile values for feet, lower legs, hands, forearms, and head (U.S. EPA 1997a). A soil adherence factor of 0.07 mg/cm^2 is assumed based on the 1998 U.S. EPA Draft Dermal Guidance (U.S. EPA 1998b). Incidental ingestion of soil is assumed to be 100 mg/day. Incidental ingestion of ground water is assumed to be 2.3 L/day with an exposure frequency of 350 days/year. The skin surface area available for contact with ground water is assumed to be 22,000 cm^2, with an event time of 0.58 hour/day. The resident adult is also analyzed for inhalation of VOCs from showering. It is assumed that the resident adult takes 350 showers/year at 35 min/shower with an inhalation rate of 15 m^3/day.

Under CTE conditions, future adult residents are assumed to have an exposure duration of 9 years for carcinogenic risks, based on additivity with child carcinogenic risks, and an exposure duration of 30 years for non-carcinogenic risks. An exposure frequency of 150 days/year is assumed. Skin surface area available for contact with soil is assumed to be 6,200 cm^2, based on the 95th percentile values for feet, lower legs, hands, forearms, and head (U.S. EPA 1997a). A soil adherence factor of 0.01 mg/cm^2 is assumed based on the 1998 U.S. EPA Draft Dermal Guidance (U.S. EPA 1998b). Incidental ingestion of soil is assumed to be 50 mg/day. Incidental ingestion of ground water is assumed to be 1.4 L/day with an exposure frequency of 250 days/year. The skin surface area available for contact with ground water is assumed to be 18,200 cm^2, with an event time of 0.17 hour/day. For the CTE scenario, it is assumed that the resident

adult takes 175 showers/year (one shower every other day) at 10 min/shower with an inhalation rate of 13.2 m^3/day.

2.2.4.2 Resident Child

Exposure parameters for resident child exposure are presented in Tables 4.2 (total soil) and 4.8 (ground water) and in the child residential risk calculation tables in Attachment B. Body weight for the child resident is assumed to be 15 kg. Under RME conditions, future child residents are assumed to have an exposure duration of 6 years with an exposure frequency of 150 days/year. Skin surface area available for contact with soil is assumed to be 3,600 cm^2, based on the 95th percentile values for feet, lower legs, hands, forearms, and head (U.S. EPA 1997a). A soil adherence value of 0.2 mg/cm^2 is assumed based on the 1998 U.S. EPA Draft Dermal Guidance (U.S. EPA 1998b). Incidental ingestion of soil is assumed to be 200 mg/day. Incidental ingestion of ground water is assumed at 1.5 L/day with an exposure frequency of 350 days/year. The skin surface area available for contact with ground water is assumed to be 10,500 cm^2, with an event time of 1 hour/day.

Under CTE conditions, future child residents are assumed to have an exposure duration of 2 years with an exposure frequency of 150 days/year. Skin surface area available for contact with soil is assumed to be 2,900 cm^2, based on the 95th percentile values for feet, lower legs, hands, forearms, and head (U.S. EPA 1997a). A soil adherence value of 0.06 mg/cm^2 is assumed based on the 1998 U.S. EPA Draft Dermal Guidance (U.S. EPA 1998b). Incidental ingestion of soil is assumed to be 100 mg/day. Incidental ingestion of ground water is assumed at 0.74 L/day with an exposure frequency of 250 days/year. The skin surface area available for contact with ground water is assumed to be 9,000 cm^2, with an event time of 0.33 hour/day.

2.2.4.3 Adult Recreational User

Exposure parameters for adult recreational users are presented in Tables 4.3 (surface soil) and 4.10 (seep water) and in the pathway-specific adult recreational user risk calculation tables in Attachment B. Body weight is assumed to be 70 kg. Under RME conditions, future adult recreational users are assumed to have an exposure duration of 24 years. An exposure frequency of 96 days/year is assumed. Skin surface area available for contact with soil is assumed to be 8,000 cm^2 for adults based on the 95th percentile values for feet, lower legs, hands, forearms, and head (U.S. EPA 1997a). A soil adherence factor of 0.32 mg/cm^2 is assumed (U.S. EPA 1998b). Incidental ingestion of soil is assumed to be 100 mg/day for the adult recreational user. An inhalation rate of 0.833 m^3/hr for 8 hours/day with an exposure frequency of 96 days/year is assumed for soil dust particulates. The adult recreational user is assumed to contact seep water present at Site 16. It is assumed that the adult recreational user ingested 0.05 L/day of seep water with an exposure frequency of 7 days/year. Skin surface area available for contact with seep water is assumed to be 6,700 cm^2 with an exposure time of 1 hour/day.

Under CTE conditions, future adult recreational users are assumed to have an exposure duration of 9 years. An exposure frequency of 48 days/year is assumed. Skin surface area available for contact with soil is assumed to be 6,200 cm^2 for adults based on the 95th percentile values for

feet, lower legs, hands, forearms, and head (U.S. EPA 1997a). A soil adherence factor of 0.16 mg/cm^2 is assumed (U.S. EPA 1998b). Incidental ingestion of soil is assumed to be 50 mg/day for the adult recreational user. An inhalation rate of 0.833 m^3/hr for 4 hours/day with an exposure frequency of 48 days/year is assumed for soil dust particulates. For the CTE scenario, it is assumed that the adult recreational user ingested 0.03 L/day of seep water with an exposure frequency of 4 days/year. Skin surface area available for contact with seep water is assumed to be 5,000 cm^2 with an exposure time of 0.5 hour/day.

2.2.4.4 Child Recreational User

Exposure parameters for child recreational users are presented in Tables 4.4 (surface soil) and 4.11 (seep water) and in the pathway-specific child recreational user risk calculation tables in Attachment B. Body weight is assumed to be 15 kg. Under RME conditions, future child recreational users are assumed to have an exposure duration of 6 years. An exposure frequency of 96 days/year is assumed. Skin surface area available for contact with soil is assumed to be 3,600 cm^2 for children based on the 95th percentile values for feet, lower legs, hands, forearms, and head (U.S. EPA 1997a). A soil adherence factor of 0.32 mg/cm^2 is assumed (U.S. EPA 1998b). Incidental ingestion of soil is assumed to be 200 mg/day for the child recreational user. An inhalation rate of 0.833 m^3/hr for 10 hours/day with an exposure frequency of 96 days/year is assumed for soil dust particulates. The child recreational user is assumed to contact seep water present at Site 16. It is assumed that the child recreational user ingested 0.05 L/day of seep water with an exposure frequency of 7 days/year. Skin surface area available for contact with seep water is assumed to be 3,300 cm^2 with an exposure time of 1 hour/day.

Under CTE conditions, future child recreational users are assumed to have an exposure duration of 2 years. An exposure frequency of 48 days/year is assumed for children. Skin surface area available for contact with soil is assumed to be 2,900 cm^2 for children based on the 95th percentile values for feet, lower legs, hands, forearms, and head (U.S. EPA 1997a). A soil adherence factor of 0.16 mg/cm^2 is assumed (U.S. EPA 1997b). Incidental ingestion of soil is assumed to be 100 mg/day for the child recreational user. An inhalation rate of 0.833 m^3/hr for 4 hours/day with an exposure frequency of 48 days/year is assumed for soil dust particulates. For the CTE scenario, it is assumed that the child recreational user ingested 0.03 L/day of seep water with an exposure frequency of 4 days/year. Skin surface area available for contact with seep water is assumed to be 2,700 cm^2 with an exposure time of 0.5 hour/day.

2.2.4.5 Construction Worker

Exposure parameters for construction worker exposure are presented in Table 4.5 for total soil and Table 4.9 for ground water. Body weight for the construction worker is assumed to be 70 kg. Under RME conditions, future construction workers are assumed to have an exposure duration of 1 year with an exposure frequency of 160 days/year. Skin surface area available for contact with total soil during construction activities is assumed to be 3,800 cm^2, representing the 95th percentile value for head, hands, and forearms (U.S. EPA 1997a). A 0.3 mg/cm^2 adherence factor is assumed (U.S. EPA 1998b). Incidental ingestion of soil is assumed to be 480 mg/day. The RME inhalation rate is assumed to be 2.5 m^3/hour for total soil particulate over an exposure

time of 8 hours per day. Incidental ingestion of ground water is assumed at 0.05 L/day with an exposure frequency of 160 days/year. The skin surface area available for contact with ground water is assumed to be 5,700 cm^2, with an event time of 8 hours/day.

Under CTE conditions, future construction workers are assumed to have an exposure duration of 1 year with an exposure frequency of 80 days/year. Skin surface area available for contact with total soil during construction activities is assumed to be 3,140 cm^2. A 0.1 mg/cm^2 adherence factor is assumed (U.S. EPA 1998b). Incidental ingestion of soil is assumed to be 100 mg/day. The CTE inhalation rate is assumed to be 1.5 m^3/hour for particulate over an exposure time of 8 hours/day. Incidental ingestion of ground water is assumed at 0.025 L/day with an exposure frequency of 80 days/year. The skin surface area available for contact with ground water is assumed to be 2,300 cm^2, with an event time of 8 hour/day.

2.2.4.6 Commercial Worker

Exposure parameters for commercial worker exposure are presented in Table 4.6 for surface soil for the commercial worker. Body weight for the commercial worker is assumed to be 70 kg. Under RME conditions, commercial workers are assumed to have an exposure duration of 25 years with an exposure frequency of 250 days/year. A surface soil ingestion rate of 50 mg/day is assumed. Dermal exposure to soil is based on 3,800 cm^2 surface area, representing the 95th percentile value for head, hands, and forearms (U.S. EPA 1989a). A 0.2 mg/cm^2 adherence factor is assumed (U.S. EPA 1998b). The RME inhalation rate is assumed to be 0.833 m^3/hour for particulate over an exposure time of 8 hours/day.

Under CTE conditions, commercial workers are assumed to have an exposure duration of 12 years with an exposure frequency of 150 days/year. A surface soil ingestion rate of 50 mg/day is assumed. CTE dermal exposure to surface soil is based on exposed 3,100 cm^2 surface area and 0.02 mg/cm^2 adherence factor. The CTE inhalation rate is assumed to be 0.833 m^3/hour for particulate over an exposure time of 4 hours/day.

2.3 TOXICITY ASSESSMENT

The toxicity assessment considers the types of potential adverse health affects associated with exposures to COPCs; the relationship between magnitude of exposure and potential adverse effects; and related uncertainties, such as the weight of evidence of a particular COPC's carcinogenicity in humans. The toxicity assessment for COPCs relies on existing toxicity information developed on specific organic compounds and inorganic constituents. U.S. EPA Guidance (U.S. EPA 1989a) specifies that the assessment is accomplished in two steps: hazard identification and dose-response assessment.

Hazard identification is the process of determining whether studies claim that exposure to a COPC may cause the incidence of an adverse effect. U.S. EPA specifies that the dose-response assessment include: (1) U.S. EPA's quantitative evaluation of the existing toxicity information, and (2) EPA's characterization of the relationship between the dose of the COPC administered or received, and the incidence of potentially adverse health effects in the exposed population.

From this quantitative dose-response relationship, specific toxicity values are derived by U.S. EPA that can be used to estimate the incidence of potentially adverse effects occurring in humans at different exposure levels (U.S. EPA 1989a). These U.S. EPA-derived toxicity values are called RfDs for non-carcinogens and SFs for potential carcinogens.

The toxicity values used for COPCs at Site 16 are presented in Tables 5.1 and 5.2 for non-carcinogens and in Table 6.1 and 6.2 for carcinogens; related chemical-specific parameters are presented in Table 5.3.

2.3.1 Toxicity Assessment for Non-Carcinogens

For all COPCs, toxicity values for non-carcinogens are taken, when available, from the Integrated Risk Information Systems (IRIS) database (U.S. EPA 2001b). IRIS chronic toxic potency concentrations are developed by U.S. EPA and undergo an extensive process of scientific peer review. Therefore, IRIS values are judged to be adequately verified.

If toxic potency concentrations for COPCs are not available from IRIS (U.S. EPA 2001b), health effects assessment summary tables (HEAST) (U.S. EPA 1997b) are used as a secondary data source. As HEAST toxicity values are not scientifically peer-reviewed for quality or scientific acceptability, they may not be derived in strict accordance with U.S. EPA approved methodologies.

If IRIS or HEAST toxic potency concentrations are not available for one route of exposure but existed for another route, the existing value is examined for technical applicability to the alternate route and subsequently utilized, if appropriate.

The methodology used by U.S. EPA for deriving toxic potency concentrations for non-carcinogens, as well as site-specific considerations for modifying or using these concentrations, are discussed in detail in Barnes and Dourson (1988) and U.S. EPA guidance (U.S. EPA 1989a). Non-carcinogens are typically judged to have a threshold daily dose below which deleterious or harmful effects are unlikely to occur. This concentration is called the no-observed-adverse-effect-level (NOAEL) and may be derived from either animal laboratory experiments or human epidemiology investigations (usually workplace studies). In developing a toxicity value or human NOAEL for non-carcinogens (i.e., an RfD), the regulatory approach is first to: (1) identify the critical toxic effect associated with chemical exposure (i.e., the most sensitive adverse effect); (2) identify the threshold dose in either an animal or human study; and (3) modify this dose to account for interspecies variability (where appropriate), differences in individual sensitivity (within-species variability), and other uncertainty and modifying factors.

Uncertainty factors are intended to account for specific types of uncertainty inherent in extrapolation from the available data. Modifying factors account for the concentration of confidence in the scientific studies from which toxicity values are derived, according to such parameters as study quality and study reproducibility. The use of these factors is a conservative approach to protection of human health and is likely to overestimate the toxic potency associated

with chemical exposure. The resulting RfD is expressed in units of milligrams of chemical per kilogram of body weight per day (mg/kg-bw/day).

Toxicity values used for exposures that involve dermal contact with chemicals typically require adjustment of the oral toxicity values (oral RfDs) to allow for the difference between the daily intake dose through dermal contact and ingestion. Most toxicity values are based on the actual administered dose, and must be corrected for the percent of chemical-specific absorption that occurs across the gastrointestinal tract prior to their use in dermal contact risk assessment (U.S. EPA 1989a, 1992b, 1998b). Recommended oral absorption efficiency factors are utilized in converting oral toxicity values to dermal toxicity values. These factors are shown in Table 5.1.

2.3.2 Toxicity Assessment for Carcinogenicity

Unlike non-carcinogens, carcinogens are generally assumed to have no threshold; that is, there is presumed to be no level of exposure below which carcinogenic effects will not manifest themselves. This "non-threshold" concept supports the idea that there are small, finite probabilities of inducing a carcinogenic response associated with every level of exposure to a potential carcinogen. U.S. EPA uses a two-part evaluation for carcinogenic effects, which includes the assignment of a weight-of-evidence classification to a chemical based on a thorough scientific examination of the body of available data, and the quantification of a cancer toxic potency concentration; i.e., the slope factor, which reflects the dose-response data for the carcinogenic endpoint(s) (U.S. EPA 1989a).

The weight-of-evidence classification system assigns a letter or alphanumeric (A through E) to each potential carcinogen that reflects an assessment of its potential to be a human carcinogen.[2] Only compounds that have a weight-of-evidence classification of C or above are considered to have carcinogenic potential in this risk assessment.

Although currently a controversial approach, chemicals that are classified as human or rodent carcinogens are typically assumed to have no threshold, in that there is presumed to be no concentration of exposure below which carcinogenic effects will not be manifested. The U.S. EPA SF is the 95UCLM of the probability of response per unit daily intake of a chemical over a lifetime. Typically, the SF is used to estimate the upper-bound lifetime probability of a person developing cancer from exposure to a given concentration of a carcinogen. SFs are generally based on experimental animal data, unless suitable epidemiological studies are available. Due to the difficulty in detecting and measuring carcinogenic endpoints at low exposure concentrations, SFs are typically developed by using a model to fit the available high-dose, experimental animal data, and then extrapolating downward to the low-dose range to which humans are typically exposed. U.S. EPA usually employs the linear multistage model to

[2]A = a known human carcinogen; B1 = a probable human carcinogen, based on sufficient animal data and limited human data; B2 = a probable human carcinogen based on sufficient animal data and inadequate or no human data; C = a possible human carcinogen; D = not classifiable as to human carcinogenicity; and E = evidence of non-carcinogenicity for humans.

derive a SF. The model is conservative, and provides an upper bound estimate of excess lifetime cancer risk. Thus, the actual risk may be lower and could be zero (U.S. EPA 1989a). These methods and approaches are discussed in greater detail in the U.S. EPA *Risk Assessment Guidance for Superfund* (U.S. EPA 1989a).

Carcinogenic SFs used for exposures that involve dermal contact typically require adjustment of the oral SF to allow for the difference between the dermal dose and the ingested dose. Most toxicity values are based upon the actual administered dose and must be corrected for the percent of chemical-specific absorption that occurs across the gastrointestinal tract prior to their use in dermal contact risk assessments (U.S. EPA 1989a). For inhalation exposures, inhalation SFs are developed if sufficient data are available.

2.3.3 Toxicity of Constituents of Potential Concern

Toxicity data available in the scientific literature are used to prepare a toxicity profile for each COPC. Each profile describes the potential for carcinogenicity and other health effects of each COPC, summarizes available data, presents a weight-of-evidence approach for identifying the hazards associated with chemical exposure to the COPC, and provides a scientific profile for selecting the most appropriate toxicity values (i.e., quantitative estimates of the strength of the dose-response) used later in the risk assessment.

A review of relevant toxicity data for each COPC is presented in Attachment A, along with a description of critical studies (i.e., studies from which the quantitative toxic potency values are derived). Toxicity values are obtained from the U.S. EPA IRIS (U.S. EPA 2001b), a peer-reviewed toxicity database. If toxicity values are not available from IRIS, values from the U.S. EPA HEAST (U.S. EPA 1997b) or from the National Center for Environmental Assessment (NCEA) are used.

2.3.3.1 Summary of Toxicity Values for Non-Carcinogenic Effects

U.S. EPA-derived toxicity values for evaluating potential chronic non-carcinogenic effects for COPCs are summarized in Tables 5.1 and 5.2. Toxicity information presented in these tables includes the following U.S. EPA provided/derived information: chronic or subchronic RfD values for exposures via the oral and inhalation pathway; reported health effects, uncertainty and modifying factors specific to the U.S. EPA-derived RfD; and the scientific source of the information.

2.3.3.2 Summary of Toxicity Values for Potential Carcinogenic Effects

U.S. EPA-derived toxicity values for evaluating potential carcinogenic effects for COPCs are summarized in Tables 6.1 and 6.2. Toxicity information presented in these tables includes the following U.S. EPA provided/derived information: a chemical-specific SF (cancer potency factor) for exposures via the oral and inhalation pathway; U.S. EPA's weight-of-evidence cancer classification; and the scientific source of the information.

2.4 RISK CHARACTERIZATION

Risk characterization is the final step of the HHRA process. In this step, the toxicity values are combined with the estimated chemical intakes for the receptor populations to quantitatively estimate both carcinogenic and non-carcinogenic risks. Risks are estimated for the following receptor populations: residents (adult and child), adult recreational users, child recreational users, construction workers, and commercial workers.
The methodologies used to estimate cancer risks and chronic and subchronic risks for non-carcinogens are described further in the sections below.

2.4.1 Hazard Quotient and Hazard Index for Non-Carcinogenic Effects

The potential human health risks associated with exposures to non-carcinogenic COPCs at Site 16 are estimated by comparing the NCADI with the RfD, as per U.S. EPA Guidance (U.S. EPA 1989a). A hazard quotient (HQ) is derived for each COPC, as shown in the equation below:

$$HQ = \frac{NCADI}{RfD} \qquad \text{(Equation 4)}$$

where:

HQ	=	Hazard quotient; ratio of average daily intake level to acceptable daily intake level (unitless)
NCADI	=	Estimated non-carcinogenic average daily dose (mg/kg-day)
RfD	=	Reference dose (mg/kg-day)

If the average daily dose exceeds the RfD, then the HQ will exceed a ratio of 1.0 and there may be concern that potential adverse systemic health effects will be observed in the exposed populations. If the NCADI does not exceed the RfD, the HQ will not exceed 1.0 and there will be no concern that potential adverse systemic health effects will be observed in the exposed populations. However, if the sum of several HQs exceeds 1.0, and the COPCs affect the same target organ, there may be concern that potential adverse systemic health effects will be observed in the exposed populations. In general, the greater the value of the HQ above 1.0, the greater the level of concern. However, the HQ does not represent a statistical probability that an adverse health effect will occur.

For consideration of exposures to more than one chemical causing systemic toxicity via several different pathways, the individual HQs are summed to provide an overall Hazard Index (HI). If the HI is less than 1.0, then no adverse health effects are likely to be associated with exposures at the site. However, if the total HI is greater than 1.0, separate endpoint-specific HIs may be calculated based on toxic endpoint of concern or target organ (e.g., HQs for neurotoxins are summed separately from HQs for renal toxins). Only if an endpoint-specific HI is greater than 1.0 is there reason for concern about potential health effects for that endpoint.

2.4.2 Cancer Risks

Carcinogenic risk is estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential carcinogen at the site. The numerical estimate of excess lifetime cancer risk is calculated by multiplying the CADI by the risk per unit dose (the SF), as shown in the following equation:

$$Risk = CADI \times SF \qquad \text{(Equation 5)}$$

where:

Risk	=	The unitless probability of an exposed individual developing cancer
CADI	=	Lifetime cancer average daily dose (mg/kg-day)
SF	=	Cancer slope factor $(mg/kg\text{-}day)^{-1}$

Because the SF is the statistical 95th percent upper-bound confidence limit on the dose-response slope, this method provides a conservative, upper-bound estimate of risk.

It should be noted that the interpretation of the significance of the cancer risk estimate is based on the appropriate public policy. U.S. EPA in the National Oil and Hazardous Substance Pollution Contingency Plan (NCP) [40 Code of Federal Regulations (CFR) Part 300] (U.S. EPA 1990) states that:

> *"...For known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent an excess upper bound lifetime cancer risk to an individual of between 10^{-4} and 10^{-6}."*

2.5 RESULTS

Chemical-specific, pathway-specific risk estimates are presented by receptor group in Attachment B. Risk characterization equations and calculations are also presented in these tables. Estimates of cumulative risks across pathways for non-carcinogenic and carcinogenic effects for each receptor population are presented in Tables 9.1 through 9.10. A summary of the risk drivers for each pathway are presented for the RME scenario in Tables 10.1 through 10.5.

Lead risks were evaluated using U.S. EPA's blood-lead level models for residents and workers. Lead risks are discussed in Subsection 2.5.6.

2.5.1 Residential Results

Site 16 residential results are presented in Tables 9.1 for the CTE scenario and 9.6 for the RME scenario. A summary of the risk drivers for the RME scenario is provided in Table 10.1. Non-carcinogenic and cancer risks for potential total soil and ground-water exposure are evaluated.

The residential adult cumulative HIs across all pathways are 3.0 for the CTE scenario and 39 for the RME scenario, respectively. For total soil, the HIs are 0.01 for CTE and 0.04 for RME. The cumulative HIs for ground water are 3.0 and 39 for CTE and RME, respectively. COPCs with an HQ exceeding 1.0 are manganese and trichloroethene. The analysis by target organ (Table 10.1) indicates that the HIs for the central nervous system, liver, and kidney exceed 1.0. This is based entirely on the ground-water pathway results.

The residential child cumulative HIs across all pathways are 6.6 for the CTE scenario and 120 for the RME scenario, respectively. For total soil, the HIs are 0.09 for CTE and 0.34 for RME. The cumulative HIs for ground water are 6.6 and 120 for CTE and RME, respectively. COPCs with an HQ exceeding 1.0 are manganese and trichloroethene. The analysis by target organ (Table 10.1) indicates that the HIs for the central nervous system, liver, and kidney all exceed 1.0. This is based entirely on the ground-water pathway results.

Carcinogenic risks for the residential scenario are combined for the adult and child resident receptors to assess potential lifetime risks. Cancer risks for the residential scenario are 4.7×10^{-5} and 2.0×10^{-3} for CTE and RME, respectively. Cancer risks for total soil are 2.0×10^{-6} and 3.8×10^{-5} for CTE and RME, respectively. Cumulative cancer risks for total soil did not exceed U.S. EPA's "acceptable risk range" of 10^{-6} to 10^{-4} (Table 10.1). Cancer risks for ground water are calculated as 4.7×10^{-5} for CTE and 2.0×10^{-3} under the RME. Residential risks are within the U.S. EPA's "acceptable risk range" of 10^{-6} to 10^{-4} (Table 10.1) for total soil but exceeded the range for ground water for the combined adult and child receptor. COPCs with risks exceeding 10^{-6} included arsenic in soil and bis(2-ethylhexyl)phthalate, 1,4-dichlorobenzene, 1,1,2-trichlorethane, 1,2-dichloroethane, benzene, trichloroethene, and vinyl chloride in ground water.

Lead risks are discussed in Subsection 2.5.6.

2.5.2 Adult Recreational User Results

Adult recreational user results are presented in Table 9.2 for the CTE scenario and Table 9.7 for the RME scenario. A summary of the risk drivers for the RME scenario is provided in Table 10.2. Non-carcinogenic and cancer risks for surface soil and seep water exposure are evaluated.

The adult recreational user cumulative HIs across all pathways are 0.002 for the CTE scenario and 0.002 for the RME scenario, respectively. For surface soil, the HIs are 0.002 for CTE and 0.014 for RME. The cumulative HIs for seep water are 0.00007 and 0.007 for CTE and RME, respectively.

Cancer risks for the adult recreational user scenario are 1.1×10^{-7} and 6.4×10^{-6} for CTE and RME, respectively. Cancer risks for surface soil are 1.1×10^{-7} and 6.3×10^{-6} for CTE and RME, respectively. Cancer risks for seep water are calculated as 2.9×10^{-10} for CTE and 4.6×10^{-8} under

the RME. Adult recreational user risks are within the U.S. EPA's "acceptable risk range" of 10^{-6} to 10^{-4} (Table 10.2).

Lead risks are discussed in Subsection 2.5.6.

2.5.3 Child Recreational User Results

Site 16 child recreational user results are presented in Table 9.3 for the CTE scenario and Table 9.8 for the RME scenario. A summary of the risk drivers for the RME scenario is provided in Table 10.1. Non-carcinogenic and cancer risks for potential surface soil and seep water exposure are evaluated.

The child recreational user cumulative HIs across all pathways are 0.02 for the CTE scenario and 0.13 for the RME scenario, respectively. For surface soil, the HIs are 0.02 for CTE and 0.11 for RME. The cumulative HIs for seep water are 0.002 and 0.02 for CTE and RME, respectively.

Cancer risks for the child recreational user scenario are 2.7×10^{-7} and 7.4×10^{-6} for CTE and RME, respectively. Cancer risks for surface soil are 2.7×10^{-7} and 7.3×10^{-6} for CTE and RME, respectively. Cancer risks for seep water are calculated as 2.3×10^{-9} for CTE and 4.6×10^{-8} under the RME. Child recreational user risks are within the U.S. EPA's "acceptable risk range" of 10^{-6} to 10^{-4} (Table 10.3).

Lead risks are discussed in Subsection 2.5.6.

2.5.4 Construction Worker Results

Site 16 construction worker results are presented in Table 9.4 for the CTE scenario and Table 9.9 for the RME scenario. A summary of the risk drivers for the RME scenario is provided in Table 10.4. Non-carcinogenic and cancer risks for potential total soil and ground-water exposure are evaluated.

The construction worker cumulative HIs across all pathways are 0.2 for the CTE scenario and 5.7 for the RME scenario, respectively. For total soil, the HIs are 0.003 for CTE and 0.09 for RME. The cumulative HIs for ground water are 0.2 and 5.6 for CTE and RME, respectively. The only COPC with an HQ exceeding 1.0 is trichloroethene in ground water.

Carcinogenic risks for the construction worker scenario are 2.7×10^{-7} and 7.2×10^{-6} for CTE and RME, respectively. Cancer risks for total soil are 8.3×10^{-8} and 2.3×10^{-6} for CTE and RME, respectively. Cancer risks for ground water are calculated as 1.9×10^{-7} for CTE and 5.0×10^{-6} under the RME. Trichloroethene (TCE) in ground water and the dioxin-toxic equivalent (TCDD-TEQ) in surface soil had cancer risks exceeding 10^{-6}. Cumulative cancer risks for the construction worker did not exceed U.S. EPA's "acceptable risk range" of 10^{-6} to 10^{-4} (Table 10.4).

2.5.5 Commercial Worker Results

Site 16 commercial worker results are presented in Table 9.5 for the CTE scenario and Table 9.10 for the RME scenario. A summary of the risk drivers for the RME scenario is provided in Table 10.5. Non-carcinogenic and cancer risks for potential surface soil exposure are evaluated.

The commercial worker cumulative HIs for surface soil are 0.002 for the CTE scenario and 0.006 for the RME scenario, respectively.

Carcinogenic risks for the commercial worker scenario are 4.3×10^{-7} and 6.2×10^{-6} for CTE and RME, respectively. TCDD-TEQ, benzo(a)pyrene, and dibenz(a,h)anthracene had cancer risks exceeding 10^{-6}. Cumulative cancer risks for the construction worker did not exceed U.S. EPA's "acceptable risk range" of 10^{-6} to 10^{-4}.

2.5.6 Lead

According to U.S. EPA, lead is classified as a B2-probable human carcinogen. However, there is no U.S. EPA value for use as a SF in quantifying cancer risks. In the absence of any U.S. EPA-published toxicity values for lead, it is currently not possible to perform a quantitative risk estimate for lead exposures using standard U.S. EPA methodology. The current U.S. EPA guidance sets forth an interim soil cleanup level for total lead at 400 parts per million (ppm) (U.S. EPA 1989b) which is considered "protective for direct contact at residential settings." According to U.S. EPA, this guidance adopts the recommendation in the 1985 Centers for Disease Control (CDC) statement on childhood lead poisoning and is to be followed when the current or future land use is residential. The recommendation states that "...lead in soil and dust appears to be responsible for blood levels in children increasing above background levels when the concentration in soil or dust exceeds 500 to 1,000 ppm..."

2.5.6.1 Residential Lead Risks

Infants and young children are the most vulnerable populations exposed to lead and have been the focus of U.S. EPA's risk assessment efforts. The relatively high vulnerability of infants and children results from a combination of factors: (1) an apparent intrinsic sensitivity of developing organs to lead; (2) behavioral characteristics that increase contact with lead from soil and dust (e.g., mouthing behavior); (3) various physiologic factors resulting in a greater deposition of airborne lead in the respiratory tract and greater absorption efficiency from the gastrointestinal tract in children than in adults; and (4) transplacental transfer of lead that establishes a lead burden in the fetus, thus increasing the risk associated with additional exposure during infancy and childhood.

For resident children, the risks associated with lead were estimated using the U.S. EPA Integrated Exposure Uptake Biokinetic (IEUBK) Model. The IEUBK Model is used to estimate blood lead concentrations resulting from exposure to environmental sources. This method has been suggested by the U.S. EPA Office of Air Quality Planning and Standards (OAQPS) Staff

Report (U.S. EPA 1989b). The IEUBK Model is a three-stage method for estimating total blood lead levels. First, the intake of lead from each source is assessed. Second, the uptake of lead from each source is determined. Finally, the relationship between the uptake of lead and blood lead concentration is applied using the "Integrated Metabolic Model for Humans of All Ages," as revised by Harley and Kneip (1985).

The Harley and Kneip model incorporates total uptake of lead derived from all exposures and the distribution of lead to the four body compartments (blood, bone, liver, and kidney) in which 95 percent of the lead is found. The model was developed based on the distribution and equilibrium of stable lead and a naturally occurring radioactive lead isotope in the bodies of infant and child baboons, and in humans during continuous lead exposure. Model validation has been performed by using data collected from lead smelter sites, by using experimental data on blood lead concentrations in infants, and by studies of lead accumulation in bones under controlled conditions in adults (U.S. EPA 1989b). The model predicts a linear increase in blood lead with increasing lead uptake. However, above a blood lead concentration of 30 µg/dL (microgram/deciliter), the relationship is not linear (U.S. EPA 1989b). Therefore, this model may only be applied for moderately low lead uptakes.

U.S. EPA has created a software program of the IEUBK Model, for predicting blood lead levels in children ages 0 to 84 months, for use on a personal computer. The most current software is LEAD version 0.99D (LEAD99D). LEAD99D is used in this assessment for estimating blood lead levels in children at this site. Standard default values used as input parameters for the model are described in U.S. EPA Guidance (U.S. EPA 1994b). The model output is a probability distribution function describing the percentage of children predicted to have blood levels exceeding 10 µg/dL. More specific information on this model is contained in U.S. EPA Guidance (U.S. EPA 1994b).

The arithmetic average for lead in total soil at Site 16 is 160 mg/kg. The RME EPC for lead in ground water is 3.63 µg/L (micrograms per liter) based on the arithmetic average. The IEUBK Model is run using the average total soil concentration as soil/dust intake and the drinking water concentration as the ground-water lead arithmetic average. Average lead concentrations were used in estimating lead risks as prescribed by U.S. EPA (U.S. EPA 1994b). All other factors are run as the default model values. Results are presented in Table 7.1. Based on the IEUBK Model, calculated average blood lead estimates for residential children are 7.93 µg/dL (Table 7.1). More than 99 percent of the population is predicted to have blood lead levels less than 10 µg/dL. Lead was not selected as a COPC in surface soil. There are no concerns for adverse health effects resulting from exposure to lead in soil or ground water.

2.5.6.2 Recreational Lead Risks

Based on the conservative nature of the assumptions in the IEUBK Model, the model is also protective for a recreational scenario. The IEUBK Model is run using the average total soil concentration as soil/dust intake and the drinking water concentration as the seep water arithmetic average (13.1 µg/L). Total soil was used instead of surface soil as a conservative measure because lead was not a COPC in surface soil. The modeling of seep water as drinking

water is highly conservative and is only performed in an attempt to demonstrate that lead concentrations in seep water do not pose a threat even under the most extreme exposure scenario. In an effort to reduce the large overestimation of risks inherent in the model assumption that seep water will be used as drinking water, one-half of the model assumed drinking water intake rates are used. Average lead concentrations were used in estimating lead risks as prescribed by U.S. EPA (U.S. EPA 1994b). All other factors are run as the default model values. Results are presented in Table 7.2. Based on this conservative run of the IEUBK Model, calculated average blood lead estimates for children are 8.76 µg/dL (Table 7.2). Given that the model results were less than the 10 µg/dL threshold and that this modeling scenario is based on a residential scenario and is so conservative, there are no concerns for adverse health effects resulting from recreational exposure to lead in seep water.

2.5.6.3 Worker Lead Risks

In addition to estimating lead risks for children, the U.S. EPA Technical Review Workgroup for Lead (TRW) has developed a model to predict blood-lead levels in adult workers (U.S. EPA 1996). This model was run to assess potential workers under a commercial setting. Model default parameters were used to predict blood lead impacts for female workers and their potential children at the site. This model was run to assess potential total soil exposure for the construction worker scenario. The exposure assumptions used for a potential construction worker are shown in Table 8.1. Risks to a commercial worker were not assessed because lead was not selected as a COPC in surface soil; therefore, lead risk is not of concern under the commercial worker scenario.

The arithmetic average in total soil at Site 16 was utilized in the Adult Lead Model to evaluate the construction worker scenario. Model results are presented in Table 8.1. Potential risks to lead exposure under the construction worker are negligible.

2.6 UNCERTAINTY ASSESSMENT

There are numerous uncertainties involved in the human health risk assessment process. These are discussed briefly in the following sections.

2.6.1 Sampling and Analysis Uncertainties

The sampling plan can have a significant impact on the results obtained in calculating human health risks at a site. To the extent that samples are taken in areas that are expected to be contaminated (biased sampling), the exposure point concentration used in calculating risk exposures and risks is likely to overestimate the actual concentration encountered at the site from random exposure across the site. This sampling bias will generally result in an overestimate of exposures and risks at a site. The soil sampling at the study areas incorporated a combination of random and biased samples. As the majority of soil samples collected are biased toward potentially contaminated areas, the measured concentrations and calculated health risks would tend to be overestimated.

Analysis uncertainty may be introduced due to laboratory contamination or error. Common laboratory contaminants include phthalates and chloroform. Bis(2-ethylhexyl)phthalate and chloroform were included as COPCs in ground water based on their detection above tap water screening values. Although it is possible that these chemicals were detected due to laboratory contamination, these chemicals were included as a conservative measure. Chemicals that are common laboratory contaminants may not be site-related and their inclusion as COPCs tends to bias risk calculations high.

2.6.2 Uncertainties Analysis of Exposure Assessment

An analysis of uncertainties is an important aspect of the exposure assessment. It provides the risk assessor and reviewer with information relevant to the individual uncertainties associated with exposure factor assumptions and their potential impact on the final assessment.

2.6.2.1 Exposure Point Concentrations

A significant uncertainty exists with the basic approach used in arriving at EPCs for the COPCs in surface and total soils.

Uncertainty results from the use of one-half detection limit for all non-detects. An objective of the guidance is to include some quantitative value for COPCs when analytical data indicate that those COPCs are not detected, so that an estimated potential intake and resultant potential risk can be calculated. This approach is referenced in *Risk Assessment Guidance for Superfund (Volume 1), Human Health Evaluation Manual* (U.S. EPA 1989a). However, this approach generally overestimates the average value, and results in overestimates of intakes and subsequent risks, particularly for COPCs with low frequencies of detection.

Inorganic constituents occur naturally in the environment. It is likely that detected concentrations of some inorganic constituents are not associated with site use and are at naturally occurring levels. Therefore, it is probable that some of the calculated risks are contributed by naturally occurring background concentrations of inorganics in site soil and are not the result of activities at the site.

2.6.2.2 Soil Ingestion Rate

Soil ingestion rates for construction workers are based on studies performed by Hawley (1985). These are short-term studies, and as they are not based on average long-term exposures, they represent an overestimate of exposure.

2.6.3 Uncertainties of Toxicity Assessment

There are numerous uncertainties associated with the toxicity assessment. These are generally due to the unavailability of data to thoroughly calculate the toxicity of COPCs. These are described in more detail in the following sections.

2.6.3.1 Uncertainties Associated With Non-Carcinogenic Effects

Interspecies Extrapolation – The majority of toxicological information comes from experiments with laboratory animals. Experimental animal data have been relied on by regulatory agencies to assess the hazards of human chemical exposures. Interspecies differences in chemical absorption, metabolism, excretion, and toxic response are not well understood; therefore, conservative assumptions are applied to animal data when extrapolating to humans. These probably result in an overestimation of toxicity.

Intraspecies Extrapolation – Differences in individual human susceptibilities to the effects of chemical exposures may be caused by such variables as genetic factors (e.g., glucose-6-phosphate dehydrogenase deficiency), lifestyle (e.g., cigarette smoking and alcohol consumption), age, hormonal status (e.g., pregnancy), and disease. To take into account the diversity of human populations and their differing susceptibilities to chemically induced injury or disease, a safety factor is used. U.S. EPA uses a factor between 1 and 10. This uncertainty may lead to overestimates of human health effects at given doses.

Exposure Routes – When experimental data available on one route of administration are different from the actual route of exposure that is of interest, route-to-route extrapolation must be performed before the risk can be assessed. Several criteria must be satisfied before route-to-route extrapolation can be undertaken. The most critical assumption is that a chemical injures the same organ(s) regardless of route, even though the injury can vary in degree. Another assumption is that the behavior of a substance in the body is similar by all routes of contact. This may not be the case when, for example, materials absorbed via the gastrointestinal tract pass through the liver prior to reaching the systemic circulation, whereas by inhalation the same chemical will reach other organs before the liver. However, when data are limited these extrapolations are made, and may result in overestimates of human toxicity.

2.6.3.2 Uncertainties Associated With Carcinogenic Effects

Interspecies Extrapolation – The majority of toxicological information for carcinogenic assessments comes from experiments with laboratory animals. There is uncertainty about whether animal carcinogens are also carcinogenic in humans. While many chemical substances are carcinogenic in one or more animal species, only a very small number of chemical substances are known to be human carcinogens. The fact that some chemicals are carcinogenic in some animal species but not in others raises the possibility that not all animal carcinogens are human carcinogens. Regulatory agencies assume that humans are as sensitive to carcinogens as the most sensitive animal species. This policy decision, designed to prevent underestimation of risk, introduces the potential to overestimate carcinogenic risk.

High-Dose to Low-Dose Extrapolation – Typical cancer bioassays provide limited low-dose data on responses in experimental animals for chemicals being assessed for carcinogenic or chronic effects. The usual dose regime involves three dose groups per assay. The first dose group is given the highest dose that can be tolerated, the second is exposed to one-half that dose, and the third group is unexposed (control group) (National Research Council [NRC] 1983). Because this

dosing method does not reflect how animals would react to much lower doses of a chemical, a dose-response assessment normally requires extrapolation from high to low doses using mathematical modeling that incorporates to varying degrees information about physiologic processes in the body (NRC 1983).

A central problem with the low-dose extrapolation models is that they all too often fit the data from animal bioassays equally well, and it is not possible to determine their validity based on goodness of fit. Several models may fit experimental data equally well, but they may not all be equally plausible biologically. The dose-response curves derived from different models diverge substantially in the dose range of interest (NRC 1983). Therefore, low-dose extrapolation is more than a curve-fitting process and considerations of biological plausibility of the models must be taken into account before choosing the best model for a particular set of data.

2.6.4 Uncertainties in Risk Characterization

Uncertainties in the risk characterization can stem from the inherent uncertainties in the data evaluation, the exposure assessment process, including any modeling of exposure point concentrations in secondary media from primary media, and the toxicity assessment process. The individual uncertainties in these respective processes are addressed previously, in the previous sections.

Uncertainty may also stem from the inclusion of chemicals that are detected at low frequencies. Chemicals that are infrequently detected may be artifacts in the data due to sampling, analytical, or other problems. Therefore, these chemicals may not be related to site operations or disposal practices. When considering Site 16 data in the context of detection frequency, it is evident that several COPCs in ground water may not be site related. 1,4-Dichlorobenzene (one detect), chloroform (two detects), and vinyl chloride (two detects) were not detected at greater than 5 percent (out of a significant sample size of 41). Inclusion of these chemicals in the quantitative risk estimates is likely to bias the results high because these chemicals may not truly be site-related.

Uncertainty may also stem from the exclusion of chemicals that are detected at levels consistent with or below background. While exclusion of such chemicals may bias the calculated risk low, chemicals that are present naturally or due to widespread anthropogenic use (e.g., polycyclic aromatic hydrocarbons [PAHs]) may not be site-related. In this assessment, only arsenic in ground water was excluded based on comparison to background. Arsenic is a naturally occurring element and is present at the site at levels consistent with background. Inorganic soil background values were not available for the site and, therefore, arsenic was treated as a COPC in soil.

The uncertainty associated with the background screening of arsenic in ground water is bounded by the conservative nature of the risk-based screening value (PRG). The RME EPC value of 2.8 µg/L is higher than the risk-based screening value; however, it should be noted that the risk based screening value is quite conservative. The risk-based screening value (tap water PRG) is based on the assumption that ground water will be ingested regularly in a residential setting as

tap water. This is very conservative for Site 16, given that ground water is not used as potable water.

In addition, as noted in Subsection 2.1.4, arsenic was detected in only 2 of 42 ground water samples. The maximum (and average detected value) of arsenic detected in ground-water is 2.8 μg/L, which is below the background value of 6.4 μg/L. Therefore, the uncertainty associated with the exclusion of arsenic as a ground water COPC is minimal.

Uncertainties associated with the probability of adverse impacts to human health can also be evaluated by examining the relative risk estimated for CTE and upper bound RME scenarios. This type of simple probability analysis is often useful to risk managers who must balance baseline risk estimates with the expected costs and benefits of remedial activities.

2.7 SUMMARY AND CONCLUSIONS

The risk assessment is conducted to assess potential non-carcinogenic effects and cancer risks from residential, recreational, and commercial/industrial scenarios for environmental media of concern at Site 16. Soil (surface and total), seep water, and ground water were assessed for potential human exposure as depicted in Figure ES-2 and Table ES-1.

2.7.1 Non-Carcinogenic Risks

Non-carcinogenic risks exceed the U.S. EPA threshold of 1.0 for residents (adult and child) and construction workers; non-carcinogenic risks do not exceed 1.0 for recreational users (adult and child) and commercial worker. The risks exceeding the threshold are driven entirely by the ground water exposure pathways.

Cumulative HIs are calculated as less than the 1.0 threshold for surface soil (commercial workers, adult recreational user, and child recreational user) and total soil (construction workers, child residents, and adult residents). Cumulative HIs are also less than the 1.0 threshold for seep water (adult recreational user, child recreational user) exposure. Thus, non-carcinogenic risk for all receptors to soil and seep water is acceptable.

Cumulative HIs for ground water (construction workers, child residents, and adult residents) are calculated above the 1.0 threshold. COPCs with HQs above 1.0 in ground water are manganese and trichloroethene. A breakdown by target organ indicates that the central nervous system, liver, and kidney exceed the 1.0 threshold indicating unacceptable non-carcinogenic risks for residential and construction worker exposure to ground water.

2.7.2 Carcinogenic Risks

Cumulative cancer risks for residential receptors exceeded U.S. EPA's "acceptable risk range" of 10^{-6} to 10^{-4} for the RME scenario only, driven by the ground-water exposure pathways. Combined adult and child resident risks for ground-water are 2.1×10^{-3} and 6.1×10^{-5} for RME and CTE, respectively. Cumulative cancer risks for total soil are within U.S. EPA's "acceptable risk

range" of 10^{-6} to 10^{-4}. The combined adult and child resident risks for total soil are 3.8×10^{-5} and 2.0×10^{-6} for RME and CTE, respectively. The only residential COPCs with risks greater than 10^{-5} are trichloroethene, and vinyl chloride in ground water. Therefore, there are no concerns for cancer effects for residential exposure to total soil.

There are no cancer risks that exceeded U.S. EPA's "acceptable risk range" of 10^{-6} to 10^{-4} for recreational users. The only COPCs with risks greater than 10^{-6} in surface soil are dioxin, arsenic, benzo(a)pyrene, and dibenz(a,h)anthracene. RME cumulative risks for the adult and child recreational users are 6.4×10^{-6} and 7.4×10^{-6}, respectively. Therefore, there are no concerns for cancer effects for recreational users.

There are no cancer risks that exceeded U.S. EPA's "acceptable risk range" of 10^{-6} to 10^{-4} for industrial receptors. Both the construction and commercial workers had cumulative risks exceeding 10^{-6}. Risks are calculated as 7.3×10^{-6} for RME and 2.7×10^{-7} for CTE for construction workers. Risks are calculated as 6.2×10^{-6} for RME and 4.3×10^{-7} for CTE for commercial workers. In both cases, cumulative risks are below 10^{-5}. Therefore, there are no concerns for cancer effects for construction or commercial workers.

2.7.3 Lead Risks

In addition, lead is evaluated for total soil, ground water and seep water. Blood lead levels are the indicators of excess lead exposure in humans. Modeled blood level results are compared to the established cutoff value or acceptable threshold of 10 µg lead/dL. This blood level is considered protective of human health for children. Lead modeling is performed for residential, recreational, and worker scenarios. Blood lead level modeling results are below 10 µg lead/dL blood for the child resident scenario, indicating that there is no concern for adverse effects from site exposure to lead in soils and ground water. This scenario provides the most conservative estimate of risks for lead and is, therefore, protective of recreational and worker exposure scenarios. However, potential risks from recreational exposure are also modeled using conservative residential assumptions for soil and conservative recreational assumptions for seep water, indicating no unacceptable risks. Likewise the construction worker scenario is evaluated for exposure to total lead in soil; the results indicate no unacceptable risks.

3. CONCLUSIONS

EA Engineering, Science, and Technology prepared this Human Health Risk Assessment (HHRA) for the U.S. Navy Engineering Field Activity Northeast (EFANE) to assess potential risks associated with human exposure to Site 16. This HHRA is submitted as an appendix to the Phase I Remedial Investigation (RI) for Site 16.

Risks were evaluated for site soil, seep water, and ground water for potential human receptors. Receptor-specific pathways are detailed in the conceptual site model presented in Figure ES-2 and Table ES-1. Non-carcinogenic risks, carcinogenic risks, and potential lead exposure risks were all evaluated. Potential risks associated with ground-water exposure for the residential (adult and child) scenario (Table 10.1) and the construction worker scenario (Table 10.4) were unacceptable for non-carcinogenic risk and were unacceptable for the residential scenario for carcinogenic risk. These risks were driven by manganese and trichloroethene in ground water.

Potential risks for all other evaluated media for all evaluated receptors were acceptable. Therefore, there are no concerns for human exposure to site soil and seep water. Blood-lead level modeling indicated that exposure to lead is not of concern at the site for any of the evaluated receptors in any of the evaluated media.

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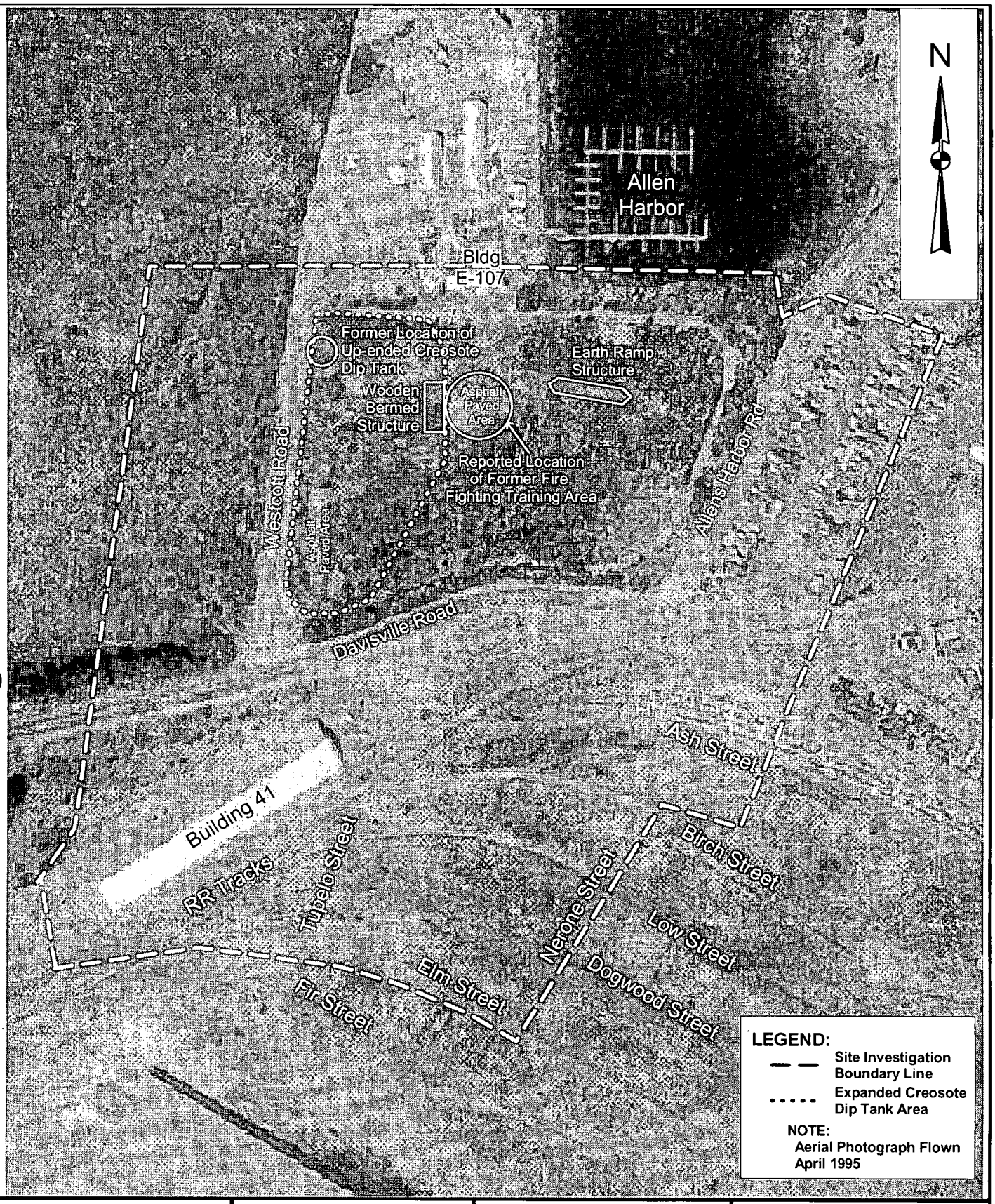

EA ENGINEERING, SCIENCE, AND TECHNOLOGY

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EFANE
IR Program Site 16
Human Health Risk Assessment
Former NCBC Davisville Facility
North Kingstown, Rhode Island

SITE MAP

FIGURE ES-1

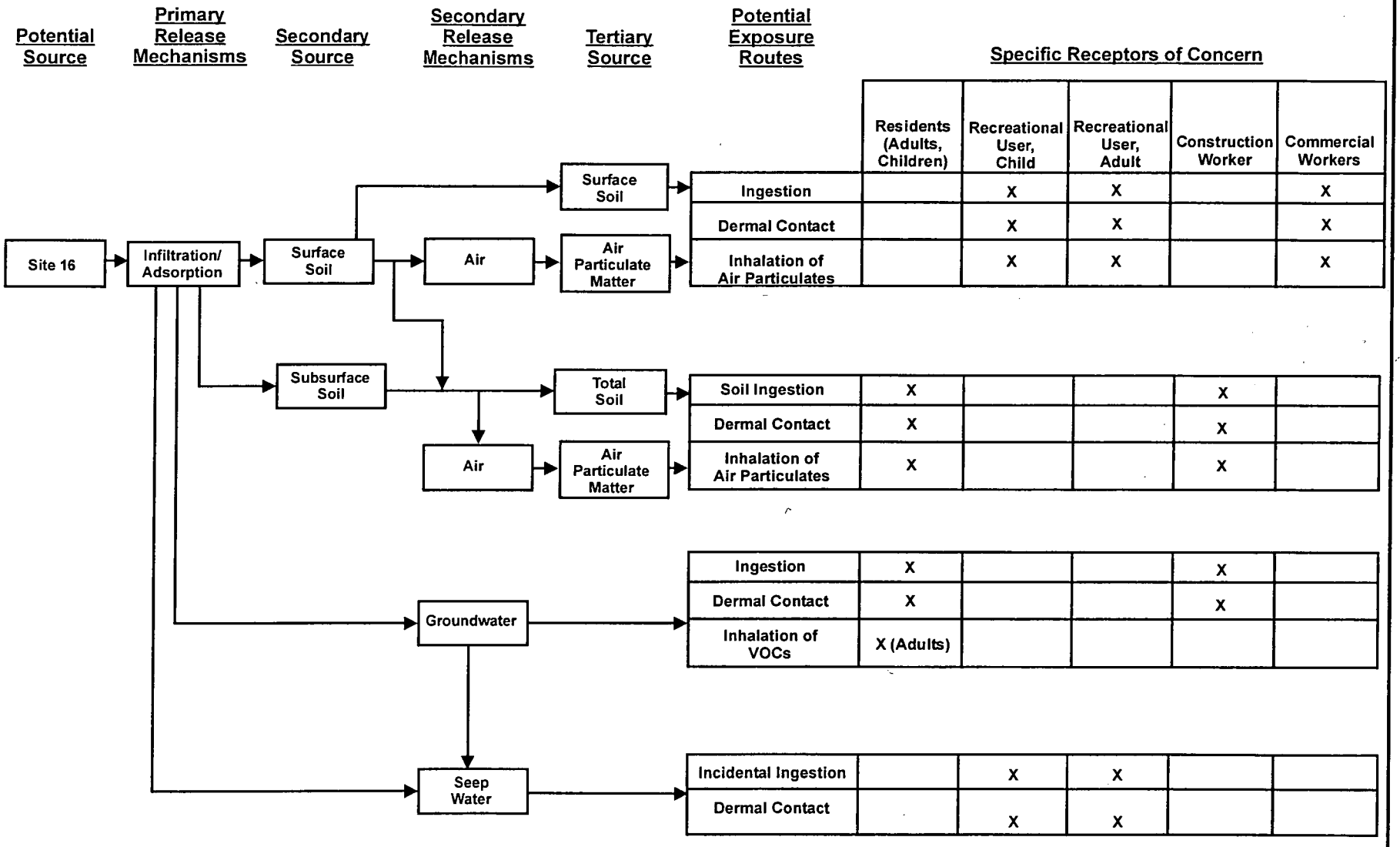

Figure ES-2. Human Health Conceptual Site Model: IR Program Site 16, NCBC Davisville.

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Table ES-1
Selection of Exposure Pathways
Davisville Site 16

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current	Surface Soil	Surface Soil	Site 16	Recreational User	Adult	Ingestion	On-Site	Quant	Recreational adults are exposed to surface soil.
						Dermal	On-Site	Quant	Recreational adults are exposed to surface soil.
					Child	Ingestion	On-Site	Quant	Recreational children are exposed to surface soil.
						Dermal	On-Site	Quant	Recreational children are exposed to surface soil.
		Air	Dust from Soil	Recreational User	Adult	Inhalation-Dust	On-Site	Quant	Recreational adults may inhale dust from surface soil.
					Child	Inhalation-Dust	On-Site	Quant	Recreational children may inhale dust from surface soil.
	Surface Water	Seep	Site 16	Recreational User	Adult	Ingestion	On-Site	Quant	Recreational adults may come in contact with seeps.
						Dermal	On-Site	Quant	Recreational adults may come in contact with seeps.
					Child	Ingestion	On-Site	Quant	Recreational children may come in contact with seeps.
						Dermal	On-Site	Quant	Recreational children may come in contact with seeps.
Future	Surface Soil	Surface Soil	Site 16	Commercial Worker	Adult	Ingestion	On-Site	Quant	Future commercial workers may be exposed to surface soil.
						Dermal	On-Site	Quant	Future commercial workers may be exposed to surface soil.
			Site 16	Recreational User	Adult	Ingestion	On-Site	Quant	Same exposure assumptions for current and future exposures.
						Dermal	On-Site	Quant	Same exposure assumptions for current and future exposures.
					Child	Ingestion	On-Site	Quant	Same exposure assumptions for current and future exposures.
						Dermal	On-Site	Quant	Same exposure assumptions for current and future exposures.
		Air	Dust from Soil	Commercial Worker	Adult	Inhalation-Dust	On-Site	Quant	Future commercial workers may be exposed to dust from surface soil.
			Dust from Soil	Recreational User	Adult	Inhalation-Dust	On-Site	Quant	Same exposure assumptions for current and future exposures.
					Child	Inhalation-Dust	On-Site	Quant	Same exposure assumptions for current and future exposures.
	Total Soil	Total Soil	Site 16	Construction Worker	Adult	Ingestion	On-Site	Quant	Future construction workers may come in contact with soil during construction activities.
						Dermal	On-Site	Quant	Future construction workers may come in contact with soil during construction activities.

Table ES-1 continued
Selection of Exposure Pathways
Davisville Site 16

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Future	Total Soil	Total Soil	Site 16	Resident	Adult	Ingestion	On-Site	Quant	Future residents may be exposed to surface and subsurface soil.
						Dermal	On-Site	Quant	Future residents may be exposed to surface and subsurface soil.
					Child	Ingestion	On-Site	Quant	Future residents may be exposed to surface and subsurface soil.
						Dermal	On-Site	Quant	Future residents may be exposed to surface and subsurface soil.
		Air	Dust from Soil	Construction Worker	Adult	Inhalation-Dust	On-Site	Quant	Future construction workers may inhale dust from soil during construction activities.
				Resident	Adult	Inhalation-Dust	On-Site	Quant	Future residents may inhale dust from surface and subsurface soil.
					Child	Inhalation-Dust	On-Site	Quant	Future residents may inhale dust from surface and subsurface soil.
	Ground Water	Ground Water	Tap Water	Resident	Adult	Ingestion	On-Site	Quant	Future residents may ingest ground water.
						Dermal	On-Site	Qual	Future residents may bathe in ground water.
						Inhalation	On-Site	Qual	Future residents may be exposed to VOCs during showering.
					Child	Ingestion	On-Site	Quant	Future residents may ingest ground water.
						Dermal	On-Site	Qual	Future residents may bathe in ground water.
				Construction Worker	Adult	Ingestion	On-Site	Qual	Future construction workers may come in contact with ground water during construction activities.
						Dermal	On-Site	Qual	Future construction workers may come in contact with ground water during construction activities.
	Surface Water	Seep	Site 16	Recreational User	Adult	Ingestion	On-Site	Quant	Same exposure assumptions for current and future exposures.
						Dermal	On-Site	Quant	Same exposure assumptions for current and future exposures.
					Child	Ingestion	On-Site	Quant	Same exposure assumptions for current and future exposures.
						Dermal	On-Site	Quant	Same exposure assumptions for current and future exposures.

Table 1
Selection of Exposure Pathways
Davisville Site 16

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current	Surface Soil	Surface Soil	Site 16	Recreational User	Adult	Ingestion	On-Site	Quant	Recreational adults are exposed to surface soil.
						Dermal	On-Site	Quant	Recreational adults are exposed to surface soil.
					Child	Ingestion	On-Site	Quant	Recreational children are exposed to surface soil.
						Dermal	On-Site	Quant	Recreational children are exposed to surface soil.
		Air	Dust from Soil	Recreational User	Adult	Inhalation-Dust	On-Site	Quant	Recreational adults may inhale dust from surface soil.
					Child	Inhalation-Dust	On-Site	Quant	Recreational children may inhale dust from surface soil.
	Surface Water	Seep	Site 16	Recreational User	Adult	Ingestion	On-Site	Quant	Recreational adults may come in contact with seeps.
						Dermal	On-Site	Quant	Recreational adults may come in contact with seeps.
					Child	Ingestion	On-Site	Quant	Recreational children may come in contact with seeps.
						Dermal	On-Site	Quant	Recreational children may come in contact with seeps.
Future	Surface Soil	Surface Soil	Site 16	Commercial Worker	Adult	Ingestion	On-Site	Quant	Future commercial workers may be exposed to surface soil.
						Dermal	On-Site	Quant	Future commercial workers may be exposed to surface soil.
			Site 16	Recreational User	Adult	Ingestion	On-Site	Quant	Same exposure assumptions for current and future exposures.
						Dermal	On-Site	Quant	Same exposure assumptions for current and future exposures.
					Child	Ingestion	On-Site	Quant	Same exposure assumptions for current and future exposures.
						Dermal	On-Site	Quant	Same exposure assumptions for current and future exposures.
		Air	Dust from Soil	Commercial Worker	Adult	Inhalation-Dust	On-Site	Quant	Future commercial workers may be exposed to dust from surface soil.
			Dust from Soil	Recreational User	Adult	Inhalation-Dust	On-Site	Quant	Same exposure assumptions for current and future exposures.
					Child	Inhalation-Dust	On-Site	Quant	Same exposure assumptions for current and future exposures.
	Total Soil	Total Soil	Site 16	Construction Worker	Adult	Ingestion	On-Site	Quant	Future construction workers may come in contact with soil during construction activities.
						Dermal	On-Site	Quant	Future construction workers may come in contact with soil during construction activities.

Table 1 continued
Selection of Exposure Pathways
Davisville Site 16

Scenario Timeframe	Medium	Exposure Medium	Exposure Point	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Future	Total Soil	Total Soil	Site 16	Resident	Adult	Ingestion	On-Site	Quant	Future residents may be exposed to surface and subsurface soil.
						Dermal	On-Site	Quant	Future residents may be exposed to surface and subsurface soil.
					Child	Ingestion	On-Site	Quant	Future residents may be exposed to surface and subsurface soil.
						Dermal	On-Site	Quant	Future residents may be exposed to surface and subsurface soil.
		Air	Dust from Soil	Construction Worker	Adult	Inhalation-Dust	On-Site	Quant	Future construction workers may inhale dust from soil during construction activities.
				Resident	Adult	Inhalation-Dust	On-Site	Quant	Future residents may inhale dust from surface and subsurface soil.
					Child	Inhalation-Dust	On-Site	Quant	Future residents may inhale dust from surface and subsurface soil.
	Ground Water	Ground Water	Tap Water	Resident	Adult	Ingestion	On-Site	Quant	Future residents may ingest ground water.
						Dermal	On-Site	Qual	Future residents may bathe in ground water.
						Inhalation	On-Site	Qual	Future residents may be exposed to VOCs during showering.
					Child	Ingestion	On-Site	Quant	Future residents may ingest ground water.
						Dermal	On-Site	Qual	Future residents may bathe in ground water.
				Construction Worker	Adult	Ingestion	On-Site	Qual	Future construction workers may come in contact with ground water during construction activities.
						Dermal	On-Site	Qual	Future construction workers may come in contact with ground water during construction activities.
	Surface Water	Seep	Site 16	Recreational User	Adult	Ingestion	On-Site	Quant	Same exposure assumptions for current and future exposures.
						Dermal	On-Site	Quant	Same exposure assumptions for current and future exposures.
					Child	Ingestion	On-Site	Quant	Same exposure assumptions for current and future exposures.
						Dermal	On-Site	Quant	Same exposure assumptions for current and future exposures.

TABLE 2.1
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
DAVISVILLE - SITE 16 - SURFACE SOIL - RESIDENTIAL

Scenario Timeframe: Current/Future
Medium: Surface Soil
Exposure Medium: Surface Soil
Exposure Point: Davisville Site 16

CAS Number	Chemical	Minimum [1] Concentration	Minimum Qualifier	Maximum [1] Concentration	Maximum Qualifier	Units	Location of Maximum Concentraion	Detection Frequency	Range of Detection Limits	Concentration [2] Used for Screening	Background Value	Screening [3] Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for [4] Contaminant Deletion or Selection
							Dioxin/Furans									
TEQ	TCDD-TEQ	0.0000026		0.00004521		mg/kg	SB16-25-0-2	8/8		0.00004521	N/A	3.9E-06 C	N/A	N/A	Yes	ASL
							Inorganics									
7429-90-5	ALUMINUM	2570		8590		mg/kg	SB16-28-0-2	9/9	1.48 - 2.9	8590	N/A	7.6E+03 N	N/A	N/A	Yes	ASL
7440-38-2	ARSENIC	1.3		4		mg/kg	SB16-28-0-2	7/9	0.18 - 0.37	4	N/A	3.9E-01 C	N/A	N/A	Yes	ASL
7440-39-3	BARIUM	14.8		40.6		mg/kg	SB16-26-0-2	9/9	0.04 - 1.5	40.6	N/A	5.4E+02 N	N/A	N/A	No	BSL
7440-41-7	BERYLLIUM	0.33		0.64		mg/kg	SB16-28-0-2	9/9	0.04 - 0.07	0.64	N/A	1.5E+01 N	N/A	N/A	No	BSL
7440-43-9	CADMIUM	0.35	J	0.56		mg/kg	SB16-24-0-2	2/9	0.04 - 0.32	0.56	N/A	3.7E+00 N	N/A	N/A	No	BSL
7440-70-2	CALCIUM METAL	282		1200		mg/kg	SB16-23-0-2	8/9	0.56 - 4.4	1200	N/A	N/A	N/A	N/A	No	NUT
7440-47-3	CHROMIUM	2.9		11.6		mg/kg	SB16-28-0-2	9/9	0.07 - 0.68	11.6	N/A	3.0E+01 C	N/A	N/A	No	BSL
7440-48-4	COBALT	2		7.8		mg/kg	SB16-28-0-2	9/9	0.27 - 1.3	7.8	N/A	4.7E+02 N	N/A	N/A	No	BSL
7440-50-8	COPPER	8.2	J	40.2	J	mg/kg	SB16-23-0-2	9/9	0.17 - 0.84	40.2	N/A	2.9E+02 N	N/A	N/A	No	BSL
7439-89-6	IRON	5930		21200		mg/kg	SB16-28-0-2	9/9	0.31 - 2.7	21200	N/A	N/A	N/A	N/A	No	NUT
7439-92-1	LEAD	13.45		98.4		mg/kg	SB16-24-0-2	9/9	0.13 - 0.2	98.4	N/A	4.0E+02	N/A	N/A	No	BSL
7439-95-4	MAGNESIUM	540		2140		mg/kg	SB16-28-0-2	9/9	1.17 - 6.7	2140	N/A	N/A	N/A	N/A	No	NUT
7439-96-5	MANGANESE	84.4	J	248	J	mg/kg	SB16-28-0-2	9/9	0.12 - 0.2	248	N/A	1.8E+02 N	N/A	N/A	Yes	ASL
7439-97-6	MERCURY (INORGANIC)	0.11		0.11		mg/kg	SB16-25-0-2	1/9	0.01 - 0.05	0.11	N/A	2.3E+00 N	N/A	N/A	No	BSL
7440-02-0	NICKEL	2.1475	_ / U	11		mg/kg	SB16-28-0-2	6/9	1.07 - 2	11	N/A	1.6E+02 N	N/A	N/A	No	BSL
7440-09-7	POTASSIUM	381		685	J	mg/kg	SB16-21-0-2	8/9	17.2 - 41.03	685	N/A	N/A	N/A	N/A	No	NUT
7782-49-2	SELENIUM	0.88		0.88		mg/kg	SB16-28-0-2	1/9	0.27 - 0.45	0.88	N/A	3.9E+01 N	N/A	N/A	No	BSL
7440-22-4	SILVER	0.32	J	0.32	J	mg/kg	SB16-25-0-2	1/9	0.2 - 0.33	0.32	N/A	3.9E+01 N	N/A	N/A	No	BSL
7440-23-5	SODIUM	17.36	U / _	82		mg/kg	SB16-23-0-2	5/9	1.29 - 5.8	82	N/A	N/A	N/A	N/A	No	NUT
7440-28-0	THALLIUM	0.65	J	0.65	J	mg/kg	SB16-27-0-2	1/9	0.25 - 0.4	0.65	N/A	5.2E-01 N	N/A	N/A	Yes	ASL
7440-62-2	VANADIUM	4.7		16.1		mg/kg	SB16-28-0-2	9/9	0.36 - 0.66	16.1	N/A	5.5E+01 N	N/A	N/A	No	BSL
7440-66-6	ZINC	29.8		85.3	J	mg/kg	SB16-28-0-2	9/9	0.14 - 1.4	85.3	N/A	2.3E+03 N	N/A	N/A	No	BSL
							PAHs									
91-57-6	2-METHYLNAPHTHALENE	0.03		0.558	U / _	mg/kg	SB16-21-0-2	2/25	0.02 - 1.11	0.558	N/A	5.6E+00 N	N/A	N/A	No	BSL
83-32-9	ACENAPHTHENE	0.014		2.4	J	mg/kg	28-SB-01B	6/29	0.02 - 1.11	2.4	N/A	3.7E+02 N	N/A	N/A	No	BSL
208-96-8	ACENAPHTHYLENE	0.012		0.77	U / _	mg/kg	SB16-21-0-2	4/29	0.02 - 1.6	0.77	N/A	3.7E+02 N	N/A	N/A	No	BSL
120-12-7	ANTHRACENE	0.005	J	4.6	J	mg/kg	28-SB-01B	11/29	0.02 - 1.11	4.6	N/A	2.2E+03 N	N/A	N/A	No	BSL
56-55-3	BENZ[A]ANTHRACENE	0.004		4.45		mg/kg	SB16-21-0-2	18/29	0.02 - 1.11	4.45	N/A	6.2E-01 C	N/A	N/A	Yes	ASL
50-32-8	BENZO[A]PYRENE	0.004	J	2.35		mg/kg	SB16-21-0-2	19/29	0.02 - 1.11	2.35	N/A	6.2E-02 C	N/A	N/A	Yes	ASL
205-99-2	BENZO[B]FLUORANTHENE	0.008		7.4		mg/kg	SB16-21-0-2	21/29	0.02 - 1.11	7.4	N/A	6.2E-01 C	N/A	N/A	Yes	ASL
191-24-2	BENZO[G,H,I]PERYLENE	0.003	J	1.05		mg/kg	SB16-21-0-2	17/29	0.02 - 1.11	1.05	N/A	2.3E+02 N	N/A	N/A	No	BSL
207-08-9	BENZO[K]FLUORANTHENE	0.003		1.8		mg/kg	SB16-21-0-2	15/29	0.02 - 1.11	1.8	N/A	6.2E+00 C	N/A	N/A	No	BSL
218-01-9	CHRYSENE	0.015	J	4.75		mg/kg	SB16-21-0-2	20/29	0.02 - 1.11	4.75	N/A	6.2E+01 C	N/A	N/A	No	BSL
53-70-3	DIBENZ[A,H]ANTHRACENE	0.009		0.75	U / _	mg/kg	SB16-21-0-2	10/29	0.002 - 1.11	0.75	N/A	6.2E-02 C	N/A	N/A	Yes	ASL
206-44-0	FLUORANTHENE	0.017	J	4.305		mg/kg	SB16-21-0-2	20/29	0.02 - 1.11	4.305	N/A	2.3E+02 N	N/A	N/A	No	BSL
86-73-7	FLUORENE	0.005	J	0.5925	_ / UJ	mg/kg	SB16-21-0-2	6/29	0.007 - 1.11	0.5925	N/A	2.6E+02 N	N/A	N/A	No	BSL
193-39-5	INDENO[1,2,3-C,D]PYRENE	0.002	J	2.3		mg/kg	SB16-21-0-2	17/29	0.02 - 1.11	2.3	N/A	6.2E-01 C	N/A	N/A	Yes	ASL
91-20-3	NAPHTHALENE	0.015		0.557	U / _	mg/kg	SB16-21-0-2	3/29	0.02 - 1.11	0.557	N/A	5.6E+00 N	N/A	N/A	No	BSL
85-01-8	PHENANTHRENE	0.006	J	0.92		mg/kg	SB16-28-0-2	17/29	0.02 - 1.11	0.92	N/A	2.3E+02 N	N/A	N/A	No	BSL
129-00-0	PYRENE	0.013	J	6.4		mg/kg	SB16-21-0-2	21/29	0.02 - 1.11	6.4	N/A	2.3E+02 N	N/A	N/A	No	BSL

TABLE 2.1
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
DAVISVILLE - SITE 16 - SURFACE SOIL - RESIDENTIAL

Scenario Timeframe: Current/Future
Medium: Surface Soil
Exposure Medium: Surface Soil
Exposure Point: Davisville Site 16

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentraion	Detection Frequency	Range of Detection Limits	Concentration (2) Used for Screening	Background Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
							PCBs									
11096-82-5	AROCLOR-1260	0.014	U / _	0.014	U / _	mg/kg	SB16-21-0-2	1/9	0.018 - 0.036	0.014	N/A	2.2E-01 C	N/A	N/A	No	BSL
							Pesticides									
319-84-6	ALPHA-HCH	0.0024	J	0.0024	J	mg/kg	SB16-28-0-2	1/8	0.0018 - 0.0021	0.0024	N/A	9.0E-02 C	N/A	N/A	No	BSL
50-29-3	DDT	0.0037		0.0037		mg/kg	SB16-26-0-2	1/8	0.0034 - 0.0042	0.0037	N/A	1.7E+00 C	N/A	N/A	No	BSL
5103-74-2	GAMMA-CHLORDANE	0.0029	J	0.0029	J	mg/kg	SB16-21-0-2	1/9	0.0018 - 0.0021	0.0029	N/A	1.6E+00 C	N/A	N/A	No	BSL
							Semivolatiles									
117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.036	J	0.106	U / J	mg/kg	EBS-28-SB08-0-2	2/25	0.13 - 0.44	0.106	N/A	3.5E+01 C	N/A	N/A	No	BSL
86-74-8	CARBAZOLE	0.066	J	0.405	J	mg/kg	SB16-21-0-2	2/25	0.069 - 0.44	0.405	N/A	2.4E+01 C	N/A	N/A	No	BSL
84-74-2	DIBUTYLPHTHALATE	0.04	J	0.056	J	mg/kg	EBS-28-SB05-0-2	6/25	0.093 - 0.44	0.056	N/A	6.1E+02 N	N/A	N/A	No	BSL
							Volatiles									
78-93-3	2-BUTANONE	0.01		0.012		mg/kg	SB16-27-0-2	3/26	0.004 - 0.011	0.012	N/A	7.3E+02 N	N/A	N/A	No	BSL
67-64-1	ACETONE	0.015	J	3.7	J	mg/kg	EBS-28-SB04-0-2	14/26	0.003 - 0.011	3.7	N/A	1.6E+02 N	N/A	N/A	No	BSL
75-09-2	METHYLENE CHLORIDE	0.004	J	0.0045	J / U	mg/kg	EBS-28-SB07-0-2	3/26	0.001 - 0.01	0.0045	N/A	8.9E+00 C	N/A	N/A	No	BSL
108-88-3	TOLUENE	0.006	J	0.006	J	mg/kg	EBS-28-SB15-0-2	1/26	0.001 - 0.01	0.006	N/A	5.2E+02	N/A	N/A	No	BSL

(1) Minimum/maximum detected concentration.
(2) Maximum concentration used as screening value.
(3) Screening Toxicity Value - Taken from USEPA Region IX Preliminary Remediation Goals (PRGs) Table, USEPA, November 2000. For non-carcinogens, value shown is equal to 1/10 the Residential PRG. For carcinogens the value shown is equal to the Residential PRG.
(4) Rationale Codes

Selection Reason: Above Screening Toxicity and Background Levels (ASL)
Deletion Reason: Essential Nutrient (NUT)
Below Screening Toxicity Level (BSL)

Definitions:
N/A = Not Applicable
ND = No Data

COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
C = Carcinogenic
N = Non-Carcinogenic

TABLE 2.2
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
DAVISVILLE - SITE 16 - SURFACE SOIL - INDUSTRIAL

Scenario Timeframe: Current/Future
Medium: Surface Soil
Exposure Medium: Surface Soil
Exposure Point: Davisville Site 16

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentraion	Detection Frequency	Range of Detection Limits	Concentration (2) Used for Screening	Background Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
							Dioxin/Furans									
TEQ	**TCDD-TEQ**	**0.0000026**		**0.00004521**		**mg/kg**	**SB16-25-0-2**	**8/8**		**0.00004521**	**N/A**	**2.7E-05 C**	**N/A**	**N/A**	**Yes**	**ASL**
							Inorganics									
7429-90-5	ALUMINUM	2570		8590		mg/kg	SB16-28-0-2	9/9	1.48 - 2.9	8590	N/A	1.0E+05	N/A	N/A	No	BSL
7440-38-2	**ARSENIC**	**1.3**		**4**		**mg/kg**	**SB16-28-0-2**	**7/9**	**0.18 - 0.37**	**4**	**N/A**	**2.7E+00 C**	**N/A**	**N/A**	**Yes**	**ASL**
7440-39-3	BARIUM	14.8		40.6		mg/kg	SB16-26-0-2	9/9	0.04 - 1.5	40.6	N/A	1.0E+05	N/A	N/A	No	BSL
7440-41-7	BERYLLIUM	0.33		0.64		mg/kg	SB16-28-0-2	9/9	0.04 - 0.07	0.64	N/A	2.2E+03 C	N/A	N/A	No	BSL
7440-43-9	CADMIUM	0.35	J	0.56		mg/kg	SB16-24-0-2	2/9	0.04 - 0.32	0.56	N/A	8.1E+01 N	N/A	N/A	No	BSL
7440-70-2	CALCIUM METAL	282		1200		mg/kg	SB16-23-0-2	8/9	0.56 - 4.4	1200	N/A	N/A	N/A	N/A	No	NUT
7440-47-3	CHROMIUM	2.9		11.6		mg/kg	SB16-28-0-2	9/9	0.07 - 0.68	11.6	N/A	6.4E+01 C	N/A	N/A	No	BSL
7440-48-4	COBALT	2		7.8		mg/kg	SB16-28-0-2	9/9	0.27 - 1.3	7.8	N/A	1.0E+05	N/A	N/A	No	BSL
7440-50-8	COPPER	8.2	J	40.2	J	mg/kg	SB16-23-0-2	9/9	0.17 - 0.84	40.2	N/A	7.6E+03 N	N/A	N/A	No	BSL
7439-89-6	IRON	5930		21200		mg/kg	SB16-28-0-2	9/9	0.31 - 2.7	21200	N/A	N/A	N/A	N/A	No	NUT
7439-92-1	LEAD	13.45		98.4		mg/kg	SB16-24-0-2	9/9	0.13 - 0.2	98.4	N/A	7.5E+02	N/A	N/A	No	BSL
7439-95-4	MAGNESIUM	540		2140		mg/kg	SB16-28-0-2	9/9	1.17 - 6.7	2140	N/A	N/A	N/A	N/A	No	NUT
7439-96-5	MANGANESE	84.4	J	248	J	mg/kg	SB16-28-0-2	9/9	0.12 - 0.2	248	N/A	3.2E+03 N	N/A	N/A	No	BSL
7439-97-6	MERCURY (INORGANIC)	0.11		0.11		mg/kg	SB16-25-0-2	1/9	0.01 - 0.05	0.11	N/A	6.1E+01 N	N/A	N/A	No	BSL
7440-02-0	NICKEL	2.1475	_ / U	11		mg/kg	SB16-28-0-2	6/9	1.07 - 2	11	N/A	4.1E+03 N	N/A	N/A	No	BSL
7440-09-7	POTASSIUM	381		685	J	mg/kg	SB16-21-0-2	8/9	17.2 - 41.03	685	N/A	N/A	N/A	N/A	No	NUT
7782-49-2	SELENIUM	0.88		0.88		mg/kg	SB16-28-0-2	1/9	0.27 - 0.45	0.88	N/A	1.0E+03 N	N/A	N/A	No	BSL
7440-22-4	SILVER	0.32	J	0.32	J	mg/kg	SB16-25-0-2	1/9	0.2 - 0.33	0.32	N/A	1.0E+03 N	N/A	N/A	No	BSL
7440-23-5	SODIUM	17.36	U / _	82		mg/kg	SB16-23-0-2	5/9	1.29 - 5.8	82	N/A	N/A	N/A	N/A	No	NUT
7440-28-0	THALLIUM	0.65	J	0.65	J	mg/kg	SB16-27-0-2	1/9	0.25 - 0.4	0.65	N/A	1.3E+01 N	N/A	N/A	No	BSL
7440-62-2	VANADIUM	4.7		16.1		mg/kg	SB16-28-0-2	9/9	0.36 - 0.66	16.1	N/A	1.4E+03 N	N/A	N/A	No	BSL
7440-66-6	ZINC	29.8		85.3	J	mg/kg	SB16-28-0-2	9/9	0.14 - 1.4	85.3	N/A	1.0E+05	N/A	N/A	No	BSL
							PAHs									
91-57-6	2-METHYLNAPHTHALENE	0.03		0.558	U / _	mg/kg	SB16-21-0-2	2/25	0.02 - 1.11	0.558	N/A	1.9E+01 N	N/A	N/A	No	BSL
83-32-9	ACENAPHTHENE	0.014		2.4	J	mg/kg	28-SB-01B	6/29	0.02 - 1.11	2.4	N/A	3.8E+03 N	N/A	N/A	No	BSL
208-96-8	ACENAPHTHYLENE	0.012		0.77	U / _	mg/kg	SB16-21-0-2	4/29	0.02 - 1.6	0.77	N/A	3.8E+03 N	N/A	N/A	No	BSL
120-12-7	ANTHRACENE	0.005	J	4.6	J	mg/kg	28-SB-01B	11/29	0.02 - 1.11	4.6	N/A	1.0E+05	N/A	N/A	No	BSL
56-55-3	**BENZ[A]ANTHRACENE**	**0.004**		**4.45**		**mg/kg**	**SB16-21-0-2**	**18/29**	**0.02 - 1.11**	**4.45**	**N/A**	**2.9E+00 C**	**N/A**	**N/A**	**Yes**	**ASL**
50-32-8	**BENZO[A]PYRENE**	**0.004**	**J**	**2.35**		**mg/kg**	**SB16-21-0-2**	**19/29**	**0.02 - 1.11**	**2.35**	**N/A**	**2.9E-01 C**	**N/A**	**N/A**	**Yes**	**ASL**
205-99-2	**BENZO[B]FLUORANTHENE**	**0.008**		**7.4**		**mg/kg**	**SB16-21-0-2**	**21/29**	**0.02 - 1.11**	**7.4**	**N/A**	**2.9E+00 C**	**N/A**	**N/A**	**Yes**	**ASL**
191-24-2	BENZO[G,H,I]PERYLENE	0.003	J	1.05		mg/kg	SB16-21-0-2	17/29	0.02 - 1.11	1.05	N/A	5.4E+03 N	N/A	N/A	No	BSL
207-08-9	BENZO[K]FLUORANTHENE	0.003		1.8		mg/kg	SB16-21-0-2	15/29	0.02 - 1.11	1.8	N/A	2.9E+01 C	N/A	N/A	No	BSL
218-01-9	CHRYSENE	0.015	J	4.75		mg/kg	SB16-21-0-2	20/29	0.02 - 1.11	4.75	N/A	2.9E+02 C	N/A	N/A	No	BSL
53-70-3	**DIBENZ[A,H]ANTHRACENE**	**0.009**		**0.75**	**U / _**	**mg/kg**	**SB16-21-0-2**	**10/29**	**0.002 - 1.11**	**0.75**	**N/A**	**2.9E-01 C**	**N/A**	**N/A**	**Yes**	**ASL**
206-44-0	FLUORANTHENE	0.017	J	4.305		mg/kg	SB16-21-0-2	20/29	0.02 - 1.11	4.305	N/A	3.0E+03 N	N/A	N/A	No	BSL
86-73-7	FLUORENE	0.005	J	0.5925	_ / UJ	mg/kg	SB16-21-0-2	6/29	0.007 - 1.11	0.5925	N/A	3.3E+03 N	N/A	N/A	No	BSL
193-39-5	INDENO[1,2,3-C,D]PYRENE	0.002	J	2.3		mg/kg	SB16-21-0-2	17/29	0.02 - 1.11	2.3	N/A	2.9E+00 C	N/A	N/A	No	BSL
91-20-3	NAPHTHALENE	0.015		0.557	U / _	mg/kg	SB16-21-0-2	3/29	0.02 - 1.11	0.557	N/A	1.9E+01 N	N/A	N/A	No	BSL
85-01-8	PHENANTHRENE	0.006	J	0.92		mg/kg	SB16-28-0-2	17/29	0.02 - 1.11	0.92	N/A	5.4E+03 N	N/A	N/A	No	BSL
129-00-0	PYRENE	0.013	J	6.4		mg/kg	SB16-21-0-2	21/29	0.02 - 1.11	6.4	N/A	5.4E+03 N	N/A	N/A	No	BSL

TABLE 2.2
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
DAVISVILLE - SITE 16 - SURFACE SOIL - INDUSTRIAL

Scenario Timeframe: Current/Future
Medium: Surface Soil
Exposure Medium: Surface Soil
Exposure Point: Davisville Site 16

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentraion	Detection Frequency	Range of Detection Limits	Concentration (2) Used for Screening	Background Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
							PCBs									
11096-82-5	AROCLOR-1260	0.014	U / _	0.014	U / _	mg/kg	SB16-21-0-2	1/9	0.018 - 0.036	0.014	N/A	1.0E+00 C	N/A	N/A	No	BSL
							Pesticides									
319-84-6	ALPHA-HCH	0.0024	J	0.0024	J	mg/kg	SB16-28-0-2	1/8	0.0018 - 0.0021	0.0024	N/A	5.9E-01 C	N/A	N/A	No	BSL
50-29-3	DDT	0.0037		0.0037		mg/kg	SB16-26-0-2	1/8	0.0034 - 0.0042	0.0037	N/A	1.2E+01 C	N/A	N/A	No	BSL
5103-74-2	GAMMA-CHLORDANE	0.0029	J	0.0029	J	mg/kg	SB16-21-0-2	1/9	0.0018 - 0.0021	0.0029	N/A	1.1E+01 C	N/A	N/A	No	BSL
							Semivolatiles									
117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.036	J	0.106	U / J	mg/kg	EBS-28-SB08-0-2	2/25	0.13 - 0.44	0.106	N/A	1.8E+02 C	N/A	N/A	No	BSL
86-74-8	CARBAZOLE	0.066	J	0.405	J	mg/kg	SB16-21-0-2	2/25	0.069 - 0.44	0.405	N/A	1.2E+02 C	N/A	N/A	No	BSL
84-74-2	DIBUTYLPHTHALATE	0.04	J	0.056	J	mg/kg	EBS-28-SB05-0-2	6/25	0.093 - 0.44	0.056	N/A	8.8E+03 N	N/A	N/A	No	BSL
							Volatiles									
78-93-3	2-BUTANONE	0.01		0.012		mg/kg	SB16-27-0-2	3/26	0.004 - 0.011	0.012	N/A	2.8E+03 N	N/A	N/A	No	BSL
67-64-1	ACETONE	0.015	J	3.7	J	mg/kg	EBS-28-SB04-0-2	14/26	0.003 - 0.011	3.7	N/A	6.2E+02 N	N/A	N/A	No	BSL
75-09-2	METHYLENE CHLORIDE	0.004	J	0.0045	J / U	mg/kg	EBS-28-SB07-0-2	3/26	0.001 - 0.01	0.0045	N/A	2.1E+01 C	N/A	N/A	No	BSL
108-88-3	TOLUENE	0.006	J	0.006	J	mg/kg	EBS-28-SB15-0-2	1/26	0.001 - 0.01	0.006	N/A	5.2E+02	N/A	N/A	No	BSL

(1) Minimum/maximum detected concentration.
(2) Maximum concentration used as screening value.
(3) Screening Toxicity Value - Taken from USEPA Region IX Preliminary Remediation Goals (PRGs) Table. USEPA, November 2000. For non-carcinogens, value shown is equal to 1/10 the Industrial PRG. For carcinogens the value shown is equal to the Industrial PRG.
(4) Rationale Codes

Selection Reason: Above Screening Toxicity and Background Levels (ASL)
Deletion Reason: Essential Nutrient (NUT)
Below Screening Toxicity Level (BSL)

Definitions:
N/A = Not Applicable
ND = No Data

COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
C = Carcinogenic
N = Non-Carcinogenic

TABLE 2.3
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
DAVISVILLE - SITE 16 - TOTAL SOIL - RESIDENTIAL

Scenario Timeframe: Current/Future
Medium: Total Soil
Exposure Medium: Total Soil
Exposure Point: Davisville Site 16

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentraion	Detection Frequency	Range of Detection Limits	Concentration (2) Used for Screening	Background Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
							Dioxin/Furans									
TEQ	TCDD-TEQ	0.0000024		0.00049668		mg/kg	SB16-28-4-6	16/16		0.00049668	N/A	3.9E-06 C	N/A	N/A	Yes	ASL
							Inorganics									
7429-90-5	ALUMINUM	2570		12400		mg/kg	SB16-22-6-8	24/24	1.28 - 16.38	12400	N/A	7.6E+03 N	N/A	N/A	Yes	ASL
7440-36-0	ANTIMONY	0.42	UJ / J	8.7		mg/kg	SB16-28-4-6	4/24	0.18 - 2.31	8.7	N/A	3.1E+00 N	N/A	N/A	Yes	ASL
7440-38-2	ARSENIC	1.3		10.6		mg/kg	SB16-28-4-6	19/24	0.15 - 1.98	10.6	N/A	3.9E-01 C	N/A	N/A	Yes	ASL
7440-39-3	BARIUM	10.8		1100		mg/kg	SB16-28-4-6	24/24	0.04 - 1.7	1100	N/A	5.4E+02 N	N/A	N/A	Yes	ASL
7440-41-7	BERYLLIUM	0.27	J	0.71		mg/kg	SB16-22-6-8	24/24	0.04 - 0.07	0.71	N/A	1.5E+01 N	N/A	N/A	No	BSL
7440-43-9	CADMIUM	0.33	J	2.4		mg/kg	SB16-28-4-6	5/24	0.04 - 0.86	2.4	N/A	3.7E+00 N	N/A	N/A	No	BSL
7440-70-2	CALCIUM METAL	282		23400		mg/kg	SB16-28-4-6	19/24	0.45 - 4.9	23400	N/A	N/A	N/A	N/A	No	NUT
7440-47-3	CHROMIUM	2.5		67.2		mg/kg	SB16-28-4-6	23/24	0.06 - 1.32	67.2	N/A	3.0E+01 C	N/A	N/A	No	BSL
7440-48-4	COBALT	2		10.2		mg/kg	MW16-03S-03-05	24/24	0.07 - 1.4	10.2	N/A	4.7E+02 N	N/A	N/A	No	BSL
7440-50-8	COPPER	6.7	J	452	J	mg/kg	SB16-28-4-6	24/24	0.15 - 0.93	452	N/A	2.9E+02 N	N/A	N/A	Yes	ASL
7439-89-6	IRON	5900		54300		mg/kg	SB16-28-4-6	24/24	0.27 - 6.72	54300	N/A	N/A	N/A	N/A	No	NUT
7439-92-1	LEAD	3.95		2650		mg/kg	SB16-28-4-6	24/24	0.12 - 16.4	2650	N/A	4.0E+02	N/A	N/A	Yes	ASL
7439-95-4	MAGNESIUM	525.2875	_ / U	4910		mg/kg	SB16-28-4-6	22/24	1.01 - 12.9	4910	N/A	N/A	N/A	N/A	No	NUT
7439-96-5	MANGANESE	70.25		587	J	mg/kg	SB16-28-4-6	24/24	0.02 - 0.44	587	N/A	1.8E+02 N	N/A	N/A	Yes	ASL
7439-97-6	MERCURY (INORGANIC)	0.04	J / U	0.96		mg/kg	SB16-28-4-6	5/24	0.01 - 0.055	0.96	N/A	2.3E+00 N	N/A	N/A	No	BSL
7440-02-0	NICKEL	2.1475	_ / U	34.5		mg/kg	SB16-28-4-6	19/24	0.11 - 3.94	34.5	N/A	1.6E+02 N	N/A	N/A	No	BSL
7440-09-7	POTASSIUM	381		1100		mg/kg	SB16-23-3-5	19/24	17.2 - 41.03	1100	N/A	N/A	N/A	N/A	No	NUT
7782-49-2	SELENIUM	0.33	J	0.92		mg/kg	MW16-03S-03-05	5/24	0.23 - 2.97	0.92	N/A	3.9E+01 N	N/A	N/A	No	BSL
7440-22-4	SILVER	0.225	J / U	6.8		mg/kg	SB16-28-4-6	7/24	0.1 - 0.9	6.8	N/A	3.9E+01 N	N/A	N/A	No	BSL
7440-23-5	SODIUM	17.36	U / _	596		mg/kg	SB16-28-4-6	16/24	1.12 - 6.4	596	N/A	N/A	N/A	N/A	No	NUT
7440-28-0	THALLIUM	0.65	J	0.65	J	mg/kg	SB16-27-0-2	1/24	0.25 - 3.27	0.65	N/A	5.2E-01 N	N/A	N/A	Yes	ASL
7440-62-2	VANADIUM	4.6		18.9		mg/kg	SB16-22-6-8	24/24	0.09 - 1.32	18.9	N/A	5.5E+01 N	N/A	N/A	No	BSL
7440-66-6	ZINC	17.3	J	1610	J	mg/kg	SB16-28-4-6	24/24	0.12 - 1.5	1610	N/A	2.3E+03 N	N/A	N/A	No	BSL
							PAHs									
91-57-6	2-METHYLNAPHTHALENE	0.03		49		mg/kg	SB16-28-4-6	5/53	0.02 - 2.4	49	N/A	5.6E+00 N	N/A	N/A	Yes	ASL
83-32-9	ACENAPHTHENE	0.014		15.1	J	mg/kg	28-SB-01D	13/61	0.02 - 2.4	15.1	N/A	3.7E+02 N	N/A	N/A	No	BSL
208-96-8	ACENAPHTHYLENE	0.012		0.77	U / _	mg/kg	SB16-21-0-2	7/61	0.02 - 2.4	0.77	N/A	3.7E+02 N	N/A	N/A	No	BSL
120-12-7	ANTHRACENE	0.005	J	4.6	J	mg/kg	28-SB-01B	21/61	0.02 - 2.4	4.6	N/A	2.2E+03 N	N/A	N/A	No	BSL
56-55-3	BENZ[A]ANTHRACENE	0.004		4.45		mg/kg	SB16-21-0-2	32/61	0.02 - 2.4	4.45	N/A	6.2E-01 C	N/A	N/A	Yes	ASL
50-32-8	BENZO[A]PYRENE	0.004	J	2.45	J	mg/kg	28-SB-01D	34/61	0.02 - 2.4	2.45	N/A	6.2E-02 C	N/A	N/A	Yes	ASL
205-99-2	BENZO[B]FLUORANTHENE	0.008		7.4		mg/kg	SB16-21-0-2	36/61	0.02 - 2.4	7.4	N/A	6.2E-01 C	N/A	N/A	Yes	ASL
191-24-2	BENZO[G,H,I]PERYLENE	0.003	J	2.05	J	mg/kg	28-SB-01D	30/61	0.02 - 2.4	2.05	N/A	2.3E+02 N	N/A	N/A	No	BSL
207-08-9	BENZO[K]FLUORANTHENE	0.003		1.8		mg/kg	SB16-21-0-2	26/61	0.02 - 2.4	1.8	N/A	6.2E+00 C	N/A	N/A	No	BSL
218-01-9	CHRYSENE	0.015	J	4.75		mg/kg	SB16-21-0-2	35/61	0.02 - 2.4	4.75	N/A	6.2E+01 C	N/A	N/A	No	BSL
53-70-3	DIBENZ[A,H]ANTHRACENE	0.009		0.75	U / _	mg/kg	SB16-21-0-2	17/61	0.002 - 2.4	0.75	N/A	6.2E-02 C	N/A	N/A	Yes	ASL
206-44-0	FLUORANTHENE	0.017	J	5.5		mg/kg	SB16-28-4-6	35/61	0.02 - 2.4	5.5	N/A	2.3E+02 N	N/A	N/A	No	BSL
86-73-7	FLUORENE	0.005	J	5.1	J	mg/kg	SB16-28-4-6	13/61	0.007 - 2.4	5.1	N/A	2.6E+02 N	N/A	N/A	No	BSL
193-39-5	INDENO[1,2,3-C,D]PYRENE	0.002	J	2.3		mg/kg	SB16-21-0-2	31/61	0.02 - 2.4	2.3	N/A	6.2E-01 C	N/A	N/A	Yes	ASL
91-20-3	NAPHTHALENE	0.006		12		mg/kg	SB16-28-4-6	10/61	0.02 - 2.4	12	N/A	5.6E+00 N	N/A	N/A	Yes	ASL
85-01-8	PHENANTHRENE	0.006	J	13	J	mg/kg	SB16-28-4-6	31/61	0.02 - 2.4	13	N/A	2.3E+02 N	N/A	N/A	No	BSL
129-00-0	PYRENE	0.013	J	6.4		mg/kg	SB16-21-0-2	36/61	0.02 - 2.4	6.4	N/A	2.3E+02 N	N/A	N/A	No	BSL

TABLE 2.3
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
DAVISVILLE - SITE 16 - TOTAL SOIL - RESIDENTIAL

Scenario Timeframe: Current/Future
Medium: Total Soil
Exposure Medium: Total Soil
Exposure Point: Davisville Site 16

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentraion	Detection Frequency	Range of Detection Limits	Concentration (2) Used for Screening	Background Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
							PCBs									
11096-82-5	AROCLOR-1260	0.014	U / _	0.014	U / _	mg/kg	SB16-21-0-2	1/24	0.018 - 0.04	0.014	N/A	2.2E-01 C	N/A	N/A	No	BSL
							Pesticides									
319-84-6	ALPHA-HCH	0.0024	J	0.0024	J	mg/kg	SB16-28-0-2	1/23	0.0018 - 0.0041	0.0024	N/A	9.0E-02 C	N/A	N/A	No	BSL
72-54-8	DDD	0.002925	UJ / J	0.002925	UJ / J	mg/kg	SB16-27-2-4	1/23	0.0034 - 0.008	0.002925	N/A	2.4E+00 C	N/A	N/A	No	BSL
50-29-3	DDT	0.0037		0.0037		mg/kg	SB16-26-0-2/SB16-26-2-4	2/23	0.0034 - 0.008	0.0037	N/A	1.7E+00 C	N/A	N/A	No	BSL
7421-93-4	ENDRIN ALDEHYDE	0.0047		0.0047		mg/kg	SB16-23-3-5	1/23	0.0034 - 0.008	0.0047	N/A	1.8E+00 N	N/A	N/A	No	BSL
5103-74-2	GAMMA-CHLORDANE	0.0029	J	0.0089	J	mg/kg	SB16-28-4-6	2/24	0.0018 - 0.0041	0.0089	N/A	1.6E+00 C	N/A	N/A	No	BSL
							Semivolatiles									
117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.036	J	0.37	U / _	mg/kg	MW16-07S-05-07	8/53	0.13 - 0.44	0.37	N/A	3.5E+01 C	N/A	N/A	No	BSL
86-74-8	CARBAZOLE	0.066	J	0.405	J	mg/kg	SB16-21-0-2	2/53	0.069 - 0.44	0.405	N/A	2.4E+01 C	N/A	N/A	No	BSL
132-64-9	DIBENZOFURAN	0.16	J	0.16	J	mg/kg	EBS-28-SB12-6-8	1/53	0.052 - 0.44	0.16	N/A	2.9E+01 N	N/A	N/A	No	BSL
84-74-2	DIBUTYLPHTHALATE	0.04	J	0.12	J	mg/kg	EBS-28-SB04-2-4	9/53	0.093 - 0.44	0.12	N/A	6.1E+02 N	N/A	N/A	No	BSL
84-66-2	DIETHYLPHTHALATE	0.061	J	0.061	J	mg/kg	EBS-28-SB12-6-8	1/53	0.069 - 0.44	0.061	N/A	4.9E+03 N	N/A	N/A	No	BSL
							Volatiles									
78-93-3	2-BUTANONE	0.007	J	0.012		mg/kg	SB16-27-0-2	6/54	0.004 - 0.665	0.012	N/A	7.3E+02 N	N/A	N/A	No	BSL
67-64-1	ACETONE	0.008		75	J	mg/kg	EBS-28-SB06-8-10	31/54	0.003 - 0.665	75	N/A	1.6E+02 N	N/A	N/A	No	BSL
71-43-2	BENZENE	0.008	J	0.034	J	mg/kg	SB16-28-4-6	2/54	0.001 - 0.335	0.034	N/A	6.5E-01 C	N/A	N/A	No	BSL
75-15-0	CARBON DISULFIDE	0.004		0.011		mg/kg	SB16-24-7-9	2/54	0.002 - 0.335	0.011	N/A	3.6E+01 N	N/A	N/A	No	BSL
100-41-4	ETHYLBENZENE	0.35		0.35		mg/kg	SB16-28-4-6	1/54	0.002 - 0.335	0.35	N/A	2.3E+02	N/A	N/A	No	BSL
MPXYLENES	M,P-XYLENES	0.62		0.62		mg/kg	SB16-28-4-6	1/20	0.005 - 0.335	0.62	N/A	2.1E+02	N/A	N/A	No	BSL
75-09-2	METHYLENE CHLORIDE	0.003	J	0.21	J	mg/kg	MW16-07S-05-07	7/54	0.001 - 0.335	0.21	N/A	8.9E+00 C	N/A	N/A	No	BSL
95-47-6	O-XYLENE	0.29		0.29		mg/kg	SB16-28-4-6	1/20	0.005 - 0.335	0.29	N/A	2.1E+02	N/A	N/A	No	BSL
108-88-3	TOLUENE	0.006	J	0.17		mg/kg	SB16-28-4-6	2/54	0.001 - 0.335	0.17	N/A	5.2E+02	N/A	N/A	No	BSL

(1) Minimum/maximum detected concentration.
(2) Maximum concentration used as screening value.
(3) Screening Toxicity Value - Taken from USEPA Region IX Preliminary Remediation Goals (PRGs) Table, USEPA, November 2000. For non-carcinogens, value shown is equal to 1/10 the Residential PRG. For carcinogens the value shown is equal to the Residential PRG.
(4) Rationale Codes

Selection Reason: Above Screening Toxicity and Background Levels (ASL)
Deletion Reason: Essential Nutrient (NUT)
Below Screening Toxicity Level (BSL)

Definitions:
N/A = Not Applicable
ND = No Data

COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
C = Carcinogenic
N = Non-Carcinogenic

TABLE 2.4
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
DAVISVILLE - SITE 16 - TOTAL SOIL - INDUSTRIAL

Scenario Timeframe: Current/Future
Medium: Total Soil
Exposure Medium: Total Soil
Exposure Point: Davisville Site 16

CAS Number	Chemical	Minimum [1] Concentration	Minimum Qualifier	Maximum [1] Concentration	Maximum Qualifier	Units	Location of Maximum Concentraion	Detection Frequency	Range of Detection Limits	Concentration [2] Used for Screening	Background Value	Screening [3] Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for [4] Contaminant Deletion or Selection
							Dioxin/Furans									
TEQ	**TCDD-TEQ**	**0.0000024**		**0.00049668**		**mg/kg**	**SB16-28-4-6**	**16/16**		**0.00049668**	**N/A**	**2.7E-05 C**	**N/A**	**N/A**	**Yes**	**ASL**
							Inorganics									
7429-90-5	ALUMINUM	2570		12400		mg/kg	SB16-22-6-8	24/24	1.28 - 16.38	12400	N/A	1.0E+05	N/A	N/A	No	BSL
7440-36-0	ANTIMONY	0.42	UJ / J	8.7		mg/kg	SB16-28-4-6	4/24	0.18 - 2.31	8.7	N/A	8.2E+01 N	N/A	N/A	No	BSL
7440-38-2	**ARSENIC**	**1.3**		**10.6**		**mg/kg**	**SB16-28-4-6**	**19/24**	**0.15 - 1.98**	**10.6**	**N/A**	**2.7E+00 C**	**N/A**	**N/A**	**Yes**	**ASL**
7440-39-3	BARIUM	10.8		1100		mg/kg	SB16-28-4-6	24/24	0.04 - 1.7	1100	N/A	1.0E+05	N/A	N/A	No	BSL
7440-41-7	BERYLLIUM	0.27	J	0.71		mg/kg	SB16-22-6-8	24/24	0.04 - 0.07	0.71	N/A	2.2E+03 C	N/A	N/A	No	BSL
7440-43-9	CADMIUM	0.33	J	2.4		mg/kg	SB16-28-4-6	5/24	0.04 - 0.86	2.4	N/A	8.1E+01 N	N/A	N/A	No	BSL
7440-70-2	CALCIUM METAL	282		23400		mg/kg	SB16-28-4-6	19/24	0.45 - 4.9	23400	N/A	N/A	N/A	N/A	No	NUT
7440-47-3	CHROMIUM	2.5		67.2		mg/kg	SB16-28-4-6	23/24	0.06 - 1.32	67.2	N/A	6.4E+01 C	N/A	N/A	No	BSL
7440-48-4	COBALT	2		10.2		mg/kg	MW16-03S-03-05	24/24	0.07 - 1.4	10.2	N/A	1.0E+05	N/A	N/A	No	BSL
7440-50-8	COPPER	6.7	J	452	J	mg/kg	SB16-28-4-6	24/24	0.15 - 0.93	452	N/A	7.6E+03 N	N/A	N/A	No	BSL
7439-89-6	IRON	5900		54300		mg/kg	SB16-28-4-6	24/24	0.27 - 6.72	54300	N/A	N/A	N/A	N/A	No	NUT
7439-92-1	**LEAD**	**3.95**		**2650**		**mg/kg**	**SB16-28-4-6**	**24/24**	**0.12 - 16.4**	**2650**	**N/A**	**7.5E+02**	**N/A**	**N/A**	**Yes**	**ASL**
7439-95-4	MAGNESIUM	525.2875	_ / U	4910		mg/kg	SB16-28-4-6	22/24	1.01 - 12.9	4910	N/A	N/A	N/A	N/A	No	NUT
7439-96-5	MANGANESE	70.25		587	J	mg/kg	SB16-28-4-6	24/24	0.02 - 0.44	587	N/A	3.2E+03 N	N/A	N/A	No	BSL
7439-97-6	MERCURY (INORGANIC)	0.04	J / U	0.96		mg/kg	SB16-28-4-6	5/24	0.01 - 0.055	0.96	N/A	6.1E+01 N	N/A	N/A	No	BSL
7440-02-0	NICKEL	2.1475	_ / U	34.5		mg/kg	SB16-28-4-6	19/24	0.11 - 3.94	34.5	N/A	4.1E+03 N	N/A	N/A	No	BSL
7440-09-7	POTASSIUM	381		1100		mg/kg	SB16-23-3-5	19/24	17.2 - 41.03	1100	N/A	N/A	N/A	N/A	No	NUT
7782-49-2	SELENIUM	0.33	J	0.92		mg/kg	MW16-03S-03-05	5/24	0.23 - 2.97	0.92	N/A	1.0E+03 N	N/A	N/A	No	BSL
7440-22-4	SILVER	0.225	J / U	6.8		mg/kg	SB16-28-4-6	7/24	0.1 - 0.9	6.8	N/A	1.0E+03 N	N/A	N/A	No	BSL
7440-23-5	SODIUM	17.36	U / _	596		mg/kg	SB16-28-4-6	16/24	1.12 - 6.4	596	N/A	N/A	N/A	N/A	No	NUT
7440-28-0	THALLIUM	0.65	J	0.65	J	mg/kg	SB16-27-0-2	1/24	0.25 - 3.27	0.65	N/A	1.3E+01 N	N/A	N/A	No	BSL
7440-62-2	VANADIUM	4.6		18.9		mg/kg	SB16-22-6-8	24/24	0.09 - 1.32	18.9	N/A	1.4E+03 N	N/A	N/A	No	BSL
7440-66-6	ZINC	17.3	J	1610	J	mg/kg	SB16-28-4-6	24/24	0.12 - 1.5	1610	N/A	1.0E+05	N/A	N/A	No	BSL
							PAHs									
91-57-6	**2-METHYLNAPHTHALENE**	**0.03**		**49**		**mg/kg**	**SB16-28-4-6**	**5/53**	**0.02 - 2.4**	**49**	**N/A**	**1.9E+01 N**	**N/A**	**N/A**	**Yes**	**ASL**
83-32-9	ACENAPHTHENE	0.014		15.1	J	mg/kg	28-SB-01D	13/61	0.02 - 2.4	15.1	N/A	3.8E+03 N	N/A	N/A	No	BSL
208-96-8	ACENAPHTHYLENE	0.012		0.77	U / _	mg/kg	SB16-21-0-2	7/61	0.02 - 2.4	0.77	N/A	3.8E+03 N	N/A	N/A	No	BSL
120-12-7	ANTHRACENE	0.005	J	4.6	J	mg/kg	28-SB-01B	21/61	0.02 - 2.4	4.6	N/A	1.0E+05	N/A	N/A	No	BSL
56-55-3	**BENZ[A]ANTHRACENE**	**0.004**		**4.45**		**mg/kg**	**SB16-21-0-2**	**32/61**	**0.02 - 2.4**	**4.45**	**N/A**	**2.9E+00 C**	**N/A**	**N/A**	**Yes**	**ASL**
50-32-8	**BENZO[A]PYRENE**	**0.004**	**J**	**2.45**	**J**	**mg/kg**	**28-SB-01D**	**34/61**	**0.02 - 2.4**	**2.45**	**N/A**	**2.9E-01 C**	**N/A**	**N/A**	**Yes**	**ASL**
205-99-2	**BENZO[B]FLUORANTHENE**	**0.008**		**7.4**		**mg/kg**	**SB16-21-0-2**	**36/61**	**0.02 - 2.4**	**7.4**	**N/A**	**2.9E+00 C**	**N/A**	**N/A**	**Yes**	**ASL**
191-24-2	BENZO[G,H,I]PERYLENE	0.003	J	2.05	J	mg/kg	28-SB-01D	30/61	0.02 - 2.4	2.05	N/A	5.4E+03 N	N/A	N/A	No	BSL
207-08-9	BENZO[K]FLUORANTHENE	0.003		1.8		mg/kg	SB16-21-0-2	26/61	0.02 - 2.4	1.8	N/A	2.9E+01 C	N/A	N/A	No	BSL
218-01-9	CHRYSENE	0.015	J	4.75		mg/kg	SB16-21-0-2	35/61	0.02 - 2.4	4.75	N/A	2.9E+02 C	N/A	N/A	No	BSL
53-70-3	**DIBENZ[A,H]ANTHRACENE**	**0.009**		**0.75**	**U / _**	**mg/kg**	**SB16-21-0-2**	**17/61**	**0.002 - 2.4**	**0.75**	**N/A**	**2.9E-01 C**	**N/A**	**N/A**	**Yes**	**ASL**
206-44-0	FLUORANTHENE	0.017	J	5.5		mg/kg	SB16-28-4-6	35/61	0.02 - 2.4	5.5	N/A	3.0E+03 N	N/A	N/A	No	BSL
86-73-7	FLUORENE	0.005	J	5.1	J	mg/kg	SB16-28-4-6	13/61	0.007 - 2.4	5.1	N/A	3.3E+03 N	N/A	N/A	No	BSL
193-39-5	INDENO[1,2,3-C,D]PYRENE	0.002	J	2.3		mg/kg	SB16-21-0-2	31/61	0.02 - 2.4	2.3	N/A	2.9E+00 C	N/A	N/A	No	BSL
91-20-3	NAPHTHALENE	0.006		12		mg/kg	SB16-28-4-6	10/61	0.02 - 2.4	12	N/A	1.9E+01 N	N/A	N/A	No	BSL
85-01-8	PHENANTHRENE	0.006	J	13	J	mg/kg	SB16-28-4-6	31/61	0.02 - 2.4	13	N/A	5.4E+03 N	N/A	N/A	No	BSL
129-00-0	PYRENE	0.013	J	6.4		mg/kg	SB16-21-0-2	36/61	0.02 - 2.4	6.4	N/A	5.4E+03 N	N/A	N/A	No	BSL

TABLE 2.4
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
DAVISVILLE - SITE 16 - TOTAL SOIL - INDUSTRIAL

Scenario Timeframe: Current/Future
Medium: Total Soil
Exposure Medium: Total Soil
Exposure Point: Davisville Site 16

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentraion	Detection Frequency	Range of Detection Limits	Concentration (2) Used for Screening	Background Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
							PCBs									
11096-82-5	AROCLOR-1260	0.014	U / _	0.014	U / _	mg/kg	SB16-21-0-2	1/24	0.018 - 0.04	0.014	N/A	1.0E+00 C	N/A	N/A	No	BSL
							Pesticides									
319-84-6	ALPHA-HCH	0.0024	J	0.0024	J	mg/kg	SB16-28-0-2	1/23	0.0018 - 0.0041	0.0024	N/A	5.9E-01 C	N/A	N/A	No	BSL
72-54-8	DDD	0.002925	UJ / J	0.002925	UJ / J	mg/kg	SB16-27-2-4	1/23	0.0034 - 0.008	0.002925	N/A	1.7E+01 C	N/A	N/A	No	BSL
50-29-3	DDT	0.0037		0.0037		mg/kg	SB16-26-0-2/SB16-26-2-4	2/23	0.0034 - 0.008	0.0037	N/A	1.2E+01 C	N/A	N/A	No	BSL
7421-93-4	ENDRIN ALDEHYDE	0.0047		0.0047		mg/kg	SB16-23-3-5	1/23	0.0034 - 0.008	0.0047	N/A	2.6E+01 N	N/A	N/A	No	BSL
5103-74-2	GAMMA-CHLORDANE	0.0029	J	0.0089	J	mg/kg	SB16-28-4-6	2/24	0.0018 - 0.0041	0.0089	N/A	1.1E+01 C	N/A	N/A	No	BSL
							Semivolatiles									
117-81-7	BIS(2-ETHYLHEXYL)PHTHALATE	0.036	J	0.37	U / _	mg/kg	MW16-07S-05-07	8/53	0.13 - 0.44	0.37	N/A	1.8E+02 C	N/A	N/A	No	BSL
86-74-8	CARBAZOLE	0.066	J	0.405	J	mg/kg	SB16-21-0-2	2/53	0.069 - 0.44	0.405	N/A	1.2E+02 C	N/A	N/A	No	BSL
132-64-9	DIBENZOFURAN	0.16	J	0.16	J	mg/kg	EBS-28-SB12-6-8	1/53	0.052 - 0.44	0.16	N/A	5.1E+02 N	N/A	N/A	No	BSL
84-74-2	DIBUTYLPHTHALATE	0.04	J	0.12	J	mg/kg	EBS-28-SB04-2-4	9/53	0.093 - 0.44	0.12	N/A	8.8E+03 N	N/A	N/A	No	BSL
84-66-2	DIETHYLPHTHALATE	0.061	J	0.061	J	mg/kg	EBS-28-SB12-6-8	1/53	0.069 - 0.44	0.061	N/A	1.0E+05	N/A	N/A	No	BSL
							Volatiles									
78-93-3	2-BUTANONE	0.007	J	0.012		mg/kg	SB16-27-0-2	6/54	0.004 - 0.665	0.012	N/A	2.8E+03 N	N/A	N/A	No	BSL
67-64-1	ACETONE	0.008		75	J	mg/kg	EBS-28-SB06-8-10	31/54	0.003 - 0.665	75	N/A	6.2E+02 N	N/A	N/A	No	BSL
71-43-2	BENZENE	0.008	J	0.034	J	mg/kg	SB16-28-4-6	2/54	0.001 - 0.335	0.034	N/A	1.5E+00 C	N/A	N/A	No	BSL
75-15-0	CARBON DISULFIDE	0.004		0.011		mg/kg	SB16-24-7-9	2/54	0.002 - 0.335	0.011	N/A	7.2E+02	N/A	N/A	No	BSL
100-41-4	ETHYLBENZENE	0.35		0.35		mg/kg	SB16-28-4-6	1/54	0.002 - 0.335	0.35	N/A	2.3E+02	N/A	N/A	No	BSL
MPXYLENES	M,P-XYLENES	0.62		0.62		mg/kg	SB16-28-4-6	1/20	0.005 - 0.335	0.62	N/A	2.1E+02	N/A	N/A	No	BSL
75-09-2	METHYLENE CHLORIDE	0.003	J	0.21	J	mg/kg	MW16-07S-05-07	7/54	0.001 - 0.335	0.21	N/A	2.1E+01 C	N/A	N/A	No	BSL
95-47-6	O-XYLENE	0.29		0.29		mg/kg	SB16-28-4-6	1/20	0.005 - 0.335	0.29	N/A	2.1E+02	N/A	N/A	No	BSL
108-88-3	TOLUENE	0.006	J	0.17		mg/kg	SB16-28-4-6	2/54	0.001 - 0.335	0.17	N/A	5.2E+02	N/A	N/A	No	BSL

(1) Minimum/maximum detected concentration.
(2) Maximum concentration used as screening value.
(3) Screening Toxicity Value - Taken from USEPA Region IX Preliminary Remediation Goals (PRGs) Table, USEPA, November 2000. For non-carcinogens, value shown is equal to 1/10 the Industrial PRG. For carcinogens the value shown is equal to the Industrial PRG.
(4) Rationale Codes

Selection Reason: Above Screening Toxicity and Background Levels (ASL)
Deletion Reason: Essential Nutrient (NUT)
Below Screening Toxicity Level (BSL)

Definitions:
N/A = Not Applicable
ND = No Data

COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
C = Carcinogenic
N = Non-Carcinogenic

TABLE 2.5
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
DAVISVILLE - SITE 16 - SEEP WATER - TAP WATER

Scenario Timeframe: Current/Future
Medium: Seep Water
Exposure Medium: Seep Water
Exposure Point: Davisville Site 16

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentraion	Detection Frequency	Range of Detection Limits	Concentration (2) Used for Screening	Background Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
							Inorganics									
7429-90-5	ALUMINUM	2630		2630		ug/l	28-SP-01	1/3	14 - 300	2630	N/A	3.6E+03 N	N/A	N/A	No	BSL
7440-38-2	ARSENIC	1.9	J	1.9	J	ug/l	28-SP-01	1/3	1.8 - 8	1.9	N/A	4.5E-02 C	N/A	N/A	Yes	ASL
7440-39-3	BARIUM	2.3		370		ug/l	28-SP-01	3/3	5 - 7.4	370	N/A	2.6E+02 N	N/A	N/A	Yes	ASL
7440-41-7	BERYLLIUM	0.21	J	0.21	J	ug/l	28-SP-01	1/3	0.2 - 5	0.21	N/A	7.3E+00 N	N/A	N/A	No	BSL
7440-70-2	CALCIUM METAL	26700		47250		ug/l	SEEP16-01	2/3	21.3 - 50	47250	N/A	N/A	N/A	N/A	No	NUT
7440-48-4	COBALT	10.2		10.2		ug/l	SEEP16-02	1/3	6.3 - 30	10.2	N/A	2.2E+02 N	N/A	N/A	No	BSL
7440-50-8	COPPER	10.7	_ / U	10.7	_ / U	ug/l	SEEP16-01	1/3	4.1 - 25	10.7	N/A	1.4E+02 N	N/A	N/A	No	BSL
7439-89-6	IRON	6480		29600		ug/l	SEEP16-01	2/3	100 - 130	29600	N/A	N/A	N/A	N/A	No	NUT
7439-92-1	LEAD	7.45	U / _	29.3		ug/l	28-SP-01	2/3	1 - 5	29.3	N/A	3.6E-02 N	N/A	N/A	Yes	ASL
7439-95-4	MAGNESIUM	4830		27000		ug/l	SEEP16-02	3/3	32.9 - 50	27000	N/A	N/A	N/A	N/A	No	NUT
7439-96-5	MANGANESE	445.5		2170		ug/l	SEEP16-02	2/3	1 - 5	2170	N/A	8.8E+01 N	N/A	N/A	Yes	ASL
7439-97-6	MERCURY (INORGANIC)	0.1		0.1		ug/l	SEEP16-02	1/3	0.1 - 0.2	0.1	N/A	1.1E+00 N	N/A	N/A	No	BSL
7440-02-0	NICKEL	1.1	J	1.1	J	ug/l	SEEP16-02	1/3	9.6 - 40	1.1	N/A	7.3E+01 N	N/A	N/A	No	BSL
7440-09-7	POTASSIUM	3600		10800		ug/l	SEEP16-02	2/3	84.2 - 1000	10800	N/A	N/A	N/A	N/A	No	NUT
7440-23-5	SODIUM	13450		182000		ug/l	SEEP16-02	3/3	28.1 - 1000	182000	N/A	N/A	N/A	N/A	No	NUT
7440-62-2	VANADIUM	7.85	_ / U	8.1	J	ug/l	28-SP-01	2/3	3.2 - 25	8.1	N/A	2.6E+01 N	N/A	N/A	No	BSL
7440-66-6	ZINC	18.05	_ / U	43.8		ug/l	SEEP16-02	2/3	6.8 - 25	43.8	N/A	1.1E+03 N	N/A	N/A	No	BSL
							PAHs									
91-57-6	2-METHYLNAPHTHALENE	0.95		0.95		ug/l	SEEP16-01	1/3	0.2 - 1	0.95	N/A	6.2E-01 N	N/A	N/A	Yes	ASL
83-32-9	ACENAPHTHENE	17		35.5		ug/l	SEEP16-01	2/3	0.2 - 1	35.5	N/A	3.7E+01 N	N/A	N/A	No	BSL
208-96-8	ACENAPHTHYLENE	0.2	_ / J	0.2	_ / J	ug/l	SEEP16-01	1/3	0.2 - 1	0.2	N/A	3.7E+01 N	N/A	N/A	No	BSL
120-12-7	ANTHRACENE	1.5	J	1.5	J	ug/l	SEEP16-01	1/3	0.2 - 1	1.5	N/A	1.8E+02 N	N/A	N/A	No	BSL
206-44-0	FLUORANTHENE	2.5	J	4		ug/l	28-SP-01	2/3	0.2 - 1	4	N/A	1.5E+02 N	N/A	N/A	No	BSL
86-73-7	FLUORENE	12		14.5		ug/l	SEEP16-01	2/3	0.2 - 1	14.5	N/A	2.4E+01 N	N/A	N/A	No	BSL
91-20-3	NAPHTHALENE	3		4	_ / J	ug/l	SEEP16-01	2/3	0.2 - 2	4	N/A	6.2E-01 N	N/A	N/A	Yes	ASL
85-01-8	PHENANTHRENE	7		7	_ / J / _	ug/l	28-SP-01/SEEP16-01	2/3	0.2 - 1	7	N/A	1.8E+01 N	N/A	N/A	No	BSL
129-00-0	PYRENE	1		2		ug/l	28-SP-01	2/3	0.2 - 1	2	N/A	1.8E+01 N	N/A	N/A	No	BSL
							Pesticides									
319-84-6	ALPHA-HCH	0.0325	_ / J	0.0325	_ / J	ug/l	SEEP16-01	1/3	0.001 - 0.05	0.0325	N/A	1.1E-02 C	N/A	N/A	Yes	ASL
60-57-1	DIELDRIN	0.02		0.02		ug/l	28-SP-01	1/3	0.003 - 0.1	0.02	N/A	4.2E-03 C	N/A	N/A	Yes	ASL
72-20-8	ENDRIN	0.0039	J	0.0039	J	ug/l	28-SP-01	1/3	0.003 - 0.1	0.0039	N/A	1.1E+00 N	N/A	N/A	No	BSL
1024-57-3	HEPTACHLOR EPOXIDE	0.02	J	0.02	J	ug/l	28-SP-01	1/3	0.001 - 0.05	0.02	N/A	7.4E-03 C	N/A	N/A	Yes	ASL
							Semivolatiles									
132-64-9	DIBENZOFURAN	8		11.5		ug/l	SEEP16-01	2/2	1 - 10	11.5	N/A	2.4E+00 N	N/A	N/A	Yes	ASL
							Volatiles									
156-59-2	CIS-1,2-DICHLOROETHENE	0.7		0.7		ug/l	SEEP16-02	1/2	2 - 2	0.7	N/A	6.1E+00 N	N/A	N/A	No	BSL
540-59-0	TOTAL 1,2-DICHLOROETHENE	0.7		0.7		ug/l	SEEP16-02	1/3	1 - 1	0.7	N/A	6.1E+00 N	N/A	N/A	No	BSL
79-01-6	TRICHLOROETHENE	0.7		0.7		ug/l	SEEP16-02	1/3	1 - 1	0.7	N/A	1.6E+00 C	N/A	N/A	No	BSL
75-01-4	VINYL CHLORIDE	0.45	/ J	0.45	/ J	ug/l	SEEP16-01	1/3	1 - 2	0.45	N/A	4.1E-02 C	N/A	N/A	Yes	ASL

(1) Minimum/maximum detected concentration.
(2) Maximum concentration used as screening value.
(3) Screening Toxicity Value - Taken from USEPA Region IX Preliminary Remediation Goals (PRGs) Table, USEPA, November 2000. For non-carcinogens, value shown is equal to 1/10 the Tap Water PRG. For carcinogens the value shown is equal to the Tap Water PRG.
(4) Rationale Codes
Selection Reason: Above Screening Toxicity and Background Levels (ASL)
Deletion Reason: Essential Nutrient (NUT)
Below Screening Toxicity Level (BSL)

Definitions:
N/A = Not Applicable
ND = No Data

COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
C = Carcinogenic
N = Non-Carcinogenic

TABLE 2.6
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
DAVISVILLE - SITE 16 - GROUNDWATER - TAP WATER

Scenario Timeframe: Current/Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Davisville Site 16

CAS Number	Chemical	Minimum [1] Concentration	Minimum Qualifier	Maximum [1] Concentration	Maximum Qualifier	Units	Location of Maximum Concentraion	Detection Frequency	Range of Detection Limits	Concentration [2] Used for Screening	Background Value	Screening [3] Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for [4] Contaminant Deletion or Selection
							Inorganics									
7429-90-5	ALUMINUM	413		3360	J	ug/l	MW16-23D	13/42	300 - 300	3,360	5,315	3.6E+03 N	N/A	N/A	No	BSL
7440-38-2	ARSENIC	2.8	J	2.8	J	ug/l	MW16-07D/MW16-04D	2/42	8 - 8	2.8	6.4	4.5E-02 C	N/A	N/A	No	BBL
7440-39-3	**BARIUM**	**3.2**		**923.5**		**ug/l**	**MW16-07S**	**42/42**	**5 - 5**	**923.5**	**80.5**	**2.6E+02 N**	**N/A**	**N/A**	**Yes**	**ASL**
7440-41-7	BERYLLIUM	1.34	U / J	1.34	U / J	ug/l	MW16-10D	1/42	5 - 5	1.34	1.3	7.3E+00 N	N/A	N/A	No	BSL
7440-43-9	CADMIUM	0.31		0.31		ug/l	MW16-19D	1/42	10 - 10	0.31	3	1.8E+00 N	N/A	N/A	No	BSL
7440-70-2	CALCIUM METAL	4910		534000		ug/l	MW16-28R	42/42	50 - 500	534,000	13,302	N/A	N/A	N/A	No	NUT
7440-47-3	CHROMIUM	0.63	J	7.5		ug/l	MW16-28R	23/42	15 - 15	7.5	214	1.1E+01 N	N/A	N/A	N/A	N/A
7440-48-4	COBALT	1.1	J	8.1	U / J	ug/l	MW16-05D	26/42	30 - 30	8.1	24.9	2.2E+02 N	N/A	N/A	No	BSL
7440-50-8	COPPER	1.6	J	6.685	J / U	ug/l	MW16-25R	4/42	25 - 25	7	25.8	1.4E+02 N	N/A	N/A	No	BSL
7439-89-6	IRON	69	J	27800	J	ug/l	MW16-05S	40/42	100 - 100	27,800	25,500	N/A	N/A	N/A	No	NUT
7439-92-1	**LEAD**	**1.6**	**J**	**51.25**	**J**	**ug/l**	**MW16-25R**	**17/42**	**5 - 5**	**51.25**	**4.8**	**3.6E-03**	**N/A**	**N/A**	**Yes**	**ASL**
7439-95-4	MAGNESIUM	1290		146000		ug/l	MW16-28R	42/42	50 - 50	146,000	5,126	N/A	N/A	N/A	No	NUT
7439-96-5	**MANGANESE**	**1.5**		**5270**		**ug/l**	**MW16-28R**	**42/42**	**5 - 5**	**5,270**	**3,292**	**8.8E+01 N**	**N/A**	**N/A**	**Yes**	**ASL**
7440-02-0	NICKEL	1.1	J	9.6		ug/l	MW16-15D	28/42	40 - 40	9.6	154	7.3E+01 N	N/A	N/A	No	BSL
7440-09-7	POTASSIUM	1350		33100		ug/l	MW16-28R	42/42	1000 - 10000	33,100	3,843	N/A	N/A	N/A	No	NUT
7782-49-2	SELENIUM	3.5	J	4.25	U / J	ug/l	MW16-10D	2/42	10 - 10	4.25	2.2	1.8E+01 N	N/A	N/A	No	BSL
7440-22-4	SILVER	1.2	J	1.2	J	ug/l	MW16-28R/MW16-17D	2/42	15 - 15	1.2	1	1.8E+01 N	N/A	N/A	No	BSL
7440-23-5	SODIUM	4840	J	1880000		ug/l	MW16-28R	42/42	1000 - 10000	1,880,000	12,346	N/A	N/A	N/A	No	NUT
7440-62-2	VANADIUM	0.53	J	6.54	U / J	ug/l	MW16-15D	17/42	25 - 25	6.54	24.4	2.6E+01 N	N/A	N/A	No	BSL
7440-66-6	ZINC	1.8		162		ug/l	MW16-28R	36/42	25 - 25	162	89.9	1.1E+03 N	N/A	N/A	No	BSL
							PAHs									
83-32-9	ACENAPHTHENE	0.1		0.1		ug/l	MW16-04S	1/42	0.2 - 1.1	0.1	N/A	3.7E+01 N	N/A	N/A	No	BSL
206-44-0	FLUORANTHENE	0.1		0.75	J	ug/l	MW16-07S	2/42	0.2 - 1.1	0.75	N/A	1.5E+02 N	N/A	N/A	No	BSL
86-73-7	FLUORENE	0.1		0.1		ug/l	MW16-04S	1/42	0.2 - 1.1	0.1	N/A	2.4E+01 N	N/A	N/A	No	BSL
91-20-3	NAPHTHALENE	0.1		0.2	J	ug/l	MW16-09D	3/42	0.2 - 1.1	0.2	N/A	6.2E-01 N	N/A	N/A	No	BSL
85-01-8	PHENANTHRENE	0.06		0.1		ug/l	MW16-04S	2/42	0.2 - 1.1	0.1	N/A	1.8E+01 N	N/A	N/A	No	BSL
							Pesticides									
319-85-7	BETA-HCH	0.011		0.011		ug/l	MW16-07D	1/42	0.05 - 0.053	0.011	N/A	3.7E-02 C	N/A	N/A	No	BSL
5103-74-2	GAMMA-CHLORDANE	0.004	J	0.004	J	ug/l	MW16-02R	1/42	0.05 - 0.053	0.004	N/A	1.9E-01 C	N/A	N/A	N/A	N/A
							Semivolatiles									
106-46-7	**1,4-DICHLOROBENZENE**	**2**	**J**	**2**	**J**	**ug/l**	**MW16-08D**	**1/41**	**10 - 11**	**2**	**N/A**	**5.0E-01 C**	**N/A**	**N/A**	**Yes**	**ASL**
117-81-7	**BIS(2-ETHYLHEXYL)PHTHALATE**	**5**		**11.5**	**UJ / J**	**ug/l**	**MW16-07S**	**5/41**	**10 - 11**	**11.5**	**N/A**	**4.8E+00 C**	**N/A**	**N/A**	**Yes**	**ASL**
84-74-2	DIBUTYLPHTHALATE	0.6		0.9		ug/l	MW16-19D	2/41	10 - 11	0.9	N/A	3.6E+02 N	N/A	N/A	No	BSL

TABLE 2.6
OCCURRENCE, DISTRIBUTION AND SELECTION OF CHEMICALS OF POTENTIAL CONCERN
DAVISVILLE - SITE 16 - GROUNDWATER - TAP WATER

Scenario Timeframe: Current/Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Davisville Site 16

CAS Number	Chemical	Minimum (1) Concentration	Minimum Qualifier	Maximum (1) Concentration	Maximum Qualifier	Units	Location of Maximum Concentraion	Detection Frequency	Range of Detection Limits	Concentration (2) Used for Screening	Background Value	Screening (3) Toxicity Value	Potential ARAR/TBC Value	Potential ARAR/TBC Source	COPC Flag	Rationale for (4) Contaminant Deletion or Selection
							Volatiles									
79-00-5	1,1,2-TRICHLOROETHANE	0.3		1		ug/l	MW16-21D	3/41	1 - 50	1	N/A	2.0E-01 C	N/A	N/A	Yes	ASL
75-34-3	1,1-DICHLOROETHANE	0.4	J	0.6		ug/l	MW16-16D	2/41	1 - 50	0.6	N/A	8.1E+01 N	N/A	N/A	No	BSL
75-35-4	1,1-DICHLOROETHENE	0.5		2		ug/l	MW16-02R	9/41	1 - 50	2	N/A	4.6E-02 C	N/A	N/A	Yes	ASL
107-06-2	1,2-DICHLOROETHANE	0.3		0.4	_ / U / _	ug/l	MW16-10D/MW16-25D	5/41	1 - 50	0.4	N/A	1.2E-01 C	N/A	N/A	Yes	ASL
78-93-3	2-BUTANONE	1		6		ug/l	MW16-07S	2/41	5 - 250	6	N/A	1.9E+02 N	N/A	N/A	No	BSL
108-10-1	4-METHYL-2-PENTANONE	1.75	U / _	1.75	U / _	ug/l	MW16-07S	1/41	3 - 150	1.75	N/A	1.6E+01 N	N/A	N/A	No	BSL
67-64-1	ACETONE	0.8	J	130	J	ug/l	MW16-15R	6/40	5 - 250	130	N/A	6.1E+01 N	N/A	N/A	Yes	ASL
71-43-2	BENZENE	0.5		1		ug/l	MW16-20D	3/41	1 - 50	1	N/A	3.5E-01 C	N/A	N/A	Yes	ASL
75-15-0	CARBON DISULFIDE	0.5	J	4		ug/l	MW16-10R	5/41	1 - 50	4	N/A	1.0E+02 N	N/A	N/A	No	BSL
75-00-3	CHLOROETHANE	0.6		0.6		ug/l	MW16-14D	1/41	2 - 100	0.6	N/A	4.6E+00 C	N/A	N/A	No	BSL
67-66-3	CHLOROFORM	0.45	U / _	0.5	J	ug/l	MW16-29D	2/41	1 - 50	0.5	N/A	1.6E-01 C	N/A	N/A	Yes	ASL
156-59-2	CIS-1,2-DICHLOROETHENE	0.8		110		ug/l	MW16-15R	16/41	2 - 100	110	N/A	6.1E+00 N	N/A	N/A	Yes	ASL
100-41-4	ETHYLBENZENE	0.5	U / _	0.65		ug/l	MW16-07S	2/41	1 - 50	0.65	N/A	1.3E+02 N	N/A	N/A	No	BSL
95-47-6	O-XYLENE	0.45		0.5		ug/l	MW16-15D	2/41	1 - 50	0.5	N/A	1.4E+02 N	N/A	N/A	No	BSL
127-18-4	TETRACHLOROETHENE	0.4		0.7		ug/l	MW16-15D	4/41	1 - 50	0.7	N/A	1.1E+00 C	N/A	N/A	No	BSL
108-88-3	TOLUENE	0.45		0.85		ug/l	MW16-25R	3/41	1 - 50	0.85	N/A	7.2E+01 N	N/A	N/A	No	BSL
540-59-0	TOTAL 1,2-DICHLOROETHENE	0.8		110		ug/l	MW16-15R	16/41	1 - 50	110	N/A	6.1E+00 N	N/A	N/A	Yes	ASL
156-60-5	TRANS-1,2-DICHLOROETHENE	0.6		3	J	ug/l	MW16-29D	7/41	2 - 100	3	N/A	1.2E+01 N	N/A	N/A	No	BSL
79-01-6	TRICHLOROETHENE	0.5		6300		ug/l	MW16-15D	29/41	1 - 50	6300	N/A	1.6E+00 C	N/A	N/A	Yes	ASL
75-01-4	VINYL CHLORIDE	0.9		2	J	ug/l	MW16-29D	2/41	2 - 100	2	N/A	4.1E-02 C	N/A	N/A	Yes	ASL

(1) Minimum/maximum detected concentration.
(2) Maximum concentration used as screening value.
(3) Screening Toxicity Value - Taken from USEPA Region IX Preliminary Remediation Goals (PRGs) Table, USEPA, November 2000. For non-carcinogens, value shown is equal to 1/10 the Tap Water PRG. For carcinogens the value shown is equal to the Tap Water PRG.
(4) Rationale Codes

Selection Reason: Above Screening Toxicity and Background Levels (ASL)
Deletion Reason: Essential Nutrient (NUT)
Below Screening Toxicity Level (BSL)
Below Background Level (BBL)

Definitions:
N/A = Not Applicable
ND = No Data

COPC = Chemical of Potential Concern
ARAR/TBC = Applicable or Relevant and Appropriate Requirement/To Be Considered
C = Carcinogenic
N = Non-Carcinogenic

TABLE 3.1
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
DAVISVILLE - SITE 16 - SURFACE SOIL - RESIDENTIAL

Scenario Timeframe: Current/Future
Medium: Surface Soil
Exposure Medium: Surface Soil
Exposure Point: Davisville Site 16

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
Dioxin/Furans												
TCDD-TEQ	mg/kg	1.13E-05	N/A	4.52E-05		mg/kg	3.79E-05	95% UCL-T	W - Test (1)	3.79E-05	95% UCL-T	Regional Guidance
Inorganics												
ALUMINUM	mg/kg	4.61E+03	N/A	8.59E+03		mg/kg	5.97E+03	95% UCL-T	W - Test (1)	5.97E+03	95% UCL-T	Regional Guidance
ARSENIC	mg/kg	1.87E+00	2.67E+00	4.00E+00		mg/kg	2.67E+00	95% UCL-N	W - Test (3)	2.67E+00	95% UCL-N	Regional Guidance
MANGANESE	mg/kg	1.45E+02	N/A	2.48E+02	J	mg/kg	1.85E+02	95% UCL-T	W - Test (1)	1.85E+02	95% UCL-T	Regional Guidance
THALLIUM	mg/kg	2.14E-01	N/A	6.50E-01	J	mg/kg	3.07E-01	95% UCL-T	W - Test (1)	3.07E-01	95% UCL-T	Regional Guidance
PAHs												
BENZ[A]ANTHRACENE	mg/kg	3.39E-01	N/A	4.45E+00		mg/kg	5.61E-01	95% UCL-T	W - Test (1)	5.61E-01	95% UCL-T	Regional Guidance
BENZO[A]PYRENE	mg/kg	2.59E-01	N/A	2.35E+00		mg/kg	4.68E-01	95% UCL-T	W - Test (1)	4.68E-01	95% UCL-T	Regional Guidance
BENZO[B]FLUORANTHENE	mg/kg	4.88E-01	N/A	7.40E+00		mg/kg	6.78E-01	95% UCL-T	W - Test (1)	6.78E-01	95% UCL-T	Regional Guidance
DIBENZ[A,H]ANTHRACENE	mg/kg	1.25E-01	N/A	7.50E-01	U / _	mg/kg	3.81E-01	95% UCL-T	W - Test (1)	3.81E-01	95% UCL-T	Regional Guidance
INDENO[1,2,3-C,D]PYRENE	mg/kg	2.20E-01	N/A	2.30E+00		mg/kg	4.17E-01	95% UCL-T	W - Test (1)	4.17E-01	4.17E-01	Regional Guidance

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

(1) Shapiro-Wilk W Test indicates data are log-normally distributed.
(2) 95% UCL exceeds maximum detected concentration. Therefore, maximum concentration used for EPC.
(3) Shapiro-Wilk W Test indicates data are normally distributed.

TABLE 3.2
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
DAVISVILLE - SITE 16 - SURFACE SOIL - INDUSTRIAL

Scenario Timeframe: Current/Future
Medium: Surface Soil
Exposure Medium: Surface Soil
Exposure Point: Davisville Site 16

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
Dioxin/Furans												
TCDD-TEQ	mg/kg	1.13E-05	N/A	4.52E-05		mg/kg	3.79E-05	95% UCL-T	W - Test (1)	3.79E-05	95% UCL-T	Regional Guidance
Inorganics												
ARSENIC	mg/kg	1.87E+00	2.67E+00	4.00E+00		mg/kg	2.67E+00	95% UCL-N	W - Test (3)	2.67E+00	95% UCL-N	Regional Guidance
PAHs												
BENZ[A]ANTHRACENE	mg/kg	3.39E-01	N/A	4.45E+00		mg/kg	5.61E-01	95% UCL-T	W - Test (1)	5.61E-01	95% UCL-T	Regional Guidance
BENZO[A]PYRENE	mg/kg	2.59E-01	N/A	2.35E+00		mg/kg	4.68E-01	95% UCL-T	W - Test (1)	4.68E-01	95% UCL-T	Regional Guidance
BENZO[B]FLUORANTHENE	mg/kg	4.88E-01	N/A	7.40E+00		mg/kg	6.78E-01	95% UCL-T	W - Test (1)	6.78E-01	95% UCL-T	Regional Guidance
DIBENZ[A,H]ANTHRACENE	mg/kg	1.25E-01	N/A	7.50E-01	U /	mg/kg	3.81E-01	95% UCL-T	W - Test (1)	3.81E-01	95% UCL-T	Regional Guidance

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

(1) Shapiro-Wilk W Test indicates data are log-normally distributed.
(2) 95% UCL exceeds maximum detected concentration. Therefore, maximum concentration used for EPC.
(3) Shapiro-Wilk W Test indicates data are normally distributed.

TABLE 3.3
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
DAVISVILLE - SITE 16 - TOTAL SOIL - RESIDENTIAL

Scenario Timeframe: Current/Future
Medium: Total Soil
Exposure Medium: Total Soil
Exposure Point: Davisville Site 16

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
Dioxin/Furans												
TCDD-TEQ	mg/kg	5.68E-05	N/A	4.97E-04		mg/kg	1.91E-04	95% UCL-T	W - Test (1)	1.91E-04	95% UCL-T	Regional Guidance
Inorganics												
ALUMINUM	mg/kg	5.57E+03	N/A	1.24E+04		mg/kg	6.61E+03	95% UCL-T	W - Test (1)	6.61E+03	95% UCL-T	Regional Guidance
ANTIMONY	mg/kg	9.61E-01	N/A	8.70E+00		mg/kg	1.32E+00	95% UCL-T	W - Test (1)	1.32E+00	95% UCL-T	Regional Guidance
ARSENIC	mg/kg	2.45E+00	N/A	1.06E+01		mg/kg	8.64E+00	95% UCL-T	W - Test (1)	8.64E+00	95% UCL-T	Regional Guidance
BARIUM	mg/kg	7.81E+01	N/A	1.10E+03		mg/kg	8.50E+01	95% UCL-T	W - Test (1)	8.50E+01	95% UCL-T	Regional Guidance
COPPER	mg/kg	4.28E+01	N/A	4.52E+02	J	mg/kg	5.56E+01	95% UCL-T	W - Test (1)	5.56E+01	95% UCL-T	Regional Guidance
LEAD	mg/kg	1.60E+02	N/A	2.65E+03		mg/kg	2.60E+02	95% UCL-T	W - Test (1)	2.60E+02	95% UCL-T	Regional Guidance
MANGANESE	mg/kg	1.66E+02	N/A	5.87E+02	J	mg/kg	2.00E+02	95% UCL-T	W - Test (1)	2.00E+02	95% UCL-T	Regional Guidance
THALLIUM	mg/kg	2.76E-01	N/A	6.50E-01	J	mg/kg	3.36E-01	95% UCL-T	W - Test (1)	3.36E-01	95% UCL-T	Regional Guidance
PAHs												
2-METHYLNAPHTHALENE	mg/kg	1.19E+00	N/A	4.90E+01		mg/kg	7.70E-01	95% UCL-T	Y - Test (4)	7.70E-01	95% UCL-T	Regional Guidance
BENZ[A]ANTHRACENE	mg/kg	3.19E-01	N/A	4.45E+00		mg/kg	4.27E-01	95% UCL-T	Y - Test (4)	4.27E-01	95% UCL-T	Regional Guidance
BENZO[A]PYRENE	mg/kg	2.89E-01	N/A	2.45E+00	J	mg/kg	4.18E-01	95% UCL-T	Y - Test (4)	4.18E-01	95% UCL-T	Regional Guidance
BENZO[B]FLUORANTHENE	mg/kg	4.27E-01	N/A	7.40E+00		mg/kg	5.17E-01	95% UCL-T	Y - Test (4)	5.17E-01	95% UCL-T	Regional Guidance
DIBENZ[A,H]ANTHRACENE	mg/kg	1.51E-01	N/A	7.50E-01	U / _	mg/kg	3.11E-01	95% UCL-T	Y - Test (4)	3.11E-01	95% UCL-T	Regional Guidance
INDENO[1,2,3-C,D]PYRENE	mg/kg	2.31E-01	N/A	2.30E+00		mg/kg	3.51E-01	95% UCL-T	Y - Test (4)	3.51E-01	95% UCL-T	Regional Guidance
NAPHTHALENE	mg/kg	3.93E-01	N/A	1.20E+01		mg/kg	4.91E-01	95% UCL-T	Y - Test (4)	4.91E-01	95% UCL-T	Regional Guidance

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

(1) Shapiro-Wilk W Test indicates data are log-normally distributed.
(2) 95% UCL exceeds maximum detected concentration. Therefore, maximum concentration used for EPC.
(3) Shapiro-Wilk W Test indicates data are normally distributed.
(4) D'Agostino Y Test indicates data are log-normally distributed.

TABLE 3.4
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
DAVISVILLE - SITE 16 - TOTAL SOIL - INDUSTRIAL

Scenario Timeframe: Current/Future
Medium: Total Soil
Exposure Medium: Total Soil
Exposure Point: Davisville Site 16

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
Dioxin/Furans												
TCDD-TEQ	mg/kg	5.68E-05	N/A	4.97E-04		mg/kg	1.91E-04	95% UCL-T	W - Test (1)	1.91E-04	95% UCL-T	Regional Guidance
Inorganics												
ARSENIC	mg/kg	2.45E+00	N/A	1.06E+01		mg/kg	8.64E+00	95% UCL-T	W - Test (1)	8.64E+00	95% UCL-T	Regional Guidance
LEAD	mg/kg	1.60E+02	N/A	2.65E+03		mg/kg	2.60E+02	95% UCL-T	W - Test (1)	2.60E+02	95% UCL-T	Regional Guidance
PAHs												
2-METHYLNAPHTHALENE	mg/kg	1.19E+00	N/A	4.90E+01		mg/kg	7.70E-01	95% UCL-T	Y - Test (4)	7.70E-01	95% UCL-T	Regional Guidance
BENZ[A]ANTHRACENE	mg/kg	3.19E-01	N/A	4.45E+00		mg/kg	4.27E-01	95% UCL-T	Y - Test (4)	4.27E-01	95% UCL-T	Regional Guidance
BENZO[A]PYRENE	mg/kg	2.89E-01	N/A	2.45E+00	J	mg/kg	4.18E-01	95% UCL-T	Y - Test (4)	4.18E-01	95% UCL-T	Regional Guidance
BENZO[B]FLUORANTHENE	mg/kg	4.27E-01	N/A	7.40E+00		mg/kg	5.17E-01	95% UCL-T	Y - Test (4)	5.17E-01	95% UCL-T	Regional Guidance
DIBENZ[A,H]ANTHRACENE	mg/kg	1.51E-01	N/A	7.50E-01	U /	mg/kg	3.11E-01	95% UCL-T	Y - Test (4)	3.11E-01	95% UCL-T	Regional Guidance

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

(1) Shapiro-Wilk W Test indicates data are log-normally distributed.
(2) 95% UCL exceeds maximum detected concentration. Therefore, maximum concentration used for EPC.
(3) Shapiro-Wilk W Test indicates data are normally distributed.
(4) D'Agostino Y Test indicates data are log-normally distributed.

TABLE 3.5
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
DAVISVILLE - SITE 16 - SEEP WATER - TAP WATER

Scenario Timeframe: Current/Future
Medium: Seep Water
Exposure Medium: Seep Water
Exposure Point: Davisville Site 16

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
Inorganics												
ARSENIC	ug/l	3.30E+00	N/A	1.90E+00	J	ug/l	1.90E+00	Max	W - Test (1)	1.90E+00	Max	Regional Guidance
BARIUM	ug/l	2.23E+02	5.50E+02	3.70E+02		ug/l	3.70E+02	Max	W - Test (2)	3.70E+02	Max	Regional Guidance
LEAD	ug/l	1.31E+01	N/A	2.93E+01		ug/l	1.75E+01	95% UCL-T	W - Test (1)	1.75E+01	95% UCL-T	Regional Guidance
MANGANESE	ug/l	8.72E+02	2.80E+03	2.17E+03		ug/l	2.17E+03	Max	W - Test (2)	2.17E+03	Max	Regional Guidance
PAHs												
2-METHYLNAPHTHALENE	ug/l	5.17E-01	1.23E+00	9.50E-01		ug/l	9.50E-01	Max	W - Test (2)	9.50E-01	Max	Regional Guidance
NAPHTHALENE	ug/l	2.37E+00	5.78E+00	4.00E+00	/ J	ug/l	4.00E+00	Max	W - Test (2)	4.00E+00	Max	Regional Guidance
Pesticides												
ALPHA-HCH	ug/l	1.93E-02	4.75E-02	3.25E-02	_ / J	ug/l	3.25E-02	Max	W - Test (2)	3.25E-02	Max	Regional Guidance
DIELDRIN	ug/l	4.00E-02	N/A	2.00E-02		ug/l	2.00E-02	Max	W - Test (1)	2.00E-02	Max	Regional Guidance
HEPTACHLOR EPOXIDE	ug/l	2.33E-02	N/A	2.00E-02	J	ug/l	2.00E-02	Max	W - Test (2)	2.00E-02	Max	Regional Guidance
Semivolatiles												
DIBENZOFURAN	ug/l	9.75E+00	N/A	1.15E+01		ug/l	1.15E+01	Max	Insignificant Population	1.15E+01	Max	Regional Guidance
Volatiles												
VINYL CHLORIDE	ug/l	6.50E-01	N/A	4.50E-01	/ J	ug/l	4.50E-01	Max	W - Test (1)	4.50E-01	Max	Regional Guidance

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

(1) Shapiro-Wilk W Test indicates data are log-normally distributed.
(2) 95% UCL exceeds maximum detected concentration. Therefore, maximum concentration used for EPC.
(3) Shapiro-Wilk W Test indicates data are normally distributed.

TABLE 3.6
MEDIUM-SPECIFIC EXPOSURE POINT CONCENTRATION SUMMARY
DAVISVILLE - SITE 16 - GROUNDWATER - TAP WATER

Scenario Timeframe: Current/Future
Medium: Groundwater
Exposure Medium: Groundwater
Exposure Point: Davisville Site 16

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
Inorganics												
BARIUM	ug/l	4.36E+01	N/A	9.24E+02		ug/l	3.78E+01	95% UCL-T	W - Test (1)	3.78E+01	95% UCL-T	Regional Guidance
LEAD	ug/l	3.63E+00	N/A	5.13E+01	J	ug/l	3.44E+00	95% UCL-T	W - Test (1)	3.44E+00	95% UCL-T	Regional Guidance
MANGANESE	ug/l	5.89E+02	N/A	5.27E+03		ug/l	1.17E+03	95% UCL-T	W - Test (1)	1.17E+03	95% UCL-T	Regional Guidance
Semivolatiles												
1,4-DICHLOROBENZENE	ug/l	4.94E+00	N/A	2.00E+00	J	ug/l	2.00E+00	Max	W - Test (1)	2.00E+00	Max	Regional Guidance
BIS(2-ETHYLHEXYL)PHTHALATE	ug/l	5.34E+00	N/A	1.15E+01	UJ / J	ug/l	5.58E+00	95% UCL-T	W - Test (1)	5.58E+00	95% UCL-T	Regional Guidance
Volatiles												
1,1,2-TRICHLOROETHANE	ug/l	1.10E+00	N/A	1.00E+00		ug/l	8.14E-01	95% UCL-T	W - Test (1)	8.14E-01	95% UCL-T	Regional Guidance
1,1-DICHLOROETHENE	ug/l	1.19E+00	N/A	2.00E+00		ug/l	9.53E-01	95% UCL-T	W - Test (1)	9.53E-01	95% UCL-T	Regional Guidance
1,2-DICHLOROETHANE	ug/l	1.08E+00	N/A	4.00E-01	_ / U / _	ug/l	4.00E-01	Max	W - Test (1)	4.00E-01	Max	Regional Guidance
ACETONE	ug/l	9.87E+00	N/A	1.30E+02	J	ug/l	8.09E+00	95% UCL-T	W - Test (1)	8.09E+00	95% UCL-T	Regional Guidance
BENZENE	ug/l	1.11E+00	N/A	1.00E+00		ug/l	8.22E-01	95% UCL-T	W - Test (1)	8.22E-01	95% UCL-T	Regional Guidance
CHLOROFORM	ug/l	1.10E+00	N/A	5.00E-01	J	ug/l	5.00E-01	Max	W - Test (1)	5.00E-01	Max	Regional Guidance
CIS-1,2-DICHLOROETHENE	ug/l	6.08E+00	N/A	1.10E+02		ug/l	5.59E+00	95% UCL-T	W - Test (1)	5.59E+00	95% UCL-T	Regional Guidance
TOTAL 1,2-DICHLOROETHENE	ug/l	6.04E+00	N/A	1.10E+02		ug/l	6.87E+00	95% UCL-T	W - Test (1)	6.87E+00	95% UCL-T	Regional Guidance
TRICHLOROETHENE	ug/l	8.91E+02	N/A	6.30E+03		ug/l	6.30E+03	Max	W - Test (2)	6.30E+03	Max	Regional Guidance
VINYL CHLORIDE	ug/l	2.22E+00	N/A	2.00E+00	J	ug/l	1.64E+00	95% UCL-T	W - Test (1)	1.64E+00	95% UCL-T	Regional Guidance

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

(1) Shapiro-Wilk W Test indicates data are log-normally distributed.
(2) 95% UCL exceeds maximum detected concentration. Therefore, maximum concentration used for EPC.

Table 4.1
VALUES USED FOR RESIDENT ADULT DAILY TOTAL SOIL INTAKE EQUATIONS
DAVISVILLE SITE 16

Scenario Timeframe: Future
Medium: Total Soil
Exposure Medium: Total Soil, Air
Exposure Point: Davisville Site 16
Receptor Population: Resident
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CTE Value	CTE Rationale/Reference
Ingestion	CR	Ingestion Rate	mg/day	100	U.S. EPA 1991	50	U.S. EPA 1991
	EF	Exposure Frequency	day/yr	150	U.S. EPA 1991	150	U.S. EPA 1991
	ED-NC	Exposure Duration	yr	30	U.S. EPA 1991	9	U.S. EPA 1991
	ED-C	Exposure Duration	yr	24	U.S. EPA 1991	9	U.S. EPA 1991
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	8,760	U.S. EPA 1991	3,285	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	kg/mg	1.00E-06	U.S. EPA 1989	1.00E-06	U.S. EPA 1989
Dermal	SA	Surface Area for Contact	cm2/event	8,000	U.S. EPA 1997a	6,200	U.S. EPA 1997a
	AF	Adherence Factor	mg/cm2	0.07	U.S. EPA 2000	0.01	U.S. EPA 2000
	EF	Exposure Frequency	event/yr	150	U.S. EPA 1991	150	BPJ
	ED-NC	Exposure Duration	yr	30	U.S. EPA 1991	9	U.S. EPA 1991
	ED-C	Exposure Duration	yr	24	U.S. EPA 1991	9	U.S. EPA 1991
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	8,760	U.S. EPA 1991	3,285	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	kg/mg	1.00E-06	U.S. EPA 1989	1.00E-06	U.S. EPA 1989
Inhalation	IR	Inhalation Rate	m3/hr	6.25E-01	U.S. EPA 1991	5.50E-01	U.S. EPA 1991
	ET	Exposure Time	hr/day	24	BPJ	10	BPJ
	EF	Exposure Frequency	day/yr	150	U.S. EPA 1991	150	U.S. EPA 1991
	ED-NC	Exposure Duration	yr	30	U.S. EPA 1991	9	U.S. EPA 1991
	ED-C	Exposure Duration	yr	24	U.S. EPA 1991	9	U.S. EPA 1991
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	8,760	U.S. EPA 1991	2,555	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a

Note : BPJ = Best Professional Judgement

Table 4.2
VALUES USED FOR RESIDENT CHILD DAILY TOTAL SOIL INTAKE EQUATIONS
DAVISVILLE SITE 16

Scenario Timeframe: Future
Medium: Total Soil
Exposure Medium: Total Soil, Air
Exposure Point: Davisville Site 16
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CTE Value	CTE Rationale/Reference
Ingestion	CR	Ingestion Rate	mg/day	200	U.S. EPA 1991	100	U.S. EPA 1991
	EF	Exposure Frequency	day/yr	150	U.S. EPA 1991	150	U.S. EPA 1991
	ED	Exposure Duration	yr	6	U.S. EPA 1991	2	U.S. EPA 1991
	BW	Body Weight	kg	15	U.S. EPA 1991	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	2,190	U.S. EPA 1991	730	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	kg/mg	1.00E-06	U.S. EPA 1989	1.00E-06	U.S. EPA 1989
Dermal	SA	Surface Area for Contact	cm2/event	3,600	U.S. EPA 2000	2,900	U.S. EPA 2000
	AF	Adherence Factor	mg/cm2	0.2	U.S. EPA 2000	0.06	U.S. EPA 2000
	EF	Exposure Frequency	event/yr	150	U.S. EPA 1991	150	U.S. EPA 1991
	ED	Exposure Duration	yr	6	U.S. EPA 1991	2	U.S. EPA 1991
	BW	Body Weight	kg	15	U.S. EPA 1991	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	2,190	U.S. EPA 1991	730	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	kg/mg	1.00E-06	U.S. EPA 1989	1.00E-06	U.S. EPA 1989
Inhalation	IR	Inhalation Rate	m3/hr	4.17E-01	U.S. EPA 1991	4.17E-01	U.S. EPA 1991
	ET	Exposure Time	hr/day	24	BPJ	10	BPJ
	EF	Exposure Frequency	day/yr	350	U.S. EPA 1991	150	U.S. EPA 1991
	ED	Exposure Duration	yr	6	U.S. EPA 1991	2	U.S. EPA 1991
	BW	Body Weight	kg	15	U.S. EPA 1991	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	2,190	U.S. EPA 1991	730	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a

Note : BPJ = Best Professional Judgement

Table 4.3
VALUES USED FOR ADULT RECREATIONAL USERS DAILY SURFACE SOIL INTAKE EQUATIONS
DAVISVILLE SITE 16

Scenario Timeframe: Current/Future
Medium: Surface Soil
Exposure Medium: Surface Soil, Air
Exposure Point: Davisville Site 16
Receptor Population: Recreational Users
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CTE Value	CTE Rationale/Reference
Ingestion	CR	Ingestion Rate	mg/day	100	U.S. EPA 1991	50	U.S. EPA 1991
	EF	Exposure Frequency	day/yr	96	BPJ	48	BPJ
	ED	Exposure Duration	yr	24	U.S. EPA 1991	9	U.S. EPA 1991
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	8,760	U.S. EPA 1991	3,285	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	kg/mg	1.00E-06	U.S. EPA 1989	1.00E-06	U.S. EPA 1989
Dermal	SA	Surface Area for Contact	cm2/event	8,000	U.S. EPA 1997a	6,200	U.S. EPA 1997a
	AF	Adherence Factor	mg/cm2	0.32	U.S. EPA 1998b	0.16	U.S. EPA 1998b
	EF	Exposure Frequency	event/yr	96	BPJ	48	BPJ
	ED	Exposure Duration	yr	24	U.S. EPA 1991	1	U.S. EPA 1991
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	8,760	U.S. EPA 1991	3,285	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	kg/mg	1.00E-06	U.S. EPA 1989	1.00E-06	U.S. EPA 1989
Inhalation	IR	Inhalation Rate	m3/hr	8.33E-01	U.S. EPA 1991	8.33E-01	U.S. EPA 1991
	ET	Exposure Time	hr/day	8	BPJ	4	BPJ
	EF	Exposure Frequency	day/yr	96	BPJ	48	BPJ
	ED	Exposure Duration	yr	24	BPJ	1	BPJ
	BW	Body Weight	kg	70	U.S. EPA 1989	70	U.S. EPA 1989
	AT-NC	Averaging time - Noncancer	days	3,650	U.S. EPA 1989	3,650	U.S. EPA 1989
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989	25,550	U.S. EPA 1989

Note : BPJ = Best Professional Judgement

Table 4.4
VALUES USED FOR CHILD RECREATIONAL USERS DAILY SURFACE SOIL INTAKE EQUATIONS
DAVISVILLE SITE 16

Scenario Timeframe: Current/Future
Medium: Surface Soil
Exposure Medium: Surface Soil, Air
Exposure Point: Davisville Site 16
Receptor Population: Recreational Users
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CTE Value	CTE Rationale/Reference
Ingestion	CR	Ingestion Rate	mg/day	200	U.S. EPA 1991	100	U.S. EPA 1991
	EF	Exposure Frequency	day/yr	96	BPJ	48	BPJ
	ED	Exposure Duration	yr	6	U.S. EPA 1991	2	U.S. EPA 1991
	BW	Body Weight	kg	15	U.S. EPA 1991	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	2,190	U.S. EPA 1991	730	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	kg/mg	1.00E-06		1.00E-06	
Dermal	SA	Surface Area for Contact	cm2/event	3,600	U.S. EPA 1997a	2,900	U.S. EPA 1997a
	AF	Adherence Factor	mg/cm2	0.32	U.S. EPA 1998b	0.16	U.S. EPA 1998b
	EF	Exposure Frequency	event/yr	96	BPJ	48	BPJ
	ED	Exposure Duration	yr	6	U.S. EPA 1991	2	U.S. EPA 1991
	BW	Body Weight	kg	15	U.S. EPA 1991	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	2,190	U.S. EPA 1991	730	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	kg/mg	1.00E-06		1.00E-06	
Inhalation	IR	Inhalation Rate	m3/hr	8.33E-01	U.S. EPA 1991	8.33E-01	U.S. EPA 1991
	ET	Exposure Time	hr/day	10	BPJ	4	BPJ
	EF	Exposure Frequency	day/yr	96	BPJ	48	BPJ
	ED	Exposure Duration	yr	6	U.S. EPA 1991	2	U.S. EPA 1991
	BW	Body Weight	kg	15	U.S. EPA 1991	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	2,190	U.S. EPA 1989a	730	U.S. EPA 1989a
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a

Note : BPJ = Best Professional Judgement

Table 4.5
VALUES USED FOR CONSTRUCTION WORKER DAILY TOTAL SOIL INTAKE EQUATIONS
DAVISVILLE SITE 16

Scenario Timeframe: Future
Medium: Total Soil
Exposure Medium: Total Soil, Air
Exposure Point: Davisville Site 16
Receptor Population: Construction Worker
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CTE Value	CTE Rationale/Reference
Ingestion	CR	Ingestion Rate	mg/day	480	U.S. EPA 1991	100	U.S. EPA 1991
	EF	Exposure Frequency	day/yr	160	U.S. EPA 1990	80	U.S. EPA 1990
	ED	Exposure Duration	yr	1	BPJ	1	BPJ
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	365	BPJ	365	BPJ
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	kg/mg	1.00E-06		1.00E-06	
Dermal	SA	Surface Area for Contact	cm2/event	3,800	U.S. EPA 1997a	3,140	U.S. EPA 1997a
	AF	Adherence Factor	mg/cm2	0.3	U.S. EPA 2000	0.1	U.S. EPA 2000
	EF	Exposure Frequency	event/yr	160	U.S. EPA 1990	80	U.S. EPA 1990
	ED	Exposure Duration	yr	1	BPJ	1	BPJ
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	365	BPJ	365	BPJ
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	kg/mg	1.00E-06		1.00E-06	
Inhalation	IR	Inhalation Rate	m3/hr	2.50	U.S. EPA 1997a	1.50	U.S. EPA 1997a
	ET	Exposure Time	hr/day	8	U.S. EPA 1991	8	U.S. EPA 1991
	EF	Exposure Frequency	day/yr	160	U.S. EPA 1990	80	U.S. EPA 1990
	ED	Exposure Duration	yr	1	BPJ	1	BPJ
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	365	BPJ	365	BPJ
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a

Note : BPJ = Best Professional Judgement

Table 4.6
VALUES USED FOR COMMERCIAL WORKER DAILY SURFACE SOIL INTAKE EQUATIONS
DAVISVILLE SITE 16

Scenario Timeframe: Future
Medium: Surface Soil
Exposure Medium: Surface Soil, Air
Exposure Point: Davisville Site 16
Receptor Population: Commercial Worker
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CTE Value	CTE Rationale/Reference
Ingestion	CR	Ingestion Rate	mg/day	50	U.S. EPA 1991	50	U.S. EPA 1991
	EF	Exposure Frequency	day/yr	250	U.S. EPA 1991	150	U.S. EPA 1991
	ED	Exposure Duration	yr	25	U.S. EPA 1991	12	U.S. EPA 1991
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	9,125	U.S. EPA 1991	4,380	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	kg/mg	1.00E-06		1.00E-06	
Dermal	SA	Surface Area for Contact	cm2/event	3,800	U.S. EPA 1989a	3,100	U.S. EPA 1989a
	AF	Adherence Factor	mg/cm2	0.2	U.S. EPA 2000	0.02	U.S. EPA 2000
	EF	Exposure Frequency	event/yr	250	U.S. EPA 1997	150	U.S. EPA 1997
	ED	Exposure Duration	yr	25	U.S. EPA 1997	12	U.S. EPA 1997
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	9,125	U.S. EPA 1991	4,380	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	kg/mg	1.00E-06		1.00E-06	
Inhalation	IR	Inhalation Rate	m3/hr	8.33E-01	U.S. EPA 1991	8.33E-01	U.S. EPA 1991
	ET	Exposure Time	hr/day	8	BPJ	4	BPJ
	EF	Exposure Frequency	day/yr	250	U.S. EPA 1991	150	BPJ
	ED	Exposure Duration	yr	25	U.S. EPA 1991	12	U.S. EPA 1991
	BW	Body Weight	kg	70	U.S. EPA 1989	70	U.S. EPA 1989
	AT-NC	Averaging time - Noncancer	days	9,125	U.S. EPA 1989	2,920	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a

Note : BPJ = Best Professional Judgement

Table 4.7
VALUES USED FOR RESIDENT ADULT DAILY TOTAL GROUNDWATER INTAKE EQUATIONS
DAVISVILLE SITE 16

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Tap Water
Exposure Point: Davisville Site 16
Receptor Population: Resident
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CTE Value	CTE Rationale/Reference
Ingestion	CR	Ingestion Rate	L/day	2.3	U.S. EPA 1991	1.4	U.S. EPA 1991
	EF	Exposure Frequency	day/yr	350	U.S. EPA 1991	250	U.S. EPA 1991
	ED-NC	Exposure Duration	yr	30	U.S. EPA 1997	9	U.S. EPA 1997
	ED-C	Exposure Duration	yr	24	U.S. EPA 1997	9	U.S. EPA 1997
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	8,760	U.S. EPA 1991	3,285	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
Dermal	SA	Surface Area for Contact	cm2	22,000	U.S. EPA 1997a	18,200	U.S. EPA 1997a
	PC	Permeability Coefficient	cm/hr	chemical-specific		chemical-specific	
	ET	Event Time	hr/day	0.58	U.S. EPA 1992	0.17	U.S. EPA 1992
	EF	Exposure Frequency	day/yr	350	U.S. EPA 1991	250	U.S. EPA 1991
	ED-NC	Exposure Duration	yr	30	U.S. EPA 1997	9	U.S. EPA 1997
	ED-C	Exposure Duration	yr	24	U.S. EPA 1997	9	U.S. EPA 1997
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	8,760	U.S. EPA 1991	3,285	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	L/cm3	1.00E-03		1.0E-03	
Inhalation	IR	Inhalation Rate	m^3/day	15	U.S. EPA 1997	13.2	U.S. EPA 1997
	EF	Exposure Frequency	day/yr	350	U.S. EPA 1991	175	U.S. EPA 1991
	ED	Exposure Duration	min/event	35	U.S. EPA 1997	10	U.S. EPA 1997
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	8,760	U.S. EPA 1991	3,285	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a

Note : BPJ = Best Professional Judgement

Table 4.8
VALUES USED FOR RESIDENT CHILD DAILY TOTAL GROUNDWATER INTAKE EQUATIONS
DAVISVILLE SITE 16

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Tap Water
Exposure Point: Davisville Site 16
Receptor Population: Resident
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CTE Value	CTE Rationale/Reference
Ingestion	CR	Ingestion Rate	L/day	1.5	U.S. EPA 1997a	0.74	U.S. EPA 1997a
	EF	Exposure Frequency	day/yr	350	U.S. EPA 1991	250	U.S. EPA 1991
	ED	Exposure Duration	yr	6	U.S. EPA 1997	2	U.S. EPA 1997
	BW	Body Weight	kg	15	U.S. EPA 1991	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	2,190	U.S. EPA 1991	730	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
Dermal	SA	Surface Area for Contact	cm2	10,500	U.S. EPA 1997a	9,000	U.S. EPA 1997a
	PC	Permeability Coefficient	cm/hr	chemical-specific		chemical-specific	
	ET	Event Time	hr/day	1	U.S. EPA 1992	0.33	U.S. EPA 1992
	EF	Exposure Frequency	day/yr	350	U.S. EPA 1991	250	U.S. EPA 1991
	ED	Exposure Duration	yr	6	U.S. EPA 1997	2	U.S. EPA 1997
	BW	Body Weight	kg	15	U.S. EPA 1991	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	2,190	U.S. EPA 1991	730	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	L/cm3	0.001		0.001	

Note : BPJ = Best Professional Judgement

Table 4.9
VALUES USED FOR CONSTRUCTION WORKER DAILY TOTAL GROUNDWATER INTAKE EQUATIONS
DAVISVILLE SITE 16

Scenario Timeframe: Future
Medium: Groundwater
Exposure Medium: Tap Water
Exposure Point: Davisville Site 16
Receptor Population: Construction Worker
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CTE Value	CTE Rationale/Reference
Ingestion	CR	Ingestion Rate	L/day	0.05	U.S. EPA 1989a	0.025	U.S. EPA 1989a
	EF	Exposure Frequency	day/yr	160	U.S. EPA 1990	80	U.S. EPA 1990
	ED	Exposure Duration	yr	1	BPJ	1	BPJ
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	365	BPJ	365	BPJ
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
Dermal	SA	Surface Area for Contact	cm2	5,700	U.S. EPA 1992	2,300	U.S. EPA 1992
	PC	Permeability Coefficient	cm/hr	chemical-specific		chemical-specific	
	ET	Event Time	hr/day	8	U.S. EPA 1991	8	U.S. EPA 1991
	EF	Exposure Frequency	day/yr	160	U.S. EPA 1990	80	U.S. EPA 1990
	ED	Exposure Duration	yr	1	U.S. EPA 1991	1	U.S. EPA 1991
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	365	U.S. EPA 1989	365	U.S. EPA 1989
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989	25,550	U.S. EPA 1989
	CF	Conversion Factor	L/cm3	0.001		0.001	

Note : BPJ = Best Professional Judgement

Table 4.10
VALUES USED FOR ADULT RECREATIONAL USER DAILY SEEP WATER INTAKE EQUATIONS
DAVISVILLE SITE 16

Scenario Timeframe: Current/Future
Medium: Seep Water
Exposure Medium: Seep Water
Exposure Point: Davisville Site 16
Receptor Population: Recreational User
Receptor Age: Adult

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CTE Value	CTE Rationale/Reference
Ingestion	CR	Ingestion Rate	L/hr	0.05	U.S. EPA 1989a	0.03	U.S. EPA 1989a
	ET	Exposure Time	hr/day	1	BPJ	0.5	BPJ
	EF	Exposure Frequency	day/yr	7	BPJ	4	BPJ
	ED	Exposure Duration	yr	24	U.S. EPA 1991	1	U.S. EPA 1991
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time-Noncancer	days	8,760	U.S. EPA 1991	3,285	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
Dermal	SA	Surface Area for Contact	cm2	6,700	U.S. EPA 1997a	5,000	U.S. EPA 1997a
	PC	Permeability Coefficient	cm/hr	chemical-specific	U.S. EPA 1992	chemical-specific	U.S. EPA 1992
	ET	Exposure Time	hr/day	1	BPJ	0.5	BPJ
	EF	Exposure Frequency	day/yr	7	BPJ	4	BPJ
	ED	Exposure Duration	yr	24	BPJ	1	BPJ
	BW	Body Weight	kg	70	U.S. EPA 1991	70	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	8,760	U.S. EPA 1991	3,285	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	L/cm3	1.00E-03		1.00E-03	

Note : BPJ = Best Professional Judgement

Table 4.11
VALUES USED FOR CHILD RECREATIONAL USER DAILY SEEP WATER INTAKE EQUATIONS
DAVISVILLE SITE 16

Scenario Timeframe: Current/Future
Medium: Seep Water
Exposure Medium: Seep Water
Exposure Point: Davisville Site 16
Receptor Population: Recreational User
Receptor Age: Child

Exposure Route	Parameter Code	Parameter Definition	Units	RME Value	RME Rationale/Reference	CTE Value	CTE Rationale/Reference
Ingestion	CR	Ingestion Rate	L/hr	0.05	U.S. EPA 1989a	0.03	U.S. EPA 1989a
	ET	Exposure Time	hr/day	1	U.S. EPA 1997a	0.5	BPJ
	EF	Exposure Frequency	day/yr	7	BPJ	4	BPJ
	ED	Exposure Duration	yr	6	U.S. EPA 1991	2	U.S. EPA 1991
	BW	Body Weight	kg	15	U.S. EPA 1991	15	U.S. EPA 1991
	AT-NC	Averaging time-Noncancer	days	2,190	U.S. EPA 1991	730	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
Dermal	SA	Surface Area for Contact	cm2	3,300	U.S. EPA 1997a	2,700	U.S. EPA 1997a
	PC	Permeability Coefficient	cm/hr	chemical-specific	U.S. EPA 1992	chemical-specific	U.S. EPA 1992
	ET	Exposure Time	hr/day	1	U.S. EPA 1997a	0.5	BPJ
	EF	Exposure Frequency	day/yr	7	BPJ	4	BPJ
	ED	Exposure Duration	yr	6	U.S. EPA 1991	2	U.S. EPA 1991
	BW	Body Weight	kg	15	U.S. EPA 1991	15	U.S. EPA 1991
	AT-NC	Averaging time - Noncancer	days	2,190	U.S. EPA 1991	730	U.S. EPA 1991
	AT-C	Averaging Time - Cancer	days	25,550	U.S. EPA 1989a	25,550	U.S. EPA 1989a
	CF	Conversion Factor	L/cm3	1.00E-03		1.00E-03	

Note : BPJ = Best Professional Judgement

Table 5.1
Non-Cancer Toxicity Data - Oral//Dermal
Davisville Site 16

Chemical of Potential Concern	Chronic/ Subchronic	Oral RfD Value (mg/kg-day)	Oral to Dermal Adjustment Factor (GI ABS) (1)	Adjusted Dermal RfD (2) (mg/kg bw-day)	Primary Target Organ	Combined Uncertainty/Modifying Factors	Sources of RfD: Target Organ	Dates of RfD: Target Organ (3) (mm/dd/yy)
Dioxin/Furans								
TCDD-TEQ	NA	NA	1	NA	NA	NA/NA	HEAST	8/7/01
Inorganics								
ALUMINUM	Subchronic	1.00E+00	1	1.00E+00	Central Nervous System	100/3	EPA-NCEA	5/30/97
ANTIMONY	Chronic	4.00E-04	0.15	6.00E-05	Blood glucose and cholesterol	1000/1	IRIS	8/7/01
ARSENIC	Chronic	3.00E-04	1	3.00E-04	Skin	3/1	IRIS	8/7/01
BARIUM	Chronic	7.00E-02	0.07	4.90E-03	Kidneys	3/1	IRIS	8/7/01
COPPER	Chronic	4.00E-02	1	4.00E-02	Gastrointestional System	None	HEAST	7/25/97
MANGANESE	Chronic	2.00E-02	0.04	8.00E-04	Central Nervous System	1/1	IRIS	8/7/01
THALLIUM	Chronic	7.00E-05	1	7.00E-05	Liver, Blood	NA/NA	IRIS	8/7/01
PAHs								
BENZ(A)ANTHRACENE	NA	NA	1	NA	NA	NA/NA	IRIS	8/7/01
BENZO[A]PYRENE	NA	NA	1	NA	NA	NA/NA	IRIS	8/7/01
BENZO(B)FLUORANTHENE	NA	NA	1	NA	NA	NA/NA	IRIS	8/7/01
DIBENZ[A, H]ANTHRACENE	NA	NA	1	NA	NA	NA/NA	IRIS	8/7/01
INDENO(1,2,3-CD)PYRENE	NA	NA	1	NA	NA	NA/NA	IRIS	8/7/01
2-METHYLNAPHTHALENE	Chronic	2.00E-02	1	2.00E-02	Central Nervous System, Eyes	NA/NA	EPA-NCEA	5/23/01
NAPHTHALENE	Subchronic	2.00E-02	1	2.00E-02	None	3000/1	IRIS	8/7/01
Pesticides/PCBs								
ALPHA-BHC	NA	NA	1	NA	NA	NA/NA	IRIS	8/7/01
DIELDRIN	NA	5.00E-05	1	5.00E-05	NA	NA/NA	IRIS	8/15/01
HEPTACHLOR EPOXIDE	Chronic	1.30E-05	1	1.30E-05	Liver, Central Nervous System	1000/1	IRIS	8/7/01
Semivolatiles								
BIS(2-ETHYLHEXYL)PHTHALATE	Chronic	2.20E-02	1	2.20E-02	Liver	1000/1	IRIS	8/15/01
1,4-DICHLOROBENZENE	Chronic	3.00E-02	1	3.00E-02	Liver	1000/1	EPA-NCEA	4/29/97
DIBENZOFURAN	Chronic	4.00E-03	1	4.00E-03	Liver, Kidney	300/1	EPA-NCEA	7/19/99
Volatiles								
1,1,2-TRICHLOROETHANE	Chronic	4.00E-03	1	4.00E-03	Respiratory System	1000/1	IRIS	8/15/01
1,1-DICHLOROETHENE	Chronic	1.00E-01	1	1.00E-01	Central Nervous System	1000/1	IRIS	8/15/01
1,2-DICHLOROETHANE	Chronic	3.00E-02	1	3.00E-02	Central Nervous System	1000/1	EPA-NCEA	4/5/93
ACETONE	Subchronic	1.00E-01	1	1.00E-01	Liver, Kidney	1000/1	IRIS	8/15/01
BENZENE	Chronic	3.00E-03	1	3.00E-03	Central Nervous System	1000/1	EPA-NCEA	9/1/98
CHLOROFORM	Chronic	1.00E-02	1	1.00E-02	Liver, Kidneys, Central Nervous System	1000/1	IRIS	8/7/01
CIS-1,2-DICHLOROETHENE	Chronic	1.00E-02	1	1.00E-02	Liver	3000/1	HEAST	5/1/95
TOTAL 1,2-DICHLOROETHENE	Chronic	2.00E-02	1	2.00E-02	Liver	3000/1	HEAST	5/1/95
TRICHLOROETHENE	Chronic	6.00E-03	1	6.00E-03	Central Nervous System, Liver, Kidney		EPA-NCEA	
VINYL CHLORIDE	Chronic	3.00E-03	1	3.00E-03	Liver	30/1	IRIS	8/15/01

N/A= Not Applicable

(1) Taken from USEPA 2000 Guidance. USEPA, 2000. *Risk Assessment Guidance for Superfund, volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment)*. Interim Guidance.

(2) Dermal toxicological values adjusted from oral values using USEPA 2000 recommended chemical-specific gastrointestinal absorption factors(GI ABS). RfDs are multiplied by the GI ABS.

(3) IRIS - Integrated Risk Information System. For IRIS values, the date IRIS was searched is provided.
HEAST - Health Effects Assessment Summary Tables. For HEAST values, the date of HEAST is provided.
EPA-NCEA - National Center for Environmental Assessment. For EPA-NCEA values, the date of the article provided by EPA-NCEA is provided.

Table 5.2
Non-Cancer Toxicity Data - Inhalation
Davisville Site 16

Chemical of Potential Concern	Chronic/ Subchronic	Value Inhalation (RfD) (mg/kg-day)	Primary Target Organ	Combined Uncertainty/Modifying Factors	Sources of RfC:RfD: Target.Organ	Dates (1) (mm/dd/yy)
Dioxin/Furans						
TCDD-TEQ	NA	NA	None	NA/NA		
Inorganics						
ALUMINUM	Chronic	1.00E-03	Respiratory System	NA/NA	EPA-NCEA	5/30/97
ANTIMONY	NA	NA	None	NA/NA	IRIS	8/7/01
ARSENIC	NA	NA	None	NA/NA	HEAST	7/25/97
BARIUM	Chronic	1.40E-04	Kidneys	10/1	EPA-NCEA	10/7/99
COPPER	NA	NA	None	NA/NA	HEAST	7/25/97
MANGANESE	Chronic	1.43E-05	Central Nervous System	1000/1	IRIS	8/15/01
THALLIUM	NA	NA	None	NA/NA	IRIS	8/15/01
PAHs						
BENZ(A)ANTHRACENE	NA	NA	None	NA/NA	EPA-NCEA	5/23/01
BENZO[A]PYRENE	NA	NA	None	NA/NA	IRIS	8/7/01
BENZO(B)FLUORANTHENE	NA	NA	None	NA/NA	EPA-NCEA	5/23/01
DIBENZ[A, H]ANTHRACENE	NA	NA	None	NA/NA	EPA-NCEA	5/23/01
INDENO(1,2,3-CD)PYRENE	NA	NA	None	NA/NA	EPA-NCEA	5/23/01
2-METHYLNAPHTHALENE	NA	NA	None	NA/NA	EPA-NCEA	5/23/01
NAPHTHALENE	Chronic	9.00E-04	Respiratory System	3000/1	IRIS	8/7/01
Pesticides/PCDDs/PCDFs						
ALPHA BHC	NA	NA	None	NA/NA	IRIS	8/15/01
DIELDRIN	NA	NA	None	NA/NA	IRIS	8/15/01
HEPTACHLOR EPOXIDE	NA	NA	None	NA/NA	IRIS	8/15/01
Semivolatiles						
BIS(2-ETHYLHEXYL)PHTHALATE	NA	NA	None	NA/NA	IRIS	8/15/01
CARBAZOLE	NA	NA	None	NA/NA	EPA-NCEA	10/7/99
1,4-DICHLOROBENZENE	Chronic	2.30E-01	Liver	30/1	IRIS	8/15/01
2,4-DIMETHYLPHENOL	NA	NA	None	NA/NA	IRIS	8/7/01
DIBENZOFURAN	NA	NA	None	NA/NA	NA	NA
2-METHYLPHENOL	NA	NA	None	NA/NA	IRIS	8/15/01
4-METHYLPHENOL	NA	NA	None	NA/NA	HEAST	7/25/97
Volatiles						
1,1,2-TRICHLOROETHANE	NA	NA	None	NA/NA	IRIS	8/15/01
1,1-DICHLOROETHENE	NA	NA	None	NA/NA	IRIS	8/15/01
1,2-DICHLOROETHANE	Chronic	1.40E-03	Central Nervous System	NA/NA		
ACETONE	NA	NA	None	NA/NA	IRIS	8/15/01
BENZENE	Chronic	1.71E-03	Central Nervous System	1000/1	EPA-NCEA	9/1/98
CHLOROFORM	Chronic	8.60E-05	Liver, Kidneys, Central Nervous System	1000/1	IRIS	8/7/01
CIS-1,2-DICHLOROETHENE	NA	NA	None	NA/NA	NA	NA
TOTAL 1,2-DICHLOROETHENE	NA	NA	None	NA/NA	IRIS	8/15/01
TRICHLOROETHENE	Chronic	NA	Liver	30/1	IRIS	8/15/01
VINYL CHLORIDE-adult/child	Chronic	2.90E-02	Liver	30/1	IRIS	8/15/01

N/A= Not Applicable
(1) For IRIS values, the date IRIS was searched is provided.
For HEAST values, the date of HEAST is provided.
For EPA-NCEA values, the date of the article provided by NCEA is provided.

Table 5.3
Chemical-Specific Parameters
Davisville Site 16

Chemical of Potential Concern	Absorption Factor	Reference	Permeability Constant (cm/hr)	Reference
Dioxin/Furans				
TCDD-TEQ	0.03	U.S. EPA, 2000	NA	On-line Database(1)
Inorganics				
ALUMINUM	NA	U.S. EPA, 2000	1.00E-03	On-line Database(1)
ANTIMONY	NA	U.S. EPA, 2000	1.00E-03	On-line Database(1)
ARSENIC	0.03	U.S. EPA, 2000	1.00E-03	On-line Database(1)
BARIUM	NA	U.S. EPA, 2000	1.00E-03	On-line Database(1)
COPPER	NA	U.S. EPA, 2000	1.00E-03	On-line Database(1)
MANGANESE	NA	U.S. EPA, 2000	1.00E-03	On-line Database(1)
THALLIUM	NA	U.S. EPA, 2000	1.00E-03	On-line Database(1)
PAHs				
BENZ(A)ANTHRACENE	0.13	U.S. EPA, 2000	8.10E-01	On-line Database(1)
BENZO[A]PYRENE	0.13	U.S. EPA, 2000	1.20E+00	On-line Database(1)
BENZO(B)FLUORANTHENE	0.13	U.S. EPA, 2000	1.20E+00	On-line Database(1)
DIBENZ[A, H]ANTHRACENE	0.13	U.S. EPA, 2000	2.70E+00	On-line Database(1)
INDENO(1,2,3-CD)PYRENE	0.13	U.S. EPA, 2000	1.90E+00	On-line Database(1)
2-METHYLNAPHTHALENE	0.13	U.S. EPA, 2000	6.90E-02	On-line Database(1)
NAPHTHALENE	0.13	U.S. EPA, 2000	6.90E-02	On-line Database(1)
Pesticides/PCDDs/PCDFs				
ALPHA BHC	0.1	U.S. EPA, 2000	1.90E-02	On-line Database(1)
DIELDRIN	0.1	U.S. EPA, 2000	1.60E-03	On-line Database(1)
HEPTACHLOR EPOXIDE	0.1	U.S. EPA, 2000	1.10E-02	On-line Database(1)
Semivolatiles				
BIS(2-ETHYLHEXYL)PHTHALATE	0.1	U.S. EPA, 2000	2.30E-02	On-line Database(1)
CARBAZOLE	0.1	U.S. EPA, 2000	9.10E-02	On-line Database(1)
1,4-DICHLOROBENZENE	0.1	U.S. EPA, 2000	6.20E-02	On-line Database(1)
2,4-DIMETHYLPHENOL	0.1	U.S. EPA, 2000	1.10E-01	On-line Database(1)
DIBENZOFURAN	0.03	U.S. EPA, 2000	6.10E-02	On-line Database(1)
2-METHYLPHENOL	0.1	U.S. EPA, 2000	1.60E-02	On-line Database(1)
Volatiles				
1,1,2-TRICHLOROETHANE	NA	U.S. EPA, 2000	NA	On-line Database(1)
1,1-DICHLOROETHENE	NA	U.S. EPA, 2000	8.40E-03	On-line Database(1)
1,2-DICHLOROETHANE	NA	U.S. EPA, 2000	8.90E-03	On-line Database(1)
ACETONE	NA	U.S. EPA, 2000	3.30E-03	On-line Database(1)
BENZENE	NA	U.S. EPA, 2000	5.70E-04	On-line Database(1)
CHLOROFORM	NA	U.S. EPA, 2000	5.80E-03	On-line Database(1)
CIS-1,2-DICHLOROETHENE	NA	U.S. EPA, 2000	8.90E-03	On-line Database(1)
TOTAL 1,2-DICHLOROETHENE	NA	U.S. EPA, 2000	1.00E-02	On-line Database(1)
TRICHLOROETHENE	NA	U.S. EPA, 2000	4.50E-02	On-line Database(1)
VINYL CHLORIDE-adult/child	NA	U.S. EPA, 2000	1.60E-02	On-line Database(1)

(1) Toxicity and Chemical-Specific Factors Database. Http://risk.lsd.ornl.gov/cgi-bin/tox. July 2001

Table 6.1
Cancer Toxicity Data - Oral/Dermal
Davisville Site 16

Chemical of Potential Concern	Oral Cancer Slope Factor	Oral to Dermal Adjustment Factor (GI ABS)[1]	Adjusted Cancer Slope Factor [2]	Units	Weight of Evidence/Cancer Guideline Description	Source	Date [3] (mm/dd/yy)
Dioxin/Furans							
TCDD-TEQ	1.50E+05	1	1.50E+05	per (mg/kg-day)			8/7/01
Inorganics							
ALUMINUM	NA	1	NA	per (mg/kg-day)	D	EPA-NCEA	5/30/97
ANTIMONY	NA	0.15	NA	per (mg/kg-day)	D	IRIS	8/7/01
ARSENIC	1.50E+00	1	1.50E+00	per (mg/kg-day)	A	IRIS	8/7/01
BARIUM	NA	0.07	NA	per (mg/kg-day)	D	EPA-NCEA	5/23/01
COPPER	NA	1	NA	per (mg/kg-day)	D	IRIS	8/7/01
MANGANESE	NA	0.04	NA	per (mg/kg-day)	D	IRIS	8/7/01
THALLIUM	NA	1	NA	per (mg/kg-day)	D	IRIS	8/7/01
PAHs							
BENZ(A)ANTHRACENE	7.30E-01	1	7.30E-01	per (mg/kg-day)	B2	EPA-NCEA	7/1/93
BENZO[A]PYRENE	7.30E+00	1	7.30E+00	per (mg/kg-day)	B2	IRIS	8/7/01
BENZO(B)FLUORANTHENE	7.30E-01	1	7.30E-01	per (mg/kg-day)	B2	EPA-NCEA	7/1/93
DIBENZ[A, H]ANTHRACENE	7.30E+00	1	7.30E+00	per (mg/kg-day)	B2	EPA-NCEA	5/23/01
INDENO(1,2,3-CD)PYRENE	7.30E-01	1	7.30E-01	per (mg/kg-day)	B2	EPA-NCEA	7/1/93
2-METHYLNAPHTHALENE	NA	1	NA	per (mg/kg-day)	C	EPA-NCEA	5/23/01
NAPHTHALENE	NA	1	NA	per (mg/kg-day)	C	IRIS	8/7/01
Pesticides/PCDDs/PCDFs							
ALPHA BHC	6.30E+00	1	6.30E+00	per (mg/kg-day)	B2	IRIS	8/7/01
DIELDRIN	1.60E+01	1	1.60E+01	per (mg/kg-day)	D	IRIS	8/15/01
HEPTACHLOR EPOXIDE	9.10E+00	1	9.10E+00	per (mg/kg-day)	B2	IRIS	8/7/01
Semivolatiles							
BIS(2-ETHYLHEXYL)PHTHALATE	1.40E-02	1	1.40E-02	per (mg/kg-day)	B2	IRIS	8/15/01
1,4-DICHLOROBENZENE	2.40E-02	1	2.40E-02	per (mg/kg-day)	NA	HEAST	7/25/97
DIBENZOFURAN	NA	1	NA	per (mg/kg-day)	D	IRIS	8/7/01
Volatiles							
1,1,2-TRICHLOROETHANE	5.70E-02	1	5.70E-02	per (mg/kg-day)	C	IRIS	8/15/01
1,1-DICHLOROETHENE	NA	1	NA	per (mg/kg-day)	NA	IRIS	8/15/01
1,2-DICHLOROETHANE	9.10E-02	1	9.10E-02	per (mg/kg-day)	B2	IRIS	8/15/01
ACETONE	NA	1	NA	per (mg/kg-day)	NA	IRIS	8/15/01
BENZENE	5.50E-02	1	5.50E-02	per (mg/kg-day)	A	IRIS	8/7/01
CHLOROFORM	6.10E-03	1	6.10E-03	per (mg/kg-day)	B2	IRIS	8/7/01
CIS-1,2-DICHLOROETHENE	NA	1	NA	per (mg/kg-day)	D	IRIS	8/15/01
TOTAL 1,2-DICHLOROETHENE	NA	1	NA	per (mg/kg-day)	D	IRIS	8/15/01
TRICHLOROETHENE	1.10E-02	1	1.10E-02	per (mg/kg-day)	NA	EPA-NCEA	5/23/01
VINYL CHLORIDE-adult/child	1.50E+00	1	1.50E+00	per (mg/kg-day)	A	IRIS	8/15/01
VINYL CHLORIDE-adult	7.50E-01	1	7.50E-01	per (mg/kg-day)	A	IRIS	8/15/01

N/A= Not Applicable
(1) Taken from USEPA 2000 Guidance.
(2) Dermal Toxicological values adjusted from oral values using USEPA 2000 recommended chemical-specific gastrointestinal absorption factors(GI ABS). CSFs are divided be the GI ABS.
(3) For IRIS values, the date IRIS was searched is provided.
For HEAST values, the date of HEAST is provided.
For EPA-NCEA values, the date of the article provided by NCEA is provided.

Weight of Evidence: A - Human carcinogen
B1 - Probable human carcinogen - indicate that llimited human data are available
B2 - Probable human carcinogen - indicates sufficient evidence in animals and inadequate or no evidence in humans
C - Posible human carcinogen
D - Not classifiable as a human carcinogen
E - Evidence of noncarcinogenicity

Table 6.2
Cancer Toxicity Data - Inhalation
Davisville Site 16

Chemical of Potential Concern	Chronic/ Subchronic	Inhalation Cancer Slope Factor	Units	Weight of Evidence/Cancer Guideline Description	Source	Date (1)
Dioxin/Furans						
TCDD-TEQ	Chronic	1.50E+05	per (mg/kg-day)			8/7/01
Inorganics						
ALUMINUM	NA	NA	per (mg/kg-day)	D	EPA-NCEA	5/30/97
ANTIMONY	NA	NA	per (mg/kg-day)	A	IRIS	8/7/01
ARSENIC	Chronic	1.51E+01	per (mg/kg-day)	D	IRIS	8/7/01
BARIUM	NA	NA	per (mg/kg-day)	NA	IRIS	8/7/01
COPPER	NA	NA	per (mg/kg-day)	D	IRIS	8/7/01
MANGANESE	NA	NA	per (mg/kg-day)	D	IRIS	8/7/01
THALLIUM	NA	NA	per (mg/kg-day)	D	IRIS	8/7/01
PAHs						
BENZ(A)ANTHRACENE	Chronic	3.10E-01	per (mg/kg-day)	B2	EPA-NCEA	7/1/93
BENZO[A]PYRENE	Chronic	3.10E+00	per (mg/kg-day)	B2	IRIS	8/7/01
BENZO(B)FLUORANTHENE	Chronic	3.10E-01	per (mg/kg-day)	B2	EPA-NCEA	7/1/93
DIBENZ[A, H]ANTHRACENE	NA	NA	per (mg/kg-day)	B2	EPA-NCEA	7/1/93
INDENO(1,2,3-CD)PYRENE	Chronic	3.10E-01	per (mg/kg-day)	B2	EPA-NCEA	7/1/93
2-METHYLNAPHTHALENE	NA	NA	per (mg/kg-day)	C	EPA-NCEA	5/23/01
NAPHTHALENE	NA	NA	per (mg/kg-day)	C	IRIS	8/7/01
Pesticides/PCDDs/PCDFs						
ALPHA BHC	Chronic	6.30E+00	per (mg/kg-day)	B2	IRIS	8/7/01
DIELDRIN	Chronic	1.60E+01	per (mg/kg-day)	B2	IRIS	8/7/01
HEPTACHLOR EPOXIDE	Chronic	9.10E+00	per (mg/kg-day)	B2	IRIS	8/7/01
Semivolatiles						
BIS(2-ETHYLHEXYL)PHTHALATE	NA	NA	per (mg/kg-day)	B2	IRIS	8/15/01
1,4-DICHLOROBENZENE	Chronic	2.20E-02	per (mg/kg-day)	NA	IRIS	8/15/01
DIBENZOFURAN	NA	NA	per (mg/kg-day)	D	IRIS	8/7/01
Volatiles						
1,1,2-TRICHLOROETHANE	Chronic	5.60E-02	per (mg/kg-day)	C	IRIS	8/15/01
1,1-DICHLOROETHENE	NA	NA	per (mg/kg-day)	NA	IRIS	8/15/01
1,2-DICHLOROETHANE	Chronic	9.10E-02	per (mg/kg-day)	B2	IRIS	8/15/01
ACETONE	NA	NA	per (mg/kg-day)	NA	IRIS	8/15/01
BENZENE	Chronic	2.90E-02	per (mg/kg-day)	A	IRIS	8/7/01
CHLOROFORM	Chronic	8.05E-02	per (mg/kg-day)	B2	IRIS	8/7/01
CIS-1,2-DICHLOROETHENE	NA	NA	per (mg/kg-day)	D	IRIS	8/15/01
TOTAL 1,2-DICHLOROETHENE	NA	NA	per (mg/kg-day)	D	IRIS	8/15/01
TRICHLOROETHENE	Chronic	6.00E-03	per (mg/kg-day)	NA	EPA-NCEA	
VINYL CHLORIDE-adult/child	Chronic	3.10E-02	per (mg/kg-day)	A	IRIS	8/15/01
VINYL CHLORIDE-adult	Chronic	1.60E-02	per (mg/kg-day)	A	IRIS	8/15/01

N/A= Not Applicable

(1) For IRIS values, the date IRIS was searched is provided.
For HEAST values, the date of HEAST is provided.
For EPA-NCEA values, the date of the article provided by NCEA is provided.

Weight of Evidence: A - Human carcinogen
B1 - Probable human carcinogen - indicate that llimited human data are available
B2 - Probable human carcinogen - indicates sufficient evidence in animals and inadequate or no evidence in humans
C - Posible human carcinogen
D - Not classifiable as a human carcinogen
E - Evidence of noncarcinogenicity

TABLE 7.1
IEUBK Model Results for Soil and Groundwater
Davisville Site 16

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 3.63 ug Pb/L; WATER Consumption: DEFAULT

SOIL & DUST: Soil: constant conc.; Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	160.0	122.0
1-2	160.0	122.0
2-3	160.0	122.0
3-4	160.0	122.0
4-5	160.0	122.0
5-6	160.0	122.0
6-7	160.0	122.0

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.70
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model; Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	3.4	6.24	3.30
1-2:	3.7	8.77	5.22
2-3:	3.4	9.23	5.26
3-4:	3.2	9.23	5.31
4-5:	2.8	7.90	4.00
5-6:	2.4	7.78	3.62
6-7:	2.3	7.93	3.43

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.58	0.34	0.00	0.02
1-2:	2.68	0.84	0.00	0.03
2-3:	3.03	0.88	0.00	0.06
3-4:	2.94	0.91	0.00	0.07
4-5:	2.88	0.96	0.00	0.07
5-6:	3.05	1.01	0.00	0.09
6-7:	3.38	1.03	0.00	0.09

TABLE 7.2
IEUBK Model Results for Soil and Seep Water
Davisville Site 16

LEAD MODEL Version 0.99d

AIR CONCENTRATION:0.100 ug Pb/m3; DEFAULT Indoor AIR Pb Conc:30.0% of outdoor. Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT
DRINKING WATER Conc: 13.10 ug Pb/L; Other WATER Parameters (non-default):

Age	Water Consumption (L/day)
0-1	0.10
1-2	0.25
2-3	0.26
3-4	0.27
4-5	0.28
5-6	0.29
6-7	0.30

SOIL & DUST: Soil: constant conc.; Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	160.0	122.0
1-2	160.0	122.0
2-3	160.0	122.0
3-4	160.0	122.0
4-5	160.0	122.0
5-6	160.0	122.0
6-7	160.0	122.0

Additional Dust Sources: None DEFAULT; Soil contribution conversion factor: 0.70
Air contribution conversion factor: 100.0
PAINT Intake: 0.00 ug Pb/day DEFAULT
MATERNAL CONTRIBUTION: Infant Model; Maternal Blood Conc: 2.50 ug Pb/dL

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	3.5	6.49	3.29
1-2:	3.9	9.39	5.19
2-3:	3.7	9.89	5.23
3-4:	3.5	9.95	5.29
4-5:	3.0	8.66	3.98
5-6:	2.7	8.56	3.60
6-7:	2.5	8.76	3.41

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.57	0.61	0.00	0.02
1-2:	2.66	1.51	0.00	0.03
2-3:	3.02	1.58	0.00	0.06
3-4:	2.93	1.66	0.00	0.07
4-5:	2.87	1.75	0.00	0.07
5-6:	3.04	1.82	0.00	0.09
6-7:	3.37	1.89	0.00	0.09

Table 8.1
Calculation of Lead Risk Based on EPA TRW Model
Construction Worker Scenario
Davisville Site 16

Parameter	Units	Reference
Central estimate of blood lead concentrations = $PbB_{adult,central}$	Calculated µg/dL	
Goal for blood lead concentration among fetuses = $PbB_{fetal,0.95}$	Calculated µg/dL	
Typical adult blood level concentration = $PbB_{adult,0}$	2.0 µg/dL	EPA, 1996
Soil lead concentration = PbS	160 µg/g	Site-Specific
Biokinetic slope factor = BKSF	0.4 µg/dL per µg/day	EPA, 1996
Intake rate of Soil = IR_S	0.1 g/day	EPA, 1991; FAQ sheet
Absolute gastrointestional absorption fraction = AF_S	0.12	EPA, 1996
Exposure frequency = EF_S	160 day/yr	EPA, 1990
Averaging Time = AT	365 days/yr	EPA, 1996
Estimated Value of the individual geometric standard deviation = GSD	2.0 dimensionless	EPA, 1996
Constant of proportionality between fetal blood lead conc. and maternal blood lead conc. = R	0.9 dimensionless	EPA, 1996

Blood Lead Level Calculations:

$$PbB_{adult,\ central} = PbB_{adult,0} + PbS*BKSF*IR_S*AF_S*EF_S/AT$$

$$PbB_{adult,central} = 2.34\ \mu g/dL$$

$$PbB_{fetal,0.95} = PbB_{adult,central} * GSD^{1.645} * R_{fetal/maternal}$$

$$PbB_{fetal,0.95} = 6.58\ \mu g/dL$$

FAQ sheet = Frequently Asked Questions document developed by EPA for the TRW model

Table 9.1
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY EXPOSURE SCENARIO
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child and Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Total Soil Child	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	6.7E-07	3.5E-08	2.1E-11	7.0E-07	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ALUMINUM	--	--	--	NA	ALUMINUM	Central Nervous System	1.5E-02	0.0E+00	4.8E-04	1.6E-02
			ANTIMONY	--	--	--	NA	ANTIMONY	Blood glucose and cholesterol	6.6E-03	0.0E+00	--	6.6E-03
			ARSENIC	2.9E-07	1.5E-08	9.1E-11	3.0E-07	ARSENIC	Skin	2.2E-02	1.2E-03	--	2.4E-02
			BARIUM	--	--	--	NA	BARIUM	Kidneys	3.1E-03	0.0E+00	4.8E-05	3.1E-03
			COPPER	--	--	--	NA	COPPER	Gastrointestional System	2.9E-03	0.0E+00	--	2.9E-03
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	2.3E-02	0.0E+00	1.0E-03	2.4E-02
			THALLIUM	--	--	--	NA	THALLIUM	Liver, Blood	1.1E-02	0.0E+00	--	1.1E-02
			PAHs					**PAHs**					
			BENZ(A)ANTHRACENE	1.8E-08	4.1E-09	2.4E-13	2.2E-08	BENZ(A)ANTHRACENE	NA	--	--	--	NA
			BENZO[A]PYRENE	1.7E-07	3.7E-08	2.2E-12	2.0E-07	BENZO[A]PYRENE	NA	--	--	--	NA
			BENZO(B)FLUORANTHENE	2.4E-08	5.5E-09	3.3E-13	3.0E-08	BENZO(B)FLUORANTHENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	8.6E-08	2.0E-08	--	1.1E-07	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			INDENO(1,2,3-CD)PYRENE	1.3E-08	3.0E-09	1.8E-13	1.6E-08	INDENO(1,2,3-CD)PYRENE	NA	--	--	--	NA
			2-METHYLNAPHTHALENE	--	--	--	NA	2-METHYLNAPHTHALENE	Central Nervous System, Eyes	1.6E-04	3.7E-05	--	2.0E-04
			NAPHTHALENE	--	--	--	NA	NAPHTHALENE	None	5.4E-05	1.2E-05	3.8E-08	6.6E-05
			(Total for Child)	1.3E-06	1.2E-07	1.2E-10	1.4E-06		(Total for Child)	8.4E-02	1.2E-03	1.5E-03	8.7E-02
	Total Soil Adult	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	3.2E-07	1.20E-08	2.68E-11	3.3E-07	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ALUMINUM	--	--	--	NA	ALUMINUM	Central Nervous System	1.6E-03	0.0E+00	1.8E-04	1.8E-03
			ANTIMONY	--	--	--	NA	ANTIMONY	Blood glucose and cholesterol	7.1E-04	0.0E+00	--	7.1E-04
			ARSENIC	1.4E-07	5.16E-09	1.16E-10	1.4E-07	ARSENIC	Skin	2.4E-03	8.9E-05	--	2.5E-03
			BARIUM	--	--	--	NA	BARIUM	Kidneys	3.3E-04	0.0E+00	1.8E-05	3.5E-04
			COPPER	--	--	--	NA	COPPER	Gastrointestional System	3.1E-04	0.0E+00	--	3.1E-04
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	2.4E-03	0.0E+00	3.7E-04	2.8E-03
			THALLIUM	--	--	--	NA	THALLIUM	Liver, Blood	1.2E-03	0.0E+00	--	1.2E-03
			PAHs					**PAHs**					
			BENZ(A)ANTHRACENE	8.8E-09	1.42E-09	3.11E-13	1.0E-08	BENZ(A)ANTHRACENE	NA	--	--	--	NA
			BENZO[A]PYRENE	8.0E-08	1.28E-08	2.82E-12	9.2E-08	BENZO[A]PYRENE	NA	--	--	--	NA
			BENZO(B)FLUORANTHENE	1.2E-08	1.90E-09	4.16E-13	1.4E-08	BENZO(B)FLUORANTHENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	4.2E-08	6.71E-09	--	4.8E-08	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			INDENO(1,2,3-CD)PYRENE	6.4E-09	1.03E-09	2.25E-13	7.4E-09	INDENO(1,2,3-CD)PYRENE	NA	--	--	--	NA
			2-METHYLNAPHTHALENE	--	--	--	NA	2-METHYLNAPHTHALENE	Central Nervous System, Eyes	1.7E-05	2.8E-06	--	2.0E-05
			NAPHTHALENE	--	--	--	NA	NAPHTHALENE	None	5.8E-06	9.3E-07	1.4E-08	6.7E-06
			(Total for Adult)	6.1E-07	4.1E-08	1.5E-10	6.5E-07		(Total for Adult)	9.0E-03	9.3E-05	5.6E-04	9.6E-03
	Total Soil Adult + Child	Site 16	**Dioxin/Furans**										
			TCDD-TEQ	9.9E-07	4.7E-08	4.8E-11	1.0E-06						
			Explosives										
			2,4,6-TRINITROTOLUENE	NA	NA	NA	NA						
			Inorganics										
			ALUMINUM	NA	NA	NA	NA						
			ANTIMONY	NA	NA	NA	NA						
			ARSENIC	4.3E-07	2.0E-08	2.1E-10	4.5E-07						
			BARIUM	NA	NA	NA	NA						
			COPPER	NA	NA	NA	NA						
			MANGANESE	NA	NA	NA	NA						
			THALLIUM	NA	NA	NA	NA						
			PAHs										
			BENZ(A)ANTHRACENE	2.7E-08	5.5E-09	5.6E-13	3.3E-08						
			BENZO[A]PYRENE	2.4E-07	5.0E-08	5.0E-12	2.9E-07						
			BENZO(B)FLUORANTHENE	3.6E-08	7.4E-09	7.4E-13	4.4E-08						
			DIBENZ[A, H]ANTHRACENE	1.3E-07	2.6E-08	NA	1.5E-07						
			INDENO(1,2,3-CD)PYRENE	2.0E-08	4.0E-09	4.0E-13	2.4E-08						
			2-METHYLNAPHTHALENE	NA	NA	NA	NA						
			NAPHTHALENE	NA	NA	NA	NA						
			(Total for Child + Adult)	6.4E-04	5.5E-05	8.9E-08	2.0E-06						
				Total Risk Across Total Soil			2.0E-06		Total Hazard Index Across Total Soil (Child)				8.7E-02
									Total Hazard Index Across Total Soil (Adult)				9.6E-03

Table 9.1
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY EXPOSURE SCENARIO
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child and Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Groundwater	Groundwater	Site 16	**Inorganics**					**Inorganics**					
			BARIUM	--	--	--	NA	BARIUM	Kidneys	2.1E-02	1.2E-03	--	2.2E-02
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	1.0E+00	1.0E-01	--	1.1E+00
			Semivolatiles					**Semivolatiles**					
			BIS(2-ETHYLHEXYL)PHTHALATE	7.2E-08	6.7E-09	--	7.9E-08	BIS(2-ETHYLHEXYL)PHTHALATE	Liver	8.2E-03	7.6E-04	--	9.0E-03
			1,4-DICHLOROBENZENE	1.1E-07	2.8E-08	--	1.4E-07	1,4-DICHLOROBENZENE	Liver	5.6E-03	1.4E-03	--	6.9E-03
			Volatiles					**Volatiles**					
			1,1,2-TRICHLOROETHANE	6.1E-08	2.0E-09	--	6.3E-08	1,1,2-TRICHLOROETHANE	Respiratory System	9.3E-03	3.1E-04	--	9.6E-03
			1,1-DICHLOROETHENE	--	--	--	NA	1,1-DICHLOROETHENE	Central Nervous System	4.0E-04	1.4E-05	--	4.2E-04
			1,2-DICHLOROETHANE	9.5E-08	2.0E-09	--	9.7E-08	1,2-DICHLOROETHANE	Central Nervous System	1.2E-03	2.6E-05	--	1.2E-03
			ACETONE	--	--	--	NA	ACETONE	Liver, Kidney	3.3E-03	7.6E-06	--	3.3E-03
			BENZENE	5.9E-08	5.0E-09	--	6.4E-08	BENZENE	Central Nervous System	1.3E-02	1.1E-03	--	1.4E-02
			CHLOROFORM	6.5E-09	2.3E-10	--	6.7E-09	CHLOROFORM	Liver, Kidneys, Central Nervous System	3.7E-03	1.3E-04	--	3.8E-03
			CIS-1,2-DICHLOROETHENE	--	--	--	NA	CIS-1,2-DICHLOROETHENE	Liver	2.1E-02	8.2E-04	--	2.1E-02
			TOTAL 1,2-DICHLOROETHENE	--	--	--	NA	TOTAL 1,2-DICHLOROETHENE	Liver	1.0E-02	4.5E-05	--	1.0E-02
			TRICHLOROETHENE	9.5E-06	6.1E-07	--	1.0E-05	TRICHLOROETHENE	Central Nervous System, Liver, Kidney	5.0E+00	3.2E-01	--	5.3E+00
			VINYL CHLORIDE	3.2E-06	9.4E-08	--	3.3E-06	VINYL CHLORIDE	Liver	2.5E-02	7.3E-04	--	2.6E-02
			(Total for Child)	1.3E-05	7.5E-07	---	1.4E-05		(Total for Child)	6.1E+00	4.3E-01	---	6.6E+00
	Groundwater Adult	Site 16	**Inorganics**					**Inorganics**					
			BARIUM	--	--	--	NA	BARIUM	Kidneys	8.5E-03	2.64E-04	--	8.8E-03
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	4.0E-01	2.19E-02	--	4.3E-01
			Semivolatiles					**Semivolatiles**					
			BIS(2-ETHYLHEXYL)PHTHALATE	1.3E-07	6.56E-09	--	1.4E-07	BIS(2-ETHYLHEXYL)PHTHALATE	Liver	3.3E-03	1.66E-04	0.0E+00	3.5E-03
			1,4-DICHLOROBENZENE	2.1E-07	2.81E-08	--	2.4E-07	1,4-DICHLOROBENZENE	Liver	2.3E-03	3.03E-04	0.0E+00	2.6E-03
			Volatiles					**Volatiles**					
			1,1,2-TRICHLOROETHANE	1.1E-07	2.01E-09	6.1E-08	1.7E-07	1,1,2-TRICHLOROETHANE	Respiratory System	3.8E-03	6.86E-05	--	3.8E-03
			1,1-DICHLOROETHENE	--	--	--	NA	1,1-DICHLOROETHENE	Central Nervous System	1.6E-04	3.14E-06	--	1.7E-04
			1,2-DICHLOROETHANE	1.7E-07	1.99E-09	1.1E-07	2.8E-07	1,2-DICHLOROETHANE	Central Nervous System	4.9E-04	5.66E-06	1.4E-02	1.4E-02
			ACETONE	--	--	--	NA	ACETONE	Liver, Kidney	1.4E-03	1.67E-06	--	1.4E-03
			BENZENE	1.1E-07	4.89E-09	4.5E-08	1.6E-07	BENZENE	Central Nervous System	5.1E-03	2.31E-04	1.5E-02	2.0E-02
			CHLOROFORM	1.2E-08	2.28E-10	1.0E-07	1.2E-07	CHLOROFORM	Liver, Kidneys, Central Nervous System	1.5E-03	2.91E-05	2.4E-01	2.5E-01
			CIS-1,2-DICHLOROETHENE	--	--	--	NA	CIS-1,2-DICHLOROETHENE	Liver	8.3E-03	1.80E-04	--	8.5E-03
			TOTAL 1,2-DICHLOROETHENE	--	--	--	NA	TOTAL 1,2-DICHLOROETHENE	Liver	4.1E-03	9.86E-06	--	4.1E-03
			TRICHLOROETHENE	1.7E-05	5.98E-07	6.3E-06	2.4E-05	TRICHLOROETHENE	Central Nervous System, Liver, Kidney	2.0E+00	7.05E-02	--	2.1E+00
			VINYL CHLORIDE	5.9E-06	9.28E-08	8.7E-08	6.0E-06	VINYL CHLORIDE	Liver	1.0E-02	1.60E-04	1.6E-03	1.2E-02
			(Total for Adult)	2.4E-05	7.3E-07	6.7E-06	3.1E-05		(Total for Adult)	2.5E+00	9.4E-02	2.7E-01	2.9E+00
	Groundwater Adult + Child	Site 16	**Inorganics**										
			BARIUM	NA	NA	NA	NA						
			MANGANESE	NA	NA	NA	NA						
			Semivolatiles										
			BIS(2-ETHYLHEXYL)PHTHALATE	2.0E-07	1.3E-08	NA	2.2E-07						
			1,4-DICHLOROBENZENE	3.2E-07	5.7E-08	NA	3.8E-07						
			Volatiles										
			1,1,2-TRICHLOROETHANE	1.7E-07	4.1E-09	6.1E-08	2.4E-07						
			1,1-DICHLOROETHENE	NA	NA	NA	NA						
			1,2-DICHLOROETHANE	2.7E-07	4.0E-09	1.1E-07	3.8E-07						
			ACETONE	NA	NA	NA	NA						
			BENZENE	1.7E-07	9.9E-09	4.5E-08	2.2E-07						
			CHLOROFORM	1.8E-08	4.6E-10	1.0E-07	1.2E-07						
			CIS-1,2-DICHLOROETHENE	NA	NA	NA	NA						
			TOTAL 1,2-DICHLOROETHENE	NA	NA	NA	NA						
			TRICHLOROETHENE	2.7E-05	1.2E-06	6.3E-06	3.4E-05						
			VINYL CHLORIDE	9.1E-06	1.9E-07	8.7E-08	9.4E-06						
			(Total for Child + Adult)	3.7E-05	1.5E-06	6.7E-06	4.5E-05		Total Hazard Index Across Groundwater (Child)				6.6E+00
			Total Risk Across Groundwater (Adult + Child)				4.5E-05		Total Hazard Index Across Groundwater (Adult)				2.9E+00
			Total Risk Across All Media and All Exposure Routes				4.7E-05		Total Hazard Index Across All Media and All Exposure Routes (Child)				6.6E+00
									Total Hazard Index Across All Media and All Exposure Routes (Adult)				2.9E+00

Table 9.2
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY EXPOSURE SCENARIO
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Recreational User
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Surface Soil	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	2.0E-08	1.4E-09	1.1E-13	2.2E-08	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ALUMINUM	--	--	--	NA	ALUMINUM	Central Nervous System	4.3E-04	0.0E+00	2.2E-06	4.4E-04
			ANTIMONY	--	--	NA	NA	ANTIMONY	Blood glucose and cholesterol	0.0E+00	0.0E+00	NA	NA
			ARSENIC	3.4E-08	2.2E-09	1.9E-12	3.6E-08	ARSENIC	Skin	5.9E-04	3.5E-04	--	9.3E-04
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	6.8E-04	0.0E+00	4.8E-06	6.9E-04
			THALLIUM	--	--	--	NA	THALLIUM	Liver, Blood	2.9E-04	0.0E+00	--	2.9E-04
			PAHs					**PAHs**					
			BENZ(A)ANTHRACENE	3.0E-09	8.6E-10	7.1E-15	3.8E-09	BENZ(A)ANTHRACENE	NA	--	--	--	NA
			BENZO[A]PYRENE	2.3E-08	6.5E-09	5.4E-14	2.9E-08	BENZO[A]PYRENE	NA	--	--	--	NA
			BENZO(B)FLUORANTHENE	4.3E-09	1.2E-09	1.0E-14	5.5E-09	BENZO(B)FLUORANTHENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	1.1E-08	3.2E-09	--	1.4E-08	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			INDENO(1,2,3-CD)PYRENE	1.9E-09	5.6E-10	4.6E-15	2.5E-09	INDENO(1,2,3-CD)PYRENE	NA	--	--	--	NA
			(Total)	9.7E-08	1.6E-08	2.1E-12	1.1E-07		(Total)	2.0E-03	3.5E-04	7.0E-06	2.3E-03
			Total Risk Across Surface Soil				**1.1E-07**		**Total Hazard Index Across Surface Soil**				**2.3E-03**
Seep Water	Seep Water	Site 16	**Inorganics**					**Inorganics**					
			ARSENIC	1.4E-10	2.8E-11	--	1.7E-10	ARSENIC	Skin	2.4E-06	4.8E-07	--	2.9E-06
			BARIUM	--	--	--	NA	BARIUM	Kidneys	6.9E-07	2.0E-06	--	2.7E-06
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	9.5E-06	4.7E-05	--	5.7E-05
			PAHs					**PAHs**					
			2-METHYLNAPHTHALENE	--	--	--	NA.	2-METHYLNAPHTHALENE	Central Nervous System, Eyes	5.6E-09	7.8E-08	--	8.3E-08
			NAPHTHALENE	--	--	--	NA	NAPHTHALENE	None	2.6E-08	3.6E-07	--	3.8E-07
			Pesticides/PCBs					**Pesticides/PCBs**					
			ALPHA-BHC	3.4E-12	1.3E-11	--	1.6E-11	ALPHA-BHC	NA	--	--	--	NA
			DIELDRIN	1.8E-11	5.7E-12	--	2.4E-11	DIELDRIN	NA	1.7E-07	5.6E-08	--	2.3E-07
			HEPTACHLOR EPOXIDE	5.9E-12	1.3E-11	--	1.9E-11	HEPTACHLOR EPOXIDE	Liver, Central Nervous System	3.9E-07	8.6E-07	--	1.2E-06
			Semivolatiles					**Semivolatiles**					
			DIBENZOFURAN	--	--	--	NA	DIBENZOFURAN	Liver, Kidney	5.3E-07	6.5E-06	--	7.0E-06
			Volatiles					**Volatiles**					
			VINYL CHLORIDE	2.7E-11	4.0E-11	--	6.7E-11	VINYL CHLORIDE	Liver	4.7E-08	6.9E-08	--	1.2E-07
			(Total)	1.9E-10	9.9E-11	--	2.9E-10		(Total)	1.4E-05	5.8E-05	--	7.1E-05
			Total Risk Across Seep Water				**2.9E-10**		**Total Hazard Index Across Seep Water**				**7.1E-05**
			Total Risk Across All Media and All Exposure Routes				**1.1E-07**		**Total Hazard Index Across All Media and All Exposure Routes**				**2.4E-03**

Table 9.3
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY EXPOSURE SCENARIO
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Recreational User
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Surface Soil	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	4.2E-08	5.9E-09	1.1E-12	4.8E-08	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ALUMINUM	--	--	--	NA	ALUMINUM	Central Nervous System	4.0E-03	0.0E+00	1.0E-04	4.1E-03
			ARSENIC	7.0E-08	9.8E-09	1.8E-11	8.0E-08	ARSENIC	Skin	5.5E-03	7.6E-04	--	6.2E-03
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	6.4E-03	0.0E+00	2.2E-04	6.6E-03
			THALLIUM	--	--	--	NA	THALLIUM	Liver, Blood	2.7E-03	0.0E+00	--	2.7E-03
			PAHs					**PAHs**					
			1,2-BENZPHENANTHRENE (Chrysene)	0.0E+00	0.0E+00	--	NA	1,2-BENZPHENANTHRENE (Chrysene)	NA	--	--	--	NA
			BENZ(A)ANTHRACENE	6.2E-09	3.7E-09	6.6E-14	9.9E-09	BENZ(A)ANTHRACENE	NA	--	--	--	NA
			BENZO[A]PYRENE	4.7E-08	2.9E-08	5.1E-13	7.6E-08	BENZO[A]PYRENE	NA	--	--	--	NA
			BENZO(B)FLUORANTHENE	8.9E-09	5.4E-09	9.6E-14	1.4E-08	BENZO(B)FLUORANTHENE	NA	--	--	--	NA
			BENZO(K)FLUORANTHENE	0.0E+00	0.0E+00	--	NA	BENZO(K)FLUORANTHENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	2.3E-08	1.4E-08	--	3.7E-08	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			INDENO(1,2,3-CD)PYRENE	4.0E-09	2.4E-09	4.3E-14	6.4E-09	INDENO(1,2,3-CD)PYRENE	NA	--	--	--	NA
			(Total)	2.0E-07	7.0E-08	2.0E-11	2.7E-07		(Total)	1.9E-02	7.6E-04	3.3E-04	2.0E-02
				Total Risk Across Surface Soil			**2.7E-07**		**Total Hazard Index Across Surface Soil**				**2.0E-02**
Seep Water	Seep Water	Site 16	**Inorganics**					**Inorganics**					
			ARSENIC	1.3E-09	1.4E-10	--	1.4E-09	ARSENIC	Skin	1.0E-04	1.1E-05	--	1.1E-04
			BARIUM	--	--	--	NA	BARIUM	Kidneys	2.9E-05	4.5E-05	--	7.4E-05
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	4.0E-04	1.1E-03	--	1.5E-03
			PAHs					**PAHs**					
			2-METHYLNAPHTHALENE	--	--	--	NA	2-METHYLNAPHTHALENE	Central Nervous System, Eyes	2.4E-07	1.8E-06	--	2.0E-06
			NAPHTHALENE	--	--	--	NA	NAPHTHALENE	None	1.1E-06	8.1E-06	--	9.1E-06
			Pesticides/PCBs					**Pesticides/PCBs**					
			ALPHA-BHC	3.2E-11	6.5E-11	--	9.7E-11	ALPHA-BHC	NA	--	--	--	NA
			DIELDRIN	1.7E-10	2.9E-11	--	2.0E-10	DIELDRIN	NA	7.3E-06	1.3E-06	--	8.6E-06
			HEPTACHLOR EPOXIDE	5.5E-11	6.6E-11	--	1.2E-10	HEPTACHLOR EPOXIDE	Liver, Central Nervous System	1.6E-05	1.9E-05	--	3.6E-05
			Semivolatiles					**Semivolatiles**					
			DIBENZOFURAN	--	--	--	NA	DIBENZOFURAN	Liver, Kidney	2.2E-05	1.5E-04	--	1.7E-04
			Volatiles					**Volatiles**					
			VINYL CHLORIDE	2.5E-10	2.0E-10	--	4.5E-10	VINYL CHLORIDE	Liver	2.0E-06	1.6E-06	--	3.5E-06
			(Total)	1.8E-09	5.0E-10	---	2.3E-09		(Total)	5.8E-04	1.3E-03	---	1.9E-03
						Total Risk Across Seep Water	**2.3E-09**		**Total Hazard Index Across Seep Water**				**1.9E-03**
			Total Risk Across All Media and All Exposure Routes				**2.7E-07**		**Total Hazard Index Across All Media and All Exposure Routes**				**2.2E-02**

Table 9.4
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY EXPOSURE SCENARIO
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Construction Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Total Soil	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	3.8E-08	3.6E-09	3.5E-12	4.2E-08	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ALUMINUM	--	--	--	NA	ALUMINUM	Central Nervous System	0.0E+00	0.0E+00	0.0E+00	NA
			ANTIMONY	--	--	--	NA	ANTIMONY	Blood glucose and cholesterol	0.0E+00	0.0E+00	--	NA
			ARSENIC	1.6E-08	1.5E-09	1.5E-11	1.8E-08	ARSENIC	Skin	2.6E-03	2.4E-04	--	2.8E-03
			BARIUM	--	--	--	NA	BARIUM	Kidneys	0.0E+00	0.0E+00	0.0E+00	NA
			COPPER	--	--	--	NA	COPPER	Gastrointestional System	0.0E+00	0.0E+00	--	NA
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	0.0E+00	0.0E+00	0.0E+00	NA
			THALLIUM	--	--	--	NA	THALLIUM	Liver, Blood	0.0E+00	0.0E+00	--	NA
			PAHs					**PAHs**					
			BENZ(A)ANTHRACENE	1.0E-09	4.3E-10	4.0E-14	1.5E-09	BENZ(A)ANTHRACENE	NA	--	--	--	NA
			BENZO[A]PYRENE	9.4E-09	3.9E-09	3.6E-13	1.3E-08	BENZO[A]PYRENE	NA	--	--	--	NA
			BENZO(B)FLUORANTHENE	1.4E-09	5.7E-10	5.4E-14	2.0E-09	BENZO(B)FLUORANTHENE	NA	--	--	--	NA
			DIBENZ[A. H]ANTHRACENE	4.9E-09	2.0E-09	--	6.9E-09	DIBENZ[A. H]ANTHRACENE	NA	--	--	--	NA
			INDENO(1,2,3-CD)PYRENE	0.0E+00	0.0E+00	0.0E+00	NA	INDENO(1,2,3-CD)PYRENE	NA	--	--	--	NA
			2-METHYLNAPHTHALENE	--	--	--	NA	2-METHYLNAPHTHALENE	Central Nervous System, Eyes	1.9E-05	7.6E-06	--	2.6E-05
			NAPHTHALENE	--	--	--	NA	NAPHTHALENE	None	0.0E+00	0.0E+00	0.0E+00	NA
			(Total)	7.1E-08	1.2E-08	1.9E-11	8.3E-08		(Total)	2.6E-03	2.5E-04	---	2.8E-03
				Total Risk Across Total Soil			8.3E-08		Total Hazard Index Across Total Soil				2.8E-03
Groundwater	Groundwater	Site 16	**Inorganics**					**Inorganics**				--	NA
			BARIUM	--	--	--	NA	BARIUM	Kidneys	4.9E-05	5.1E-04	--	5.6E-04
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	2.3E-03	4.2E-02	--	4.5E-02
			Semivolatiles					**Semivolatiles**					
			BIS(2-ETHYLHEXYL)PHTHALATE	8.4E-11	1.4E-09	--	1.5E-09	BIS(2-ETHYLHEXYL)PHTHALATE	Liver	1.9E-05	3.2E-04	--	3.4E-04
			1,4-DICHLOROBENZENE	1.3E-10	6.0E-09	--	6.2E-09	1,4-DICHLOROBENZENE	Liver	1.3E-05	5.9E-04	--	6.0E-04
			Volatiles					**Volatiles**					
			1,1,2-TRICHLOROETHANE	7.0E-11	4.3E-10	--	5.0E-10	1,1,2-TRICHLOROETHANE	Respiratory System	2.2E-05	1.3E-04	--	1.5E-04
			1,1-DICHLOROETHENE	--	--	--	NA	1,1-DICHLOROETHENE	Central Nervous System	9.3E-07	6.1E-06	--	7.0E-06
			1,2-DICHLOROETHANE	1.1E-10	4.3E-10	--	5.4E-10	1,2-DICHLOROETHANE	Central Nervous System	2.8E-06	1.1E-05	--	1.4E-05
			ACETONE	--	--	--	NA	ACETONE	Liver, Kidney	7.7E-06	3.2E-06	--	1.1E-05
			BENZENE	6.8E-11	1.1E-09	--	1.1E-09	BENZENE	Central Nervous System	2.9E-05	4.5E-04	--	4.8E-04
			CHLOROFORM	7.5E-12	4.9E-11	--	5.7E-11	CHLOROFORM	Liver, Kidneys, Central Nervous System	8.6E-06	5.6E-05	--	6.5E-05
			CIS-1,2-DICHLOROETHENE	--	--	--	NA	CIS-1,2-DICHLOROETHENE	Liver	4.8E-05	3.5E-04	--	4.0E-04
			TOTAL 1,2-DICHLOROETHENE	--	--	--	NA	TOTAL 1,2-DICHLOROETHENE	Liver	2.4E-05	1.9E-05	--	4.3E-05
			TRICHLOROETHENE	1.1E-08	1.3E-07	--	1.4E-07	TRICHLOROETHENE	Central Nervous System, Liver, Kidney	1.2E-02	1.4E-01	--	1.5E-01
			VINYL CHLORIDE	3.7E-09	2.0E-08	--	2.4E-08	VINYL CHLORIDE	Liver	5.8E-05	3.1E-04	--	3.7E-04
			(Total)	1.5E-08	1.6E-07	---	1.7E-07		(Total)	1.4E-02	1.8E-01	---	2.0E-01
				Total Risk Across Groundwater			1.7E-07		Total Hazard Index Across Groundwater				2.0E-01
			Total Risk Across All Media and All Exposure Routes				2.6E-07		Total Hazard Index Across All Media and All Exposure Routes				2.0E-01

Table 9.5
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
CENTRAL TENDENCY EXPOSURE SCENARIO
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Commercial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Surface Soil	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	8.5E-08	3.2E-09	4.3E-12	8.8E-08	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ARSENIC	1.4E-07	5.3E-09	7.2E-11	1.5E-07	ARSENIC	Skin	1.8E-03	6.8E-05	--	1.9E-03
			PAHs					**PAHs**					
			BENZ(A)ANTHRACENE	1.2E-08	2.0E-09	2.7E-13	1.4E-08	BENZ(A)ANTHRACENE	NA	--	--	--	NA
			BENZO[A]PYRENE	9.5E-08	1.5E-08	2.0E-12	1.1E-07	BENZO[A]PYRENE	NA	--	--	--	NA
			BENZO(B)FLUORANTHENE	1.8E-08	2.9E-09	3.7E-12	2.1E-08	BENZO(B)FLUORANTHENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	4.6E-08	7.4E-09	--	5.3E-08	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			(Total)	4.0E-07	3.6E-08	8.2E-11	4.3E-07		(Total)	1.8E-03	6.8E-05	---	1.9E-03
			Total Risk Across Surface Soil				**4.3E-07**		**Total Hazard Index Across Surface Soil**				**1.9E-03**
			Total Risk Across All Media and All Exposure Routes				**4.3E-07**		**Total Hazard Index Across All Media and All Exposure Routes**				**1.9E-03**

Table 9.6
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child and Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Total Soil Child	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	1.3E-05	1.5E-06	1.2E-09	1.5E-05	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ALUMINUM	--	--	--	NA	ALUMINUM	Central Nervous System	3.6E-02	0.0E+00	3.2E-03	3.9E-02
			ANTIMONY	--	--	--	NA	ANTIMONY	Blood glucose and cholesterol	1.8E-02	0.0E+00	--	1.8E-02
			ARSENIC	6.1E-06	6.6E-07	5.4E-09	6.7E-06	ARSENIC	Skin	1.6E-01	1.7E-02	--	1.7E-01
			BARJUM	--	--	--	NA	BARJUM	Kidneys	6.7E-03	0.0E+00	2.9E-04	6.9E-03
			COPPER	--	--	--	NA	COPPER	Gastrointestional System	7.6E-03	0.0E+00	--	7.6E-03
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	5.5E-02	0.0E+00	6.8E-03	6.2E-02
			THALLIUM	--	--	--	NA	THALLIUM	Liver, Blood	2.6E-02	0.0E+00	--	2.6E-02
			PAHs					**PAHs**					
			BENZ(A)ANTHRACENE	1.5E-07	6.9E-08	5.5E-12	2.1E-07	BENZ(A)ANTHRACENE	NA	--	--	--	NA
			BENZO[A]PYRENE	1.4E-06	6.7E-07	5.4E-11	2.1E-06	BENZO[A]PYRENE	NA	--	--	--	NA
			BENZO(B)FLUORANTHENE	1.8E-07	8.3E-08	6.7E-12	2.6E-07	BENZO(B)FLUORANTHENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	1.1E-06	5.0E-07	--	1.6E-06	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			INDENO(1,2,3-CD)PYRENE	1.2E-07	5.6E-08	4.5E-12	1.8E-07	INDENO(1,2,3-CD)PYRENE	NA	--	--	--	NA
			2-METHYLNAPHTHALENE	--	--	--	NA	2-METHYLNAPHTHALENE	Central Nervous System, Eyes	2.1E-04	9.9E-05	--	3.1E-04
			NAPHTHALENE	--	--	--	NA	NAPHTHALENE	None	1.3E-04	6.3E-05	2.6E-07	2.0E-04
			(Total for Child)	2.2E-05	3.5E-06	6.7E-09	2.6E-05		(Total for Child)	3.1E-01	1.7E-02	1.0E-02	3.4E-01
	Total Soil Adult	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	5.8E-06	9.7E-07	6.6E-10	6.7E-06	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					NA
			ALUMINUM	--	--	--	NA	ALUMINUM	Central Nervous System	3.9E-03	0.0E+00	4.4E-04	4.3E-03
			ANTIMONY	--	--	--	NA	ANTIMONY	Blood glucose and cholesterol	1.9E-03	0.0E+00	--	1.9E-03
			ARSENIC	2.6E-06	4.4E-07	3.0E-09	3.0E-06	ARSENIC	Skin	1.7E-02	2.8E-03	--	2.0E-02
			BARJUM	--	--	--	NA	BARJUM	Kidneys	7.1E-04	0.0E+00	4.1E-05	7.5E-04
			COPPER	--	--	--	NA	COPPER	Gastrointestional System	8.2E-04	0.0E+00	--	8.2E-04
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	5.9E-03	0.0E+00	9.3E-04	6.8E-03
			THALLIUM	--	--	--	NA	THALLIUM	Liver, Blood	2.8E-03	0.0E+00	--	2.8E-03
			PAHs					**PAHs**					
			BENZ(A)ANTHRACENE	6.3E-08	4.6E-08	3.0E-12	1.1E-07	BENZ(A)ANTHRACENE	NA	--	--	--	NA
			BENZO[A]PYRENE	6.1E-07	4.5E-07	3.0E-11	1.1E-06	BENZO[A]PYRENE	NA	--	--	--	NA
			BENZO(B)FLUORANTHENE	7.6E-08	5.5E-08	3.7E-12	1.3E-07	BENZO(B)FLUORANTHENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	4.6E-07	3.3E-07	--	7.9E-07	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			INDENO(1,2,3-CD)PYRENE	5.2E-08	3.8E-08	2.5E-12	8.9E-08	INDENO(1,2,3-CD)PYRENE	NA	--	--	--	NA
			2-METHYLNAPHTHALENE	--	--	--	NA	2-METHYLNAPHTHALENE	Central Nervous System, Eyes	2.3E-05	1.6E-05	--	3.9E-05
			NAPHTHALENE	--	--	--	NA	NAPHTHALENE	None	1.4E-05	1.0E-05	3.6E-08	2.5E-05
			(Total for Adult)	9.6E-06	2.3E-06	3.7E-09	1.2E-05		(Total for Adult)	3.3E-02	2.9E-03	1.4E-03	3.7E-02
	Total Soil Adult + Child	Site 16	**Dioxin/Furans**										
			TCDD-TEQ	1.9E-05	2.4E-06	1.8E-09	2.2E-05						
			Inorganics										
			ALUMINUM	NA	NA	NA	NA						
			ANTIMONY	NA	NA	NA	NA						
			ARSENIC	8.7E-06	1.1E-06	8.4E-09	9.8E-06						
			BARJUM	NA	NA	NA	NA						
			COPPER	NA	NA	NA	NA						
			MANGANESE	NA	NA	NA	NA						
			THALLIUM	NA	NA	NA	NA						
			PAHs										
			BENZ(A)ANTHRACENE	2.1E-07	1.1E-07	8.5E-12	3.2E-07						
			BENZO[A]PYRENE	2.0E-06	1.1E-06	8.3E-11	3.2E-06						
			BENZO(B)FLUORANTHENE	2.5E-07	1.4E-07	1.0E-11	3.9E-07						
			DIBENZ[A, H]ANTHRACENE	1.5E-06	8.3E-07	NA	2.4E-06						
			INDENO(1,2,3-CD)PYRENE	1.7E-07	9.4E-08	7.0E-12	2.7E-07						
			2-METHYLNAPHTHALENE	NA	NA	NA	NA						
			NAPHTHALENE	NA	NA	NA	NA						
			(Total for Child + Adult)	3.2E-05	5.8E-06	1.0E-08	3.8E-05		Total Hazard Index Across Total Soil (Child)				3.4E-01
				Total Risk Across Total Soil			3.8E-05		Total Hazard Index Across Total Soil (Adult)				3.7E-02

Table 9.6
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child and Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Groundwater	Groundwater Child	Site 16	Inorganics					Inorganics					
			BARIUM	--	--	--	NA	BARIUM	Kidneys	5.2E-02	5.2E-03	--	5.7E-02
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	5.6E+00	9.8E-01	--	6.6E+00
			Semivolatiles			--		Semivolatiles				--	
			BIS(2-ETHYLHEXYL)PHTHALATE	6.4E-07	1.0E-07	--	7.5E-07	BIS(2-ETHYLHEXYL)PHTHALATE	Liver	2.4E-02	3.9E-03	--	2.8E-02
			1,4-DICHLOROBENZENE	1.0E-06	4.4E-07	--	1.5E-06	1,4-DICHLOROBENZENE	Liver	1.6E-02	7.1E-03	--	2.4E-02
			Volatiles			--		Volatiles				--	
			1,1,2-TRICHLOROETHANE	3.8E-07	2.2E-08	--	4.0E-07	1,1,2-TRICHLOROETHANE	Respiratory System	2.0E-02	1.1E-03	--	2.1E-02
			1,1-DICHLOROETHENE	--	--	--	NA	1,1-DICHLOROETHENE	Central Nervous System	9.1E-04	5.7E-05	--	9.7E-04
			1,2-DICHLOROETHANE	5.9E-07	2.2E-08	--	6.1E-07	1,2-DICHLOROETHANE	Central Nervous System	2.5E-03	9.3E-05	--	2.6E-03
			ACETONE	--	--	--	NA	ACETONE	Liver, Kidney	7.8E-03	3.1E-05	--	7.8E-03
			BENZENE	3.7E-07	5.5E-08	--	4.3E-07	BENZENE	Central Nervous System	2.6E-02	3.9E-03	--	3.0E-02
			CHLOROFORM	4.0E-08	2.5E-09	--	4.3E-08	CHLOROFORM	Liver, Kidneys, Central Nervous System	7.7E-03	4.8E-04	--	8.2E-03
			CIS-1,2-DICHLOROETHENE	--	--	--	NA	CIS-1,2-DICHLOROETHENE	Liver	5.4E-02	3.8E-03	--	5.7E-02
			TOTAL 1,2-DICHLOROETHENE	--	--	--	NA	TOTAL 1,2-DICHLOROETHENE	Liver	3.3E-02	2.5E-04	--	3.3E-02
			TRICHLOROETHENE	5.7E-04	6.4E-05	--	6.3E-04	TRICHLOROETHENE	Central Nervous System, Liver, Kidney	1.0E+02	1.1E+01	--	1.1E+02
			VINYL CHLORIDE	2.0E-05	1.0E-06	--	2.1E-05	VINYL CHLORIDE	Liver	5.2E-02	2.7E-03	--	5.5E-02
			(Total for Child)	5.9E-04	6.5E-05	---	6.6E-04		(Total for Child)	1.1E+02	1.2E+01	---	1.2E+02
	Groundwater Adult	Site 16	Inorganics					Inorganics					
			BARIUM	--	--	--	NA	BARIUM	Kidneys	1.7E-02	1.36E-03	--	1.8E-02
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	1.8E+00	2.57E-01	--	2.1E+00
			Semivolatiles				NA	Semivolatiles					
			BIS(2-ETHYLHEXYL)PHTHALATE	8.4E-07	1.08E-07	--	9.5E-07	BIS(2-ETHYLHEXYL)PHTHALATE	Liver	8.0E-03	1.03E-03	--	9.0E-03
			1,4-DICHLOROBENZENE	1.3E-06	4.62E-07	--	1.8E-06	1,4-DICHLOROBENZENE	Liver	5.4E-03	1.87E-03	--	7.3E-03
			Volatiles					Volatiles					
			1,1,2-TRICHLOROETHANE	5.0E-07	2.35E-08	5.0E-07	1.0E-06	1,1,2-TRICHLOROETHANE	Respiratory System	6.4E-03	3.01E-04	--	6.7E-03
			1,1-DICHLOROETHENE	--	--	--	NA	1,1-DICHLOROETHENE	Central Nervous System	3.0E-04	1.49E-05	--	3.2E-04
			1,2-DICHLOROETHANE	7.7E-07	2.28E-08	8.9E-07	1.7E-06	1,2-DICHLOROETHANE	Central Nervous System	8.3E-04	2.44E-05	5.4E-02	5.5E-02
			ACETONE	--	--	--	NA	ACETONE	Liver, Kidney	2.5E-03	8.11E-06	--	2.6E-03
			BENZENE	4.9E-07	5.72E-08	3.7E-07	9.2E-07	BENZENE	Central Nervous System	8.6E-03	1.01E-03	5.8E-02	6.8E-02
			CHLOROFORM	5.3E-08	2.62E-09	8.4E-07	9.0E-07	CHLOROFORM	Liver, Kidneys, Central Nervous System	2.5E-03	1.25E-04	9.5E-01	9.5E-01
			CIS-1,2-DICHLOROETHENE	--	--	--	NA	CIS-1,2-DICHLOROETHENE	Liver	1.8E-02	9.83E-04	--	1.9E-02
			TOTAL 1,2-DICHLOROETHENE	--	--	--	NA	TOTAL 1,2-DICHLOROETHENE	Liver	1.1E-02	6.64E-05	--	1.1E-02
			TRICHLOROETHENE	7.5E-04	6.68E-05	4.9E-04	1.3E-03	TRICHLOROETHENE	Central Nervous System, Liver, Kidney	3.3E+01	2.95E+00	--	3.6E+01
			VINYL CHLORIDE	2.7E-05	1.08E-06	7.2E-07	2.8E-05	VINYL CHLORIDE	Liver	1.7E-02	7.02E-04	6.2E-03	2.4E-02
			(Total for Adult)	7.8E-04	6.9E-05	5.0E-04	1.3E-03		(Total for Adult)	3.5E+01	3.2E+00	1.1E+00	3.9E+01
	Groundwater Adult + Child	Site 16	Inorganics										
			BARIUM	NA	NA	NA	NA						
			MANGANESE	NA	NA	NA	NA						
			Semivolatiles										
			BIS(2-ETHYLHEXYL)PHTHALATE	1.5E-06	2.1E-07	NA	1.7E-06						
			1,4-DICHLOROBENZENE	2.4E-06	9.0E-07	NA	3.3E-06						
			Volatiles										
			1,1,2-TRICHLOROETHANE	8.8E-07	4.6E-08	5.0E-07	1.4E-06						
			1,1-DICHLOROETHENE	NA	NA	NA	NA						
			1,2-DICHLOROETHANE	1.4E-06	4.5E-08	8.9E-07	2.3E-06						
			ACETONE	NA	NA	NA	NA						
			BENZENE	8.6E-07	1.1E-07	3.7E-07	1.3E-06						
			CHLOROFORM	9.3E-08	5.1E-09	8.4E-07	9.4E-07						
			CIS-1,2-DICHLOROETHENE	NA	NA	NA	NA						
			TOTAL 1,2-DICHLOROETHENE	NA	NA	NA	NA						
			TRICHLOROETHENE	1.3E-03	1.3E-04	4.9E-04	1.9E-03						
			VINYL CHLORIDE	4.7E-05	2.1E-06	7.2E-07	5.0E-05						
			(Total for Child + Adult)	1.4E-03	1.3E-04	5.0E-04	2.0E-03		Total Hazard Index Across Groundwater (Child)				1.2E+02
			Total Risk Across Groundwater (Adult + Child)				2.0E-03		Total Hazard Index Across Groundwater (Adult)				3.9E+01
			Total Risk Across All Media and All Exposure Routes				2.0E-03		Total Hazard Index Across All Media and All Exposure Routes (Child)				1.2E+02
									Total Hazard Index Across All Media and All Exposure Routes (Adult)				3.9E+01

Table 9.7
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Recreational User
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Surface Soil	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	7.3E-07	5.6E-07	3.7E-11	1.3E-06	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ALUMINUM	--	--	--	NA	ALUMINUM	Central Nervous System	2.2E-03	0.0E+00	2.7E-04	2.5E-03
			ARSENIC	5.2E-07	4.0E-07	2.6E-10	9.1E-07	ARSENIC	Skin	3.3E-03	2.6E-03	--	5.9E-03
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	3.5E-03	0.0E+00	5.9E-04	4.1E-03
			THALLIUM	--	--	--	NA	THALLIUM	Liver, Blood	1.6E-03	0.0E+00	--	1.6E-03
			PAHs					**PAHs**					
			BENZ(A)ANTHRACENE	5.3E-08	1.8E-07	1.1E-12	2.3E-07	BENZ(A)ANTHRACENE	NA	--	--	--	NA
			BENZO[A]PYRENE	4.4E-07	1.5E-06	9.4E-12	1.9E-06	BENZO[A]PYRENE	NA	--	--	--	NA
			BENZO(B)FLUORANTHENE	6.4E-08	2.1E-07	1.4E-12	2.8E-07	BENZO(B)FLUORANTHENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	3.6E-07	1.2E-06	--	1.6E-06	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			INDENO(1,2,3-CD)PYRENE	3.9E-08	1.3E-07	8.4E-13	1.7E-07	INDENO(1,2,3-CD)PYRENE	NA	--	--	--	NA
			(Total)	2.2E-06	4.1E-06	3.1E-10	6.3E-06		(Total)	1.1E-02	2.6E-03	8.6E-04	1.4E-02
			Total Risk Across Surface Soil				6.3E-06		Total Hazard Index Across Surface Soil				1.4E-02
Seep Water	Seep Water	Site 16	**Inorganics**					**Inorganics**					
			ARSENIC	2.4E-08	3.2E-09	--	2.7E-08	ARSENIC	Skin	1.6E-04	2.1E-05	--	1.8E-04
			BARIUM	--	--	--	NA	BARIUM	Kidneys	7.2E-05	1.4E-04	--	2.1E-04
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	1.5E-03	5.0E-03	--	6.5E-03
			PAHs					**PAHs**					
			2-METHYLNAPHTHALENE	--	--	--	NA	2-METHYLNAPHTHALENE	Central Nervous System, Eyes	6.5E-07	6.0E-06	--	6.7E-06
			NAPHTHALENE	--	--	--	NA	NAPHTHALENE	None	2.7E-06	2.5E-05	--	2.8E-05
			Pesticides/PCBs					**Pesticides/PCBs**					
			ALPHA-BHC	9.6E-10	2.4E-09	--	3.4E-09	ALPHA-BHC	NA	--	--	--	NA
			DIELDRIN	3.2E-09	6.8E-10	--	3.9E-09	DIELDRIN	NA	1.2E-05	2.5E-06	--	1.4E-05
			HEPTACHLOR EPOXIDE	8.5E-10	1.3E-09	--	2.1E-09	HEPTACHLOR EPOXIDE	Liver, Central Nervous System	2.1E-05	3.1E-05	--	5.2E-05
			Semivolatiles					**Semivolatiles**					
			DIBENZOFURAN	--	--	--	NA	DIBENZOFURAN	Liver, Kidney	3.9E-05	3.2E-04	--	3.6E-04
			Volatiles					**Volatiles**					
			VINYL CHLORIDE	4.7E-09	4.6E-09	--	9.3E-09	VINYL CHLORIDE	Liver	3.1E-06	3.0E-06	--	6.0E-06
			(Total)	3.4E-08	1.2E-08	---	4.6E-08		(Total)	1.8E-03	5.5E-03	---	7.3E-03
			Total Risk Across Seep Water				4.6E-08		Total Hazard Index Across Seep Water				7.3E-03
			Total Risk Across All Media and All Exposure Routes				6.4E-06		Total Hazard Index Across All Media and All Exposure Routes				2.1E-02

Table 9.8
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Recreational User
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Surface Soil	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	1.7E-06	3.0E-07	5.4E-11	2.0E-06	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ALUMINUM	--	--	--	NA	ALUMINUM	Central Nervous System	2.1E-02	0.0E+00	6.6E-04	2.2E-02
			ARSENIC	1.2E-06	2.1E-07	3.8E-10	1.4E-06	ARSENIC	Skin	3.1E-02	5.4E-03	--	3.7E-02
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	3.2E-02	0.0E+00	1.4E-03	3.4E-02
			THALLIUM	--	--	--	NA	THALLIUM	Liver, Blood	1.5E-02	0.0E+00	--	1.5E-02
			PAHs					**PAHs**					
			BENZ(A)ANTHRACENE	1.2E-07	9.2E-08	1.6E-12	2.2E-07	BENZ(A)ANTHRACENE	NA	--	--	--	NA
			BENZO[A]PYRENE	1.0E-06	7.7E-07	1.4E-11	1.8E-06	BENZO[A]PYRENE	NA	--	--	--	NA
			BENZO(B)FLUORANTHENE	1.5E-07	1.1E-07	2.0E-12	2.6E-07	BENZO(B)FLUORANTHENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	8.4E-07	6.3E-07	--	1.5E-06	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			INDENO(1,2,3-CD)PYRENE	9.2E-08	6.9E-08	1.2E-12	1.6E-07	INDENO(1,2,3-CD)PYRENE	NA	--	--	--	NA
			(Total)	5.1E-06	2.2E-06	4.5E-10	7.3E-06		(Total)	1.0E-01	5.4E-03	2.1E-03	1.1E-01
				Total Risk Across Surface Soil			7.3E-06		Total Hazard Index Across Surface Soil				1.1E-01
Seep Water	Seep Water	Site 16	**Inorganics**					**Inorganics**					
			ARSENIC	2.8E-08	1.9E-09	--	3.0E-08	ARSENIC	Skin	7.3E-04	4.8E-05	--	7.8E-04
			BARIUM	--	--	--	NA	BARIUM	Kidneys	3.4E-04	3.2E-04	--	6.6E-04
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	6.9E-03	1.1E-02	--	1.8E-02
			PAHs					**PAHs**					
			2-METHYLNAPHTHALENE	--	--	--	NA	2-METHYLNAPHTHALENE	None	3.0E-06	1.4E-05	--	1.7E-05
			NAPHTHALENE	--	--	--	NA	NAPHTHALENE	None	1.3E-05	5.8E-05	--	7.1E-05
			Pesticides/PCBs					**Pesticides/PCBs**					
			ALPHA-BHC	1.1E-09	1.4E-09	--	2.5E-09	ALPHA-BHC	Immune System and Growth	--	--	--	NA
			DIELDRIN	3.7E-09	3.9E-10	--	4.1E-09	DIELDRIN	NA	5.4E-05	5.7E-06	--	6.0E-05
			HEPTACHLOR EPOXIDE	1.0E-09	7.2E-10	--	1.7E-09	HEPTACHLOR EPOXIDE	Liver, Central Nervous System	9.8E-05	7.1E-05	--	1.7E-04
			Semivolatiles					**Semivolatiles**					
			DIBENZOFURAN	--	--	--	NA	DIBENZOFURAN	Liver, Kidney	1.8E-04	7.4E-04	--	9.2E-04
			Volatiles					**Volatiles**					
			VINYL CHLORIDE	5.5E-09	2.6E-09	--	8.1E-09	VINYL CHLORIDE	Liver	1.4E-05	6.9E-06	--	2.1E-05
			(Total)	3.9E-08	7.0E-09	---	4.6E-08		(Total)	8.4E-03	1.3E-02	---	2.1E-02
						Total Risk Across Seep Water	4.6E-08		Total Hazard Index Across Seep Water				2.1E-02
			Total Risk Across All Media and All Exposure Routes				7.4E-06		Total Hazard Index Across All Media and All Exposure Routes				1.3E-01

Table 9.9
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Construction Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Total Soil	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	1.2E-06	8.8E-08	3.9E-11	1.3E-06	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ARSENIC	5.6E-07	4.0E-08	1.8E-10	6.0E-07	ARSENIC	Skin	8.7E-02	6.2E-03	--	9.3E-02
			PAHs					**PAHs**					
			BENZ(A)ANTHRACENE	1.3E-08	4.1E-09	1.8E-13	1.8E-08	BENZ(A)ANTHRACENE	NA	--	--	--	NA
			BENZO[A]PYRENE	1.3E-07	4.0E-08	1.8E-12	1.7E-07	BENZO[A]PYRENE	NA	--	--	--	NA
			BENZO(B)FLUORANTHENE	1.6E-08	5.0E-09	2.2E-13	2.1E-08	BENZO(B)FLUORANTHENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	9.7E-08	3.0E-08	--	1.3E-07	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			2-METHYLNAPHTHALENE	--	--	--	NA	2-METHYLNAPHTHALENE	Central Nervous System, Eyes	1.2E-04	3.6E-05	--	1.5E-04
			(Total)	2.0E-06	2.1E-07	2.2E-10	2.3E-06		(Total)	8.7E-02	6.2E-03	---	9.3E-02
			Total Risk Across Total Soil				**2.3E-06**		**Total Hazard Index Across Total Soil**				**9.3E-02**
Groundwater	Groundwater	Site 16	**Inorganics**					**Inorganics**				--	NA
			BARIUM	--	--	--	NA	BARIUM	Kidneys	1.7E-04	2.2E-03	--	2.4E-03
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	1.8E-02	4.2E-01	--	4.4E-01
			Semivolatiles					**Semivolatiles**					
			BIS(2-ETHYLHEXYL)PHTHALATE	3.5E-10	7.3E-09	--	7.7E-09	BIS(2-ETHYLHEXYL)PHTHALATE	Liver	7.9E-05	1.7E-03	--	1.7E-03
			1,4-DICHLOROBENZENE	5.5E-10	3.1E-08	--	3.2E-08	1,4-DICHLOROBENZENE	Liver	5.4E-05	3.0E-03	--	3.1E-03
			Volatiles					**Volatiles**					
			1,1,2-TRICHLOROETHANE	2.1E-10	1.6E-09	--	1.8E-09	1,1,2-TRICHLOROETHANE	Respiratory System	6.4E-05	4.9E-04	--	5.5E-04
			1,1-DICHLOROETHENE	--	--	--	NA	1,1-DICHLOROETHENE	Central Nervous System	3.0E-06	2.4E-05	--	2.7E-05
			1,2-DICHLOROETHANE	3.2E-10	1.5E-09	--	1.9E-09	1,2-DICHLOROETHANE	Central Nervous System	8.2E-06	4.0E-05	--	4.8E-05
			ACETONE	--	--	--	NA	ACETONE	Liver, Kidney	2.5E-05	1.3E-05	--	3.8E-05
			BENZENE	2.0E-10	3.9E-09	--	4.1E-09	BENZENE	Central Nervous System	8.6E-05	1.6E-03	--	1.7E-03
			CHLOROFORM	2.2E-11	1.8E-10	--	2.0E-10	CHLOROFORM	Liver, Kidneys, Central Nervous System	2.5E-05	2.0E-04	--	2.3E-04
			CIS-1,2-DICHLOROETHENE	--	--	--	NA	CIS-1,2-DICHLOROETHENE	Liver	1.8E-04	1.6E-03	--	1.8E-03
			TOTAL 1,2-DICHLOROETHENE	--	--	--	NA	TOTAL 1,2-DICHLOROETHENE	Liver	1.1E-04	1.1E-04	--	2.2E-04
			TRICHLOROETHENE	3.1E-07	4.5E-06	--	4.8E-06	TRICHLOROETHENE	Central Nervous System, Liver, Kidney	3.3E-01	4.8E+00	--	5.1E+00
			VINYL CHLORIDE	1.1E-08	7.3E-08	--	8.4E-08	VINYL CHLORIDE	Liver	1.7E-04	1.1E-03	--	1.3E-03
			(Total)	3.2E-07	4.6E-06	---	5.0E-06		(Total)	3.5E-01	5.2E+00	---	5.6E+00
			Total Risk Across Groundwater				**5.0E-06**		**Total Hazard Index Across Groundwater**				**5.6E+00**
			Total Risk Across All Media and All Exposure Routes				**7.2E-06**		**Total Hazard Index Across All Media and All Exposure Routes**				**5.7E+00**

Table 9.10
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
REASONABLE MAXIMUM EXPOSURE
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Commercial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Surface Soil	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	9.9E-07	4.5E-07	1.0E-10	1.4E-06	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ARSENIC	7.0E-07	3.2E-07	7.1E-10	1.0E-06	ARSENIC	Skin	4.4E-03	2.0E-03	--	6.3E-03
			PAHs					**PAHs**					
			BENZ(A)ANTHRACENE	7.2E-08	1.4E-07	3.1E-12	2.1E-07	BENZ(A)ANTHRACENE	NA	--	--	--	NA
			BENZO[A]PYRENE	6.0E-07	1.2E-06	2.6E-11	1.8E-06	BENZO[A]PYRENE	NA	--	--	--	NA
			BENZO(B)FLUORANTHENE	8.6E-08	1.7E-07	3.7E-12	2.6E-07	BENZO(B)FLUORANTHENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	4.9E-07	9.6E-07	--	1.4E-06	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			(Total)	2.9E-06	3.2E-06	8.4E-10	6.2E-06		(Total)	4.4E-03	2.0E-03	---	6.3E-03
			Total Risk Across Surface Soil				6.2E-06		Total Hazard Index Across Surface Soil				6.3E-03
			Total Risk Across All Media and All Exposure Routes				6.2E-06		Total Hazard Index Across All Media and All Exposure Routes				6.3E-03

Table 10.1
RISK ASSESSMENT SUMMARY
REASONABLE MAXIMUM EXPOSURE
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child and Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Total Soil Child	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	1.3E-05	1.5E-06	1.2E-09	1.5E-05	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ARSENIC	6.1E-06	6.6E-07	5.4E-09	6.7E-06	ARSENIC	Skin	1.6E-01	1.7E-02	--	1.7E-01
			PAHs					**PAHs**					
			BENZO[A]PYRENE	1.4E-06	6.7E-07	5.4E-11	2.1E-06	BENZO[A]PYRENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	1.1E-06	5.0E-07	--	1.6E-06	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			(Total for Child)	2.2E-05	3.3E-06	6.7E-09	2.5E-05		(Total for Child)	1.6E-01	1.7E-02	---	1.7E-01
	Total Soil Adult	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	5.8E-06	9.7E-07	6.6E-10	6.7E-06	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					NA
			ARSENIC	2.6E-06	4.4E-07	3.0E-09	3.0E-06	ARSENIC	Skin	--	--	--	NA
			PAHs					**PAHs**					
			BENZO[A]PYRENE	6.1E-07	4.5E-07	3.0E-11	1.1E-06	BENZO[A]PYRENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	4.6E-07	3.3E-07	--	7.9E-07	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			(Total for Adult)	9.4E-06	2.2E-06	3.7E-09	1.2E-05		(Total for Adult)	---	---	---	---
	Total Soil Adult + Child	Site 16	**Dioxin/Furans**										
			TCDD-TEQ	1.9E-05	2.4E-06	1.8E-09	2.2E-05						
			Inorganics										
			ARSENIC	8.7E-06	1.1E-06	8.4E-09	9.8E-06						
			PAHs										
			BENZO[A]PYRENE	2.0E-06	1.1E-06	8.3E-11	3.2E-06						
			DIBENZ[A, H]ANTHRACENE	1.5E-06	8.3E-07	NA	2.4E-06						
			(Total for Child + Adult)	3.1E-05	5.5E-06	1.0E-08	3.7E-05		Total Hazard Index Across Total Soil (Child)				1.7E-01
				Total Risk Across Total Soil			3.7E-05		Total Hazard Index Across Total Soil (Adult)				---
Groundwater	Groundwater Child	Site 16	**Inorganics**					**Inorganics**					
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	5.6E+00	9.8E-01	--	6.6E+00
			Semivolatiles			--		**Semivolatiles**				--	
			BIS(2-ETHYLHEXYL)PHTHALATE	6.4E-07	1.0E-07	--	7.5E-07	BIS(2-ETHYLHEXYL)PHTHALATE	Liver	--	--	--	NA
			1,4-DICHLOROBENZENE	1.0E-06	4.4E-07	--	1.5E-06	1,4-DICHLOROBENZENE	Liver	--	--	--	NA
			Volatiles			--		**Volatiles**				--	
			TRICHLOROETHENE	5.7E-04	6.4E-05	--	6.3E-04	TRICHLOROETHENE	Central Nervous System, Liver, Kidney	1.0E+02	1.1E+01	--	1.1E+02
			VINYL CHLORIDE	2.0E-05	1.0E-06	--	2.1E-05	VINYL CHLORIDE	Liver	--	--	--	NA
			(Total for Child)	5.9E-04	6.5E-05	---	6.6E-04		(Total for Child)	1.1E+02	1.2E+01	---	1.2E+02

Table 10.1
RISK ASSESSMENT SUMMARY
REASONABLE MAXIMUM EXPOSURE
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Resident
Receptor Age: Child and Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Groundwater	Groundwater Adult	Site 16	**Inorganics**					**Inorganics**					
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	1.8E+00	2.57E-01	--	NA 2.1E+00
			Semivolatiles					**Semivolatiles**					
			BIS(2-ETHYLHEXYL)PHTHALATE	8.4E-07	1.1E-07	--	9.5E-07	BIS(2-ETHYLHEXYL)PHTHALATE	Liver	--	--	--	NA
			1,4-DICHLOROBENZENE	1.3E-06	4.6E-07	--	1.8E-06	1,4-DICHLOROBENZENE	Liver	--	--	--	NA
			Volatiles					**Volatiles**					
			1,1,2-TRICHLOROETHANE	5.0E-07	2.3E-08	5.0E-07	1.0E-06	1,1,2-TRICHLOROETHANE	Respiratory System	--	--	--	NA
			1,2-DICHLOROETHANE	7.7E-07	2.3E-08	8.9E-07	1.7E-06	1,2-DICHLOROETHANE	Central Nervous System	--	--	--	NA
			CHLOROFORM	--	--	--	NA	CHLOROFORM	Liver, Kidneys, Central Nervous System	2.5E-03	1.25E-04	9.5E-01	9.5E-01
			TRICHLOROETHENE	7.5E-04	6.7E-05	4.9E-04	1.3E-03	TRICHLOROETHENE	Central Nervous System, Liver, Kidney	3.3E+01	2.95E+00	--	3.6E+01
			VINYL CHLORIDE	2.7E-05	1.1E-06	7.2E-07	2.8E-05	VINYL CHLORIDE	Liver	--	--	--	NA
			(Total for Adult)	7.8E-04	6.9E-05	5.0E-04	1.3E-03		(Total for Adult)	3.5E+01	3.2E+00	9.5E-01	3.9E+01
	Groundwater Adult + Child	Site 16	**Inorganics**										
			MANGANESE	NA	NA	NA	NA						
			Semivolatiles										
			BIS(2-ETHYLHEXYL)PHTHALATE	1.5E-06	2.1E-07	NA	1.7E-06						
			1,4-DICHLOROBENZENE	2.4E-06	9.0E-07	NA	3.3E-06						
			Volatiles										
			1,1,2-TRICHLOROETHANE	5.0E-07	2.3E-08	5.0E-07	1.0E-06						
			1,2-DICHLOROETHANE	7.7E-07	2.3E-08	8.9E-07	1.7E-06						
			TRICHLOROETHENE	1.3E-03	1.3E-04	4.9E-04	1.9E-03						
			VINYL CHLORIDE	4.7E-05	2.1E-06	7.2E-07	5.0E-05						
			(Total for Child + Adult)	1.4E-03	1.3E-04	5.0E-04	2.0E-03		Total Hazard Index Across Groundwater (Child)				1.2E+02
			Total Risk Across Groundwater (Adult + Child)				2.0E-03		Total Hazard Index Across Groundwater (Adult)				3.9E+01
			Total Risk Across All Media and All Exposure Routes				2.0E-03		Total Hazard Index Across All Media and All Exposure Routes (Child)				1.2E+02
									Total Hazard Index Across All Media and All Exposure Routes (Adult)				3.9E+01

Target Organ Breakdown for Skin (Child)	1.7E-01
Target Organ Breakdown for Central Nervous System (Child)	1.2E+02
Target Organ Breakdown for Liver (Child)	1.1E+02
Target Organ Breakdown for Kidney (Child)	1.1E+02
Target Organ Breakdown for Skin (Adult)	NA
Target Organ Breakdown for Central Nervous System (Adult)	3.9E+01
Target Organ Breakdown for Liver (Adult)	3.7E+01
Target Organ Breakdown for Kidney (Adult)	3.7E+01

Table 10.2
RISK ASSESSMENT SUMMARY
REASONABLE MAXIMUM EXPOSURE
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Recreational User
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Surface Soil	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	7.3E-07	5.6E-07	3.7E-11	1.3E-06	TCDD-TEQ	NA	--	--	--	NA
			PAHs					**PAHs**					
			BENZO[A]PYRENE	4.4E-07	1.5E-06	9.4E-12	1.9E-06	BENZO[A]PYRENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	3.6E-07	1.2E-06	--	1.6E-06	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			(Total)	1.5E-06	3.2E-06	4.6E-11	4.8E-06		(Total)	---	---	---	---
			Total Risk Across Surface Soil				**4.8E-06**		**Total Hazard Index Across Surface Soil**				---
			Total Risk Across Seep Water				---		**Total Hazard Index Across Seep Water**				---
			Total Risk Across All Media and All Exposure Routes				4.8E-06		Total Hazard Index Across All Media and All Exposure Routes				---

Table 10.3
SUMMARY OF RECEPTOR RISKS AND HAZARDS FOR COPCs
RISK ASSESSMENT SUMMARY
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Recreational User
Receptor Age: Child

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Surface Soil	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	1.7E-06	3.0E-07	5.4E-11	2.0E-06	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ARSENIC	1.2E-06	2.1E-07	3.8E-10	1.4E-06	ARSENIC	Skin	--	--	--	NA
			PAHs					**PAHs**					
			BENZO[A]PYRENE	1.0E-06	7.7E-07	1.4E-11	1.8E-06	BENZO[A]PYRENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	8.4E-07	6.3E-07	--	1.5E-06	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			(Total)	4.8E-06	1.9E-06	4.5E-10	6.7E-06	(Total)		---	---	---	---
				Total Risk Across Surface Soil			6.7E-06		Total Hazard Index Across Surface Soil				---
				Total Risk Across Seep Water			---		Total Hazard Index Across Seep Water				---
			Total Risk Across All Media and All Exposure Routes				6.7E-06		Total Hazard Index Across All Media and All Exposure Routes				---

Table 10.4
RISK ASSESSMENT SUMMARY
REASONABLE MAXIMUM EXPOSURE
DAVISVILLE SITE 16

Location: Site 16

Scenario Timeframe: Future
Receptor Population: Construction Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Total Soil	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	1.2E-06	8.8E-08	3.9E-11	1.3E-06	TCDD-TEQ	NA	--	--	--	NA
			(Total)	1.2E-06	8.8E-08	3.9E-11	1.3E-06		(Total)	---	---	---	---
			Total Risk Across Total Soil				**1.3E-06**		**Total Hazard Index Across Total Soil**				**---**
Groundwater	Groundwater	Site 16	**Inorganics**					**Inorganics**					
			MANGANESE	--	--	--	NA	MANGANESE	Central Nervous System	1.8E-02	4.2E-01	--	4.4E-01
			Volatiles					**Volatiles**					
			TRICHLOROETHENE	3.1E-07	4.5E-06	--	4.8E-06	TRICHLOROETHENE	Central Nervous System, Liver, Kidney	3.3E-01	4.8E+00	--	5.1E+00
			(Total)	3.1E-07	4.5E-06	---	4.8E-06		(Total)	3.5E-01	5.2E+00	---	5.6E+00
			Total Risk Across Groundwater				**4.8E-06**		**Total Hazard Index Across Groundwater**				**5.6E+00**
			Total Risk Across All Media and All Exposure Routes				**6.2E-06**		**Total Hazard Index Across All Media and All Exposure Routes**				**5.6E+00**

Target Organ Breakdown for Central Nervous System	**5.6E+00**
Target Organ Breakdown for Liver	**5.1E+00**
Target Organ Breakdown for Kidney	**5.1E+00**

Table 10.5
RISK ASSESSMENT SUMMARY
REASONABLE MAXIMUM EXPOSURE
DAVISVILLE SITE 16

Location: Site 16
Scenario Timeframe: Future
Receptor Population: Commercial Worker
Receptor Age: Adult

Medium	Exposure Medium	Exposure Point	Chemical	Carcinogenic Risk				Chemical	Non-Carcinogenic Hazard Quotient				
				Ingestion	Dermal	Inhalation	Exposure Routes Total		Primary Target Organ	Ingestion	Dermal	Inhalation	Exposure Routes Total
Soil	Surface Soil	Site 16	**Dioxin/Furans**					**Dioxin/Furans**					
			TCDD-TEQ	9.9E-07	4.5E-07	1.0E-10	1.4E-06	TCDD-TEQ	NA	--	--	--	NA
			Inorganics					**Inorganics**					
			ARSENIC	7.0E-07	3.2E-07	7.1E-10	1.0E-06	ARSENIC	Skin	--	--	--	NA
			PAHs					**PAHs**					
			BENZO[A]PYRENE	6.0E-07	1.2E-06	2.6E-11	1.8E-06	BENZO[A]PYRENE	NA	--	--	--	NA
			DIBENZ[A, H]ANTHRACENE	4.9E-07	9.6E-07	--	1.4E-06	DIBENZ[A, H]ANTHRACENE	NA	--	--	--	NA
			(Total)	2.8E-06	2.9E-06	8.4E-10	5.7E-06		(Total)	---	---	---	---
			Total Risk Across Surface Soil				5.7E-06		Total Hazard Index Across Surface Soil				---
			Total Risk Across All Media and All Exposure Routes				5.7E-06		Total Hazard Index Across All Media and All Exposure Routes				---

Attachment A

Toxicological Profiles

Davisville Site 16
Human Health Risk Assessment

ACETONE

Acetone (C_3H_6O, CAS Registry Number 67-64-1) is a commonly used industrial solvent. It is also found in many household products, such as nail polish removers, paint removers, detergents or cleansers, many liquid or paste waxes or polishes, and particle board. Isopropyl alcohol metabolizes to acetone in the body (ATSDR 1994).

Cancer

Acetone is currently unclassified as to its human carcinogenicity (Category D) (USEPA 2001).

Mutagenicity

Acetone is negative in the following mutagenicity/genotoxicity assays: Syrian hamster embryo clonal assay; cell transformation in mouse and F344 rat embryos; Ames assay without metabolic activation; and *in vitro* cytogenetics and sister chromatid exchange in nonhuman cells (Calabrese and Kenyon 1991). Acetone was positive in *in vitro* cytogenetics assays with fungal (yeast) cells and cultured hamster fibroblasts (Calabrese and Kenyon 1991).

Systemic Toxicity

Acetone administered by gavage to groups of albino rats (30/sex/group) at 100 mg/kg-day (EPA 1986) for 90 days produced no observable effects. At 500 mg/kg-day, kidney weights significantly increased in females, hyaline droplet accumulation increased significantly in males and tubular degeneration increased in the kidneys of males. These effects continued to increase with dose. At 2500 mg/kg-day, RBC parameters were significantly increased in males at 30 days and in both males and females at 90 days; female kidney weights increased significantly; increased kidney-to-body and kidney-to-brain weight ratios occurred in both males and females, and accumulation of hyaline droplets became significant in females (USEPA 2001).

Inhalation toxicity data on acetone are limited. Subchronic exposures (3 hr/d, 5 d/wk for 8 wks) of rats to 0 or 19,000 ppm acetone elicited narcotic effects and other minor effects, but no mortality, gross or microscopic lesions, or altered hematological parameters were noted (Bruckner and Peterson, *in press*, cited in Calabrese and Kenyon 1991). While acetone can elicit narcotic effects in humans, exposures typically are of high-concentration and short duration.

Acetone dissolves fats from skin, causing dermatitis (Manahan 1989).

Toxicokinetics

Acetone is readily absorbed in direct proportion to dose by all routes of exposure (inhalation, oral, and dermal)(Rowe and Wolf 1963; Krasavage et al. 1982). Once absorbed, the highly soluble acetone is taken up into the blood, is rapidly and widely distributed throughout the tissues according to their water content. Large doses are excreted unchanged via the lungs, with lesser amounts appearing in the urine and sweat (Haggard et al. 1944; Rowe and Wolf 1963; Krasavage et al. 1982; Wigaeus et al. 1982). Lower doses are metabolized as described below(Haggard et al. 1944).

Metabolism

Acetone can be metabolized by three separate gluconeogenic pathways (Price and Rittenerg 1950; Sakami 1950; Mourkides et al. 1959; Casazza et al. 1984; Kosugi et al. 1986a,b). One pathway involves the conversion of acetone via methylglyoxal or methylglyoxal and D-lactate to D-glucose; two other pathways involve the conversion of acetone via L-1,2-propanediol that converts to the gluconeogenic precursors L-lactate or acetate. The ultimate fate of acetone is the conversion via glucose to ATP and carbon dioxide.

Toxicity Values

EPA has established an oral reference dose (RfD) for acetone of 1×10^{-1} mg/kg-day (USEPA 2001), based on changes in kidney and liver weights and nephrotoxicity in albino rats in a subchronic study (USEPA 1986). An uncertainty factor of 1,000 was incorporated in the calculations (10 for species extrapolation, 10 for human sensitivity variations, and 10 for extrapolating from subchronic to chronic exposures) (USEPA 2001).

EPA has not established cancer slope factors for acetone (USEPA 2001).

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ALUMINUM

Aluminum (Al, CAS Registry No. 7429-90-5) has a wide variety of uses in the building, construction, photographic, and food packaging industries, and many other uses in the manufacture of steel, aircraft, automobiles, printing inks, dental alloys, medications, explosives, incendiaries, and fireworks.

Cancer

Several studies of workers in aluminum reduction facilities showed excessive deaths from lung, pancreatic, lymphatic, brain, or bladder cancers, but they were confounded by simultaneous exposures to tobacco smoke or PAHs from coal tars. (ATSDR, 1990). An inhalation study by Kobayashi et al. (1968) was too seriously flawed in design and reporting to produce reliable information.

Noncarcinogenic Toxicity

Health effects research in humans and animals has shown that elevated levels of aluminum in the body may be toxic, particularly to the central nervous, skeletal and hematological systems. Much of aluminum toxicity depends on variations in absorption and chemical form of the aluminum complexes, their oxidation states, and interactions with dietary constituents. The toxicity of aluminum can be divided into three major categories: (1) the effect of aluminum compounds in the gastrointestinal tract following ingestion, (2) the effect in the lungs following inhalation, and (3) systemic toxicity of aluminum (ATSDR 1990). Aluminum compounds can alter absorption of other elements (i.e., fluoride, calcium, iron, cholesterol, phosphorus) in the gastrointestinal tract and alter gastrointestinal tract mobility by inhibition of acetylcholine-induced contractions. Inhalation of aluminum dusts can lead to the development of pulmonary fibrosis, producing both restrictive and obstructive pulmonary disease. One of the greatest health concerns is aluminum's proposed association with three forms of chronic encephalopathy in humans: senile dementia of the Alzheimer type, amyotrophic lateral sclerosis, and Parkinsonism-dementia (Ganrot, 1986), but there is no evidence that aluminum plays a role in the development of these diseases. A progressive fatal neurologic syndrome (characterized first by a speech disorder, followed by dementia, convulsions, and myoclonus) has been noted in patients on long-term intermittent hemodialysis treatment for chronic renal failure, and may be due to aluminum intoxication. Aluminum content of brain, muscle, and bone tissues is increased in these patients. Sources of the excess aluminum may be from oral aluminum hydroxide commonly given to these patients or from aluminum in dialysis fluid derived from tap water used to prepare the dialysate fluid.

Neurotoxicity

The neurotoxicity of aluminum is well documented in rabbits and cats, causing in these animals a range of behavioral abnormalities and brain neurofibrillary degenerative

changes at high doses injected parenterally or intracranially; hamsters and monkeys were found to be much less sensitive (Ganrot 1986, Lione 1985). Subchronic studies in rats and mice have demonstrated in the test animals such neurobehavioral deficits as impaired operant learning, changes in grip strength and startle response, and impaired motor coordination (Bernuzzi et al. 1989; Donald et al. 1989; Golub et al. 1989, 1992, 1995; Muller et al. 1990). Florence et al (1994) fed aluminum to rats for 6 months and found histopathological changes (vacuolation of cell bodies, swelling of astrocytic processes) of unknown functional significance in the rat brain as a result. These changes have not been causally related to neurobehavioral deficits.

Neurodevelopmental Toxicity

Minimal neurotoxicity was observed in the offspring of mice exposed to aluminum lactate (Golub et al. 1995). Groups of 16 pregnant Swiss-Webster mice were fed 25 (control group), 500, or 1000 mg Al/kg with aluminum lactate throughout gestation and lactation (Donald et al. 1989). No treatment-related changes were observed in maternal survival, body weight, food intake, toxic signs or neurobehaviour. However, a battery of neurobehavioral tests performed on pups showed that a significant ($p=0.007$) number of pups in the high-dose group had impaired vertical-screen climb performance. In the low-dose group, given the equivalent of 100 milligrams per kilogram per day (mg/kg-day), a decrease in forelimb grip strength and an increase in the amount of foot splay were observed.

Colomina et al. (1992) studied the influence of lactate on the potential developmental toxicity of aluminum. Groups of 11-13 Swiss albino CD_1 mice were administered 57.5 mg Al/kg bw-day as aluminum hydroxide, aluminum lactate, or aluminum hydroxide together with lactic acid (570 mg/kg-day) by gavage on gestation days 6-15. Other groups were treated only with the same amount of lactic acid or distilled water (controls). Maternal weight gain was significantly lower in the aluminum lactate-treated mice--and significantly less food was consumed by this group--than in controls. In the offspring of these females, fetal body weight was also significantly reduced and accompanied by increased incidence of cleft palate, dorsal hyperkyphosis, and delayed parietal ossification. These developmental effects were not seen in any of the pups of the control groups or the aluminum hydroxide dosing groups. A flaw in this study was the lack of accounting for aluminum in the diet of the test animals, since commercial grain-based mouse feeds contain 200-1200 ppm aluminum and varying amounts of other trace metals (Golub et al. 1992).

Colomina et al. (1994) also administered doses of 0 or 104 mg Al/kg bw-day as aluminum hydroxide on days 6-15 of gestation to female Swiss mice. Again, dietary aluminum was not reported. A NOAEL of 104 mg Al/kg-day was reported in this study, which examined maternal body or organ weight, and the per litter number of implantations, resorptions, dead fetuses, sex ratio and fetal body weight, as well as their percentage of positive postimplantation loss. The study found no significant differences between test groups and controls. Thus, from the two Colomina studies, it can be

concluded that aluminum lactate appears to be a more potent developmental toxicant in mice than the less water-soluble aluminum hydroxide (ATSDR 1990).

Toxicokinetics

Aluminum occurs normally in human body tissue, with the highest concentration (half of the normal body burden of 30-50 mg) found in bone, and about one-fourth found in the lungs (Ganrot 1986). Animal experiments have shown that it can also accumulate in the brain (especially the hippocampus), liver, heart and other muscle tissue, and the kidneys. Retention in these organs and tissues apparently increases with age and with the length of the exposure period (ATSDR 1990). The urinary aluminum half-life in workers exposed via inhalation for less than a year was 9 days; in those exposed for more than 10 years, the urinary half-life was 6 months (Sjogren et al. 1988). Both human and animal evidence indicates that absorption through the gastrointestinal tract is low after ingestion of aluminum, but is enhanced by vitamin D and parathyroid hormone and is affected in various ways by other ions (ATSDR 1990). Evidence also suggests that aluminum does not easily transfer from the blood to the brain except under conditions of high blood aluminum concentrations (ATSDR 1990).

An acute oral study found a three-fold increase in urinary levels of aluminum in patients after ingestion of aluminum hydroxide (Gorsky et al. 1979), implying absorption. In three volunteers, urinary levels of aluminum rapidly increased following an 8-hour inhalation exposure to aluminum dust from 3 μg/L to 14-414 μg/L (Sjogren et al. 1985).

Several animal inhalation studies detected aluminum in the lungs after exposure; however, other tissue, urine, and plasma levels were not assessed to definitively establish absorption (ATSDR 1990).

Metabolism

Because aluminum is an element, it is always found attached to other chemicals, and these attachments can change within the body. The free ion (Al^{+3}) is easily bound to other substances and structures. Once in the blood, the free ion competes with other ions to bind mainly with transferrin in the plasma, but it also binds to bone tissue in metabolically active areas of the bone (Ganrot 1986). Uptake by organs and tissues from transferrin is relatively slow. Most ingested aluminum is unabsorbed and eliminated in the feces. The kidney is the major route of excretion following inhalation, and the major route for any aluminum absorbed via ingestion (ATSDR 1990).

Toxicity Values

The available systemic toxicity data on elemental aluminum have been found to be inadequate for quantitative risk assessment (USEPA 2001). A chronic oral RfD (1 mg/kg-day) value was obtained from EPA's Environmental Criteria and Assessment Office, Cincinnati, based on a LOAEL of 100 mg/kg-day (Donald et al. 1989, Golub et

al. 1995) for neurotoxicity, with application of an uncertainty factor of 100 (3 for use of a minimal LOAEL, 10 for species extrapolation, and 3 for human variability).

An inhalation reference dose of $1x10^{-3}$ has been established from EPA's Environmental Criteria and Assessment Office, Cincinnati. EPA (USEPA 2001) has not established SFs for aluminum.

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ANTIMONY

Antimony (Sb, CAS registry number 7440-36-0) is an element typically used in metal alloys, rubber, matches, ceramics, pigments, paints, lacquers, and textiles. Some trivalent organic antimony compounds are used to treat parasitic diseases, such as *Schistosomiasis* (ATSDR 1990).

Cancer

Antimony is currently not classified with regard to its potential for carcinogenicity (USEPA 2001). Lung tumors have been observed in rats exposed via inhalation to 4.2 or 36 mg/m^3 antimony as antimony trioxide (Groth et al. 1986; Watt 1980, 1983) or to 17.48 mg/m^3 antimony as antimony trisulfide for 1 year (Groth et al. 1986; Wong et al. 1979). An increased incidence of lung tumors was not observed, however, in rats exposed via inhalation to 3.76 mg antimony/m^3 as antimony trioxide for 1 year (Bio/dynamics 1989) or in pigs exposed to 4.2 mg antimony/m^3 as antimony trioxide for 1 year (Watt 1983). The carcinogenic potential of antimony compounds in rodents may be related to particle size and rates of deposition and clearance from the respiratory tract. Oral exposure to 0.35 mg/kg-day of potassium antimony tartrate over a lifetime did not induce cancer in rats (Schroeder et al. 1970) or mice (Kanisawa and Schroeder 1969). No data are available to evaluate the potential carcinogenicity of antimony compounds via dermal exposure.

Mutagenicity

No *in vivo* genotoxic studies have been conducted with antimony compounds. *In vitro* studies have yielded mixed results with antimony compounds giving positive results for DNA damage in bacterial tests (Kanematasu et al. 1980) and for chromosomal aberrations and breakage in mammalian cell assays (Tu and Sivak 1984; Paton and Allison 1972), and negative results for gene mutation and chromosomal transformation in mammalian cell assays (Tu and Sivak 1984).

Systemic Toxicity

Adverse health effects have been observed in humans and animals following oral exposure to a variety of antimony compounds (ATSDR 1990). Oral administration of a single dose of an organic antimony compound resulted in death in rats (Bradley and Frederick 1941) which was attributable to myocardial failure. Mortality was not observed in rats following a single dose of inorganic antimony (Fleming 1982; Myers et al. 1978; Smyth and Thompson 1945), suggesting that inorganic antimony compounds are less toxic than organic antimony compounds. Chronic ingestion by rats of 0.35 mg/kg-day of potassium antimony tartrate in drinking water has resulted in increased mortality and altered blood chemistry (Schroeder et al. 1970). Other studies with potassium antimony tartrate have not demonstrated any effects on longevity (Schroeder et al. 1968; Kanisawa and Schroeder 1969).

Antimony compounds are frequently used in medicine as anti-parasitical agents. Therapeutic administration of antimony-containing compounds has produced cardiac effects, liver toxicity, pulmonary congestion, skin reactions, vomiting, diarrhea, gastric discomfort and ulcers in treated humans (ATSDR 1990). Acute exposure to an antimony-containing therapeutic agent administered via intramuscular injection resulted in the death of an adult and a child (Rugemalila 1980); however, it is not clear whether these deaths were attributable solely or in part to antimony administration.

The respiratory tract is a target organ in both humans and animals following inhalation exposure to antimony. High occupational inhalation exposure to antimony dusts has resulted in respiratory tract irritation, pneumoconiosis, and impaired pulmonary function in workers (Cooper et al. 1968; Potkonjak and Pavlovich 1983). These workers, however, were also exposed to other toxic compounds, including arsenic oxide, iron oxide, hydrogen chloride, and hydrogen sulfide, and thus it is not possible to attribute the observed adverse effects solely or in part to antimony exposure. Dermatosis and ocular irritation have also been reported in humans following exposure to airborne antimony (Potkonjak and Vishnijich 1983).

Animal studies have shown that lung fibrosis and lipoid pneumonia may occur as a result of chronic inhalation exposure to antimony trisulfide or antimony trioxide (ATSDR 1990). Other symptoms include: changes in blood pressure and EKG readings; gastrointestinal effects such as vomiting, diarrhea, gastric discomfort and ulcers; and liver toxicity.

Exposure to airborne antimony has been reported to cause dermatosis and ocular conjunctivitis in humans which may result from dermal contact with the antimony aerosol (ATSDR 1990). Alopecia was noted in rats exposed to airborne antimony trioxide at doses of 0.92 mg/m^3 or above (Bio/dynamics 1985); however, it was not clear whether this effect was due to dermal contact or to a systemic effect resulting from airborne exposure.

Toxicokinetics

Data from animal studies indicate that absorption of antimony compounds from the respiratory tract is a function of particle size (ATSDR 1990). A greater deposition of antimony tartrate in the upper respiratory tract was observed with a particle size of 1.6 um mean mass aerodynamic diameter (MMAD) compared with exposure to particles of 0.7 or 0.3 um MMAD (Felicetti et al. 1974a; Thomas et al. 1973). Absorption and lung clearance rates also varied with particle size (Thomas et al. 1973). No information is available on differences in absorption rates via inhalation among antimony compounds (ATSDR 1990).

In animals, antimony is slowly absorbed from the gastrointestinal tract at rates ranging from 2 percent to 7 percent for antimony tartrate and antimony trichloride (Felicetti et al. 1974b; Gerber et al. 1982). It has been suggested that gastrointestinal absorption of antimony salts in humans is probably less than 10 percent (ATSDR 1990). Although

quantitative information on differences in absorption among antimony compounds is not available, the International Commission on Radiological Protection (ICRP 1981) has recommended that 10 percent for antimony tartrate, and 1 percent for all other forms of antimony, be used as reference values for gastrointestinal human absorption.

Distribution in animals of trivalent antimony absorbed via inhalation tends to result in concentration in red blood cells and liver, while pentavalent antimony concentrates in the plasma (Goyer 1991). Other target organs of distribution include the thyroid gland, skeleton and fur (Felicetti et al. 1974b). Following oral absorption, the major sites of accumulation of antimony compounds are the liver, kidney, bone, lung, spleen, and thyroid (Sunagawa 1981). Accumulation in these organs, however, is not dose-related, suggesting that decreased absorption occurs at higher antimony concentrations.

Metabolism

Antimony does not undergo biotransformation. Its metabolism appears to consist mainly of covalent interactions with sulfhydryl groups and phosphate, and reversible binding interactions with endogenous ligands such as proteins (ATSDR 1990). The toxicologic significance of these interactions is not known (ATSDR 1990).

Excretion appears to occur mainly in the urine in humans occupationally exposed to airborne antimony trioxide (Ludersdorf et al. 1987). In animals, antimony is excreted via the urine and feces. If antimony is administered parenterally, the urine/feces ratio of antimony excretion depends on the valence form of the compound: for trivalent antimony injection, urine is the predominant medium of excretion, whereas for pentavalent antimony injection, excretion in the feces predominates (Edel et al. 1983; Felicetti et al. 1974b). With regard to oral exposure, animal studies have shown that ingested antimony is not well absorbed from the gastrointestinal tract and that fecal excretion is the primary route of excretion (Felicetti et al. 1974b; Gerber et al. 1982). It is likely that this route is also predominant for humans who ingest antimony compounds.

Toxicity Values

EPA (USEPA 2001) has established an oral reference dose of 4×10^{-4} mg/kg-day for antimony compounds, based on a chronic study in which rats were administered 0.350 mg/kg potassium antimony tartrate daily in drinking water (Schroeder et al. 1970). Significantly shortened life spans, altered cholesterol levels, and–in males–decreased blood glucose levels were critical effects. A decrease in mean heart weight of males was also noted. Since there was only one dose group, a NOAEL could not be determined. The oral RfD was calculated using an uncertainty factor of 1000 (10 for interspecies conversion, 10 for intrahuman sensitivity, and 10 for the failure to determine a NOAEL) (USEPA 2001).

EPA has not calculated an inhalation reference concentration or cancer slope factors for this compound. (USEPA 2001).

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ARSENIC

Arsenic (As, MW 74.9, CAS Registry No. 7440-38-2) is a ubiquitous metalloid present in the environment from natural and anthropogenic sources. Arsenic is a natural component of the earth's crust. It originates from natural sources such as volcanoes and erosion from mineral deposits, but also from human activities such as chemical production and use, coal combustion, and waste disposal. Arsenic can exist in several different valence states and in many different inorganic and organic forms. The form of arsenic with the greatest commercial importance is inorganic arsenic trioxide (As_2O_3), which is produced from flue dust collected during the smelting of copper and lead. There has been no commercial production of As_2O_3 in the United States since 1985, although it is imported for industrial use.

Arsenic is one of the most widely studied toxicants. Analysis of the toxic effects of arsenic is complicated by the fact that arsenic can exist in several different inorganic and organic compounds. Most cases of human toxicity have been associated with exposure to inorganic arsenic (ATSDR 1993). While As_2O_3 is the most common inorganic arsenical in air, a variety of inorganic trivalent arsenites or pentavalent arsenates occur in water, soil, and food (ATSDR 1993). Trivalent arsenites are somewhat more toxic than pentavalent arsenates (USEPA 2001); however, the difference in relative potency is small. An additional complexity in the analysis of arsenic toxicity is that there are no suitable animal models for carcinogenicity.

Cancer

EPA has classified arsenic as a human carcinogen (Category A), based on observations of increased lung cancer mortality in populations exposed mainly via inhalation and observations of increased skin cancer incidence and increased mortality from multiple internal organ cancers in populations consuming drinking water containing high concentrations of arsenic (USEPA 2001).

Evidence from a large number of occupational epidemiologic studies indicates that high-dose inhalation exposure to inorganic arsenic is associated with an increase in lung cancer. Most studies involve workers exposed primarily to arsenic trioxide dust in air at copper smelters (e.g., Enterline et al.1987; Enterline and Marsh 1982; Axelson et al.1978), but increased incidence of lung cancer has also been observed at chemical plants where exposure was primarily to arsenate (Ott et al.1974; Sobel et al.1988). Although several studies suggest that residents living near smelters or arsenical chemicals may also have increased risk of lung cancer (Pershagen 1985; Brown et al.1984), the increases are small and may not be biologically significant (e.g., Frost et al.1987).

Several human studies provide sufficient exposure data to permit quantification of cancer risk (Tseng et al.1968; Tseng 1977). In general, the data indicate that there is an approximately linear increase in relative risk as a function of increasing cumulative exposure (USEPA 2001). Animal studies do not show evidence of a carcinogenic effect

from inhalation exposure of arsenic (USEPA 2001); however, two intratracheal instillation studies in hamsters have demonstrated that both arsenite and arsenate can increase the incidence of lung adenomas and/or carcinomas (Ishinishi et al.1983; Pershagen and Bjorklund 1985).

One of the most reliable epidemiologic studies that provide dose-response data is the investigation by Tseng et al. (1968). In this study, the quantitative risk of skin cancer from exposure to inorganic arsenic by ingestion was examined. In this investigation, 40,421 people in Taiwan were exposed to arsenic in drinking water at levels ranging from 100 to 1,800 ug/L. The control population consisted of 7,500 people who were exposed to undetectable levels up to 17 ug/L arsenic in drinking water. Results showed an age- and dose-dependent increase in the incidence of skin cancer in the exposed population, while there were no arsenic-related skin cancers detected in the control population. EPA used this study to calculate a unit risk of $5x10^{-5}$ based on lifetime exposure to water containing 1 ug/L arsenic.

Mutagenicity

Arsenic has been tested in a range of prokaryotic and eukaryotic systems. In general, arsenic is either inactive or extremely weak as an inducer of gene mutations *in vitro*; however, it is clastogenic and induces sister chromatid exchanges in a variety of cell types, including human cells (USEPA 1988). Arsenic does not appear to induce chromosome aberrations *in vivo* in experimental animals; however, it may do so in humans (NRC 1977). Several studies also suggest that arsenic may affect DNA by the inhibition of DNA repair processes or by base-pair substitution (USEPA 1988).

Noncarcinogenic Toxicity

Workers exposed to arsenic dusts in air may experience irritation to the mucus membrane of the nose and throat, which may lead to laryngitis, bronchitis, or rhinitis (Morton and Caron 1989). Very high exposures may cause perforation of the nasal septum (Pinto and McGill 1953). These effects, including perforation, were usually mild and did not result either in impaired respiration or illness. Several studies revealed that inhalation of high levels of arsenic dust or fumes led to nausea, vomiting, and diarrhea in workers (Beckett et al. 1986; Bolla-Wilson and Bleecker 1987; Morton and Caron 1989). Neurological injury may occur in humans after inhalation of inorganic arsenic, including peripheral neuropathy of sensory and motor neurons (numbness, loss of reflexes, muscle weakness) (Feldman et al. 1979; Landau et al. 1977). Hallucinations, agitation, emotional lability, and memory loss may also result (Beckett et al. 1986; Morton and Caron 1989). Adverse neurological effects tend to diminish, but may persist past exposure period (Beckett et al. 1986).

Developmental effects of arsenic exposure have been implicated in the unborn fetuses of pregnant women living near a copper-smelting plant in Bulgaria (Tabacova 1986). Fatal defects (e.g., small forebrains and underdeveloped earpits) occurred at a rate of 3.6 per 1,000 live births, which is three times the national rate. From a cohort of about 15,000

women living near the plant, increased placental concentration of arsenic, elevated lipid peroxides, and decreased GSH in maternal and cord blood were found. This suggested that arsenic was responsible for the oxidative damage in the pathogenesis of prenatal birth defects. The hypothesis was tested in an experimental *in vitro* mouse embryo model.

Chronic exposure of experimental animals to arsenic has been investigated by several studies. Tissues adversely affected by arsenic exposure include the gastrointestinal, cardiovascular, hematopoietic, renal, nervous, and respiratory systems.

In a chronic toxicity study (Byron et al. 1967), beagle dogs (6/group) were fed sodium arsenate or sodium arsenite at doses equivalent to 5, 25, 50, or 125 ppm arsenic in the diet, corresponding to an average daily dose of 0.2, 1.0, 2.1, or 5.2 mg As/kg-day with an assumed body weight of 12 kg for each dog. Four out of six dogs fed the highest dose of As died within 9 months. The main effects were anorexia and listlessness, with weight loss of 44-61 percent from the beginning of the study. Moderate anemia and atrophy of numerous tissues were revealed after hematologic and histologic examinations. A chronic NOAEL of 2.1 mg As/kg-day was identified in dogs since there was no difference between controls and dogs fed 50 ppm As^{3+} or As^{5+} in their diet.

In a 1-year study involving rhesus monkeys were exposed to As^{5+} (in the form of $2Na_3(PO_4AsO_4VO_4)$ "NaF" $18H_2O$) in milk at doses of 2.8 mg/kg-day (3-day and 8-week old monkeys) and 5.7 mg/kg-day (8-weeks old) (Heywood and Sortwell 1979). Effects seen in one monkey dosed with 2.8 mg As/kg-day beginning at 3 days of life included sudden weakness, dehydration along with bronchopneumonia, hemorrhage, edema, and necrosis in the brain. Another animal from the highest dose of arsenic, had acute inflammation of the small intestine and moderate regression of the thymus. Surviving animals had normal EEGs and normal levels of neurological function. There was no evidence of delayed toxicity in surviving animals.

Male weanling Sprague-Dawley rats (10/group) were exposed to 0 or 50 ug/mL sodium arsenate for 320 days in drinking water (Carmignani et al. 1983). Histological examination revealed that the liver and kidneys accumulated significant levels of arsenic at 25.2 and 43.0 mg/kg, respectively, and swollen hepatocytes were noted near the centrilobular veins of the liver. Focal changes in the glomerulus and tubules were seen in the kidneys. No changes were noted in the myocardium, gastrocnemius, arterial vessels, lungs, brain, or sciatic nerves. Sympathetic hyperactivity, hypersensitivity, or both were induced by arsenic, and the authors speculated that these findings may explain the cardiovascular effects in people chronically exposed to arsenic.

CD male and female mice were fed 5 mg As^{3+}/L sodium arsenite in their drinking water, which corresponds to 0.35 mg/kg-day, in a special environment designed to minimize exposure to trace metals (Schroeder and Balassa 1967). After 180 days, growth rate and body weights were not affected, but a decrease in body weight in males was apparent after 360 and 540 days. There was also a decrease in survival rates at 18 months in males

and 21 months in females, with a median life span that was reduced by 74 and 76 days in males and females, respectively.

Developmental Toxicity

High levels of arsenic can cause developmental effects in animals. Slight decreases in fetal weight resulted after mice were exposed to 2 mg As/m^3 as As_2O_3 on days 9-12 of gestation. Higher levels of arsenic (20 mg/m^3) produced skeletal malformations and an increase in fetal deaths (Nagymajtenyi et al. 1985). Other studies have reported an increase in fetal mortality from 2-68 mg As/kg-day sodium arsenite (Baxley et al. 1981; Hood and Harrison 1982). Baxley et al. (1981) exposed pregnant CD-1 mice to single oral doses of 1, 20, 40, or 45 mg/kg sodium arsenate by gavage on days 8-15 of gestation. The frequency of dead or resorbed fetuses was significantly elevated in animals exposed to 40 or 45 mg/kg on days 10, 12, 13, 14, or 15 of gestation. Hood and Harrison (1982) exposed pregnant hamsters to a single gavage dose of 25 mg/kg sodium arsenite on days 8, 11, or 12 of gestation, or 20 mg/kg on days 9 or 10. Prenatal mortality was significantly elevated in animals dosed with 25 mg/kg on gestational days 8 or 12. Small increases in the percentage of fetuses that were malformed were noted in treated groups, although these were not significant. Similarly, Hood and Harrison (1982) performed a similar experiment in hamsters dosed by a single intraperitoneal amount of 5 mg/kg sodium arsenite on days 8, 11, or 12, or 2.5 mg/kg on days 9 or 10 of gestation. Again, prenatal mortality was elevated, and although there were small increases in malformations, they were not significant. Hood and Harrison concluded that arsenite is significantly less toxic when administered orally than intraperitoneally. Intraperitoneal injections of 45 mg/kg sodium arsenite on days 6-12 of gestation in pregnant Swiss-Webster mice resulted in the following fetal malformations: exencephaly, micrognathia, protruding tongue, agnathia, open eye, exophthalmos, anophthalmia, missing pinna, cleft lip, hydrocephalus, umbilical hernia, eventration, ectrodactyly, micromelia, limb and tail malformations, and skeletal defects. Similar adverse effects were seen in exposed Wistar rat fetuses (Beaudoin 1974), golden hamsters (Willhite 1981; Carpenter 1987).

Fetal mortality was increased and malformation resulted in experimental animals exposed to organic forms of arsenic. Albino CD rats and CD-1 mice given repeated doses of DMA, a metabolite of arsenic, during gestation had significantly elevated fetal mortality and showed such developmental effects as skeletal anomalies (delayed ossification and supernumerary ribs), malformed palate, cleft lip, reduced fetal weight (Rogers et al. 1981).

Reproductive Toxicity

In a three-generation reproductive toxicity study in Charles River mice given sodium arsenate in drinking water at an average dose of 0.35 mg As^{3+}/kg-day, no significant effects were detected, although a trend toward a decreased number of pups per litter and slightly altered male:female sex ratios were observed (from 1.03 to 1.30 in the F_2 generation, and from 1.00 to 1.71 in the F_3 generation (Schroeder and Mitchener 1971).

In another study, male and female Harlan/ICR Swiss mice dosed 3 times per week for 10 weeks with 0, 11.9, or 119 mg/kg-day MMA, a metabolite of As, prior to mating and during gestation produced fewer litters than normal (Prukop and Savage 1986). None of the animals receiving the highest dose of MMA produced litters, and only 50 percent of the animals dosed with 11.9 mg/kg-day MMA produced litters, compared with 80-100 percent in controls. This was attributed to decreased fertility of the male mice.

No studies have been located that discuss other systemic effects of exposure to arsenic in experimental animals.

Toxicokinetics

Most of the existing data on the toxicokinetics of arsenic is on the inorganic form. Both arsenate and arsenite are well absorbed by both the oral and inhalation routes.

Arsenic in air exists as particulate matter, and absorption by inhalation involves deposition of the particles onto the surface of the lungs and absorption of arsenic from the deposited material. Deposition was estimated to be about 40 percent and absorption was 75-85 percent in lung cancer patients exposed to arsenic in cigarette smoke (Holland et al. 1959), making the percentage of inhaled arsenic 30 34 percent. Animal studies on As (i.e., sodium arsenite, sodium arsenate, and arsenic trioxide) via intratracheal instillation suggest that nearly all of the deposited material is absorbed, because clearance of the compounds from the lungs was rapid and nearly complete (60-90 percent cleared within 1 day) (Marafante and Vahter 1987; Rhoads and Sanders 1985). In contrast, insoluble forms of As (i.e., arsenic sulfide and lead arsenate) cleared more slowly, suggesting that the rate of absorption is lower for the insoluble forms of arsenic (ATSDR 1993).

Absorption of arsenates and arsenites across the gastrointestinal tract is nearly complete. Measurements of human fecal excretions given oral doses of arsenite reported that less than 5 percent was recovered, indicating that absorption was about 95 percent (Bettley and O'Shea 1975). Again, ingestion of less insoluble forms of arsenic such as arsenic triselenide did not lead to a high percentage of absorption across the gastrointestinal tract (Mappes 1977). The data on dermal absorption of inorganic arsenic are limited and not quantitative.

Once absorbed, arsenic is distributed throughout the body to the liver, kidney, skeleton, gastrointestinal tract, and other tissues. Autopsies of people exposed to background levels of arsenic have revealed that arsenic is present in all tissues of the human body at approximately comparable concentrations (Liebscher and Smith 1968). Absorbed arsenic can also cross the placenta and be distributed to fetuses (Hood et al. 1987, 1988). The metabolites of both inorganic and organic arsenic appeared to be distributed equally in all body tissue following oral exposure (Takahashi et al. 1988; Yamauchi and Yamamura 1984, 1985; Stevens et al. 1977; Yamauchi et al. 1988).

A review by Thompson (1993) indicates that several *in vivo* and *in vitro* studies have elucidated the metabolic and detoxification pathway for arsenic in mammals (Vahter 1981; Vahter and Envall 1983; Hirata et al. 1988; Marafante and Vahter 1987; Takahashi et al. 1988; Maiorino and Aposhian 1985; Marafante et al. 1985; Vahter and Marafante 1983), including humans (Buchet et al. 1981a,b; Crecelius 1977; Lovell and Farmer 1985; Smith et al. 1977; Tam et al. 1979; Vahter 1986). Analysis of urinary excretion products from humans and animals revealed increased levels of inorganic As^{3+}, As^{5+}, methylarsonic acid (MMA), and dimethylarsonic acid (DMA). The metabolism of inorganic arsenic involve two processes: oxidation/reduction of As^{5+} and As^{3+} species and methylation. Specifically, inorganic arsenic is converted via methylation in the liver to MMA and to DMA, which is the principal metabolite. Both MMA and DMA form conjugates with glutathione or glutathione derivatives and are excreted in urine. Since methylation is enzyme-dependent, saturation kinetics appear to determine the toxicity, or carcinogenicity, of arsenic in humans. At low doses, arsenic can be effectively detoxified, whereas at higher doses the detoxification pathway may become increasingly saturated, thereby increasing the possibility of macromolecular binding, resulting in pathological changes which could include tumors (Cheng et al. 1992; Smith et al. 1992). Data on the point at which saturation is reached is unclear.

Toxicity Values

Data by Tseng (1977) and Tseng et al. (1968) from a population epidemiology study in Taiwan were used to derive an ingestion RfD of 0.0003 mg/kg-day (USEPA 2001). The critical effects were considered to be hyperpigmentation, keratosis, and possible vascular complications. Since hyperpigmentation and keratosis of the skin are lesions that can progress to skin neoplasms, this toxic endpoint is considered to be appropriate for RfD derivation. The NOAEL is derived by multiplying the average concentration in drinking water by 4.5 L/day (because of the hot climate, typical daily drinking estimates are approximately doubled for this study population), adding in the contribution of arsenic in food, and dividing exposure by average body weight for Taiwanese adults (Abernathy et al. 1989). The RfD of 0.0003 mg/kg-day incorporates an additional uncertainty factor of 3 for the lack of data concerning the potential toxicity of arsenic. EPA has not derived an inhalation RfC for arsenic (USEPA 2001).

Considerable scientific controversy has surrounded the derivation of an estimated SF for arsenic by ingestion, principally through drinking water. Based on a study in Taiwan (Tseng 1977; Tseng et al. 1968), EPA has developed a drinking water unit risk of $5x10^{-5}$ $(ug/L)^{-1}$, which was used to generate an oral slope factor of 1.5 $(mg/kg\text{-}day)^{-1}$ (USEPA 2001). The SF for ingestion was adopted in this risk assessment for skin contact, as a default condition. The inhalation SF is based on the geometric means of several occupational epidemiologic studies in which a significant increase in the incidence of lung cancer occurred among workers exposed to high concentrations of airborne arsenic. The inhalation SF is $4.3x10^{-3}$ $(ug/m^3)^{-1}$, or $1.5x10^{-2}$ $(ug/kg\text{-}day)^{-1}$ (USEPA 2001).

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BARIUM

Barium (CAS 7440-39-3) is dense alkaline earth metal that does not occur free in nature; it exists in combination with other elements in a divalent cation form, primarily as barium sulfate or barium carbonate ores. Barium enters the environment through the weathering of rocks, minerals, and anthropogenic release mechanisms, such as industrial processes and coal and oil combustion (USEPA 1998).

Carcinogenicity

EPA (USEPA 2001) indicates that there are insufficient data for an assessment of the carcinogenicity of barium compounds (classified Group D). Similarly, ATSDR (1992) notes a lack of carcinogenicity studies via inhalation or dermal exposure routes, but summarizes two rodent studies (Schroeder and Mitchener 1975 a,b) that observed no significant differences in tumor incidence between treated and control animals. ATSDR, however, considers these rodent studies inadequate for evaluating the carcinogenic potential of barium due to insufficient numbers of animals, no determination of an MTD, only one exposure dose, incomplete histology, and an unspecified purity of the test material.

Mutagenicity

Information on the mutagenicity of barium compounds is limited. No in vivo studies have been conducted. Barium chloride dihydrate was not mutagenic in *Salmonella typhimurium* strains TA97, TA98, TA100, TA1535, or TA1537, with or without exogenous metabolic activation (S9). It was mutagenic in L5178Y mouse lymphoma cells in the presence of S9, but it did not induce sister chromatid exchanges or chromosomal aberrations in cultured Chinese hamster ovary cells, with or without S9 (NTP 1994).

At various gradually increasing concentrations, barium nitrate gave negative results in both the Ames test (with and without metabolic activation), and in the mitotic crossing-over test, the mitotic genic conversion test, and the retromutation test in *Saccharomyces cerevisiae*, with and without metabolic activation (Monaco et al. 1991). Barium chromate was not mutagenic in water (Venier et al. 1985).

Systemic Toxicity

Exposures to barium via the oral route are used in the majority of studies evaluating potential toxicity. ATSDR (1992) indicates a variety of effects following subchronic or chronic oral administration of barium, including mortality, respiratory, cardiovascular, hematological, musculoskeletal, hepatic, renal, neurological, and other effects. For both subchronic (15 to 364 days) and chronic (>365 days) exposures, cardiovascular effects are the most sensitive endpoint. With subchronic exposures, rodent NOAELs for all systemic effects except cardiovascular are approximately 10 mg/kg-d. For cardiovascular

effects, rat NOAELs are reported as low as 0.64-0.71 mg/kg-d (Perry et al. 1983, 1985, 1989) and the human NOAEL is reported as 0.21 mg/kg-d (Wones et al. 1990).

Studies identifying the adverse effects of subchronic or chronic barium exposure via inhalation are limited in their utility for linking adverse health effects with barium exposure (ATSDR 1992). Even with the limited utility of these studies, these studies suggest a potential association between non-acute barium inhalation exposures and respiratory, cardiovascular, hematological, hepatic, developmental, and reproductive effects (ATSDR 1992).

Toxicokinetics

No studies of the absorption of barium in humans have been found. Animal studies suggest that the absorption of inhaled barium depends on how much reaches the alveolar spaces within the lungs, the upper respiratory tract clearance rate, and the solubility of the particular form of barium under study. In animal studies, barium has been traced to epithelial membranes, the lymphatic system, and the skeleton. In humans barium has been found primarily in the skeleton and teeth (ATSDR 1992).

Metabolism

Barium taken by mouth is poorly absorbed, and most of the dose is excreted in the feces, with about three percent excreted in urine. With intravenous injections, about 9 percent is excreted in urine and 84 percent in feces (ATSDR 1992).

Toxicity Values

EPA (USEPA 2001) has established an oral RfD for barium of 7×10^{-2} mg/kg-day, based on increased blood pressure from subchronic and chronic human drinking water studies (Brenniman and Levy,1984; Wones et al. 1990) and other supporting rodent studies in which kidney weight increased (Perry et al. 1983; McCauley et al. 1985; Schroeder and Mitchener, 1975a,b; Tardiff et al. 1980). The RfD is based upon a NOAEL of 10 mg/L, converted to 0.21 mg/kg-d, with the application of an aggregate uncertainty factor of 3. The uncertainty factor is modified from the default 10-fold values for the use of a subchronic study and for protection of sensitive individuals because EPA considers the critical study's unique focus and other supporting studies as supporting an uncertainty factor of 3.

Although human and animal data indicate that the respiratory system is a target for barium toxicity, and that systemic effects such as hypertension may result from exposure, EPA cannot recommend an inhalation RfC at the present time (USEPA 2001), because both the human and animal study parameters and results were poorly reported.

Barium is is presently listed by EPA in Group D (not classifiable as to human carcinogenicity); but it is considered not likely to be carcinogenic to humans via oral

exposure under EPA's proposed guidelines for carcinogenic risk assessment (USEPA 1996). Its carcinogenic potential following inhalation exposure cannot be determined.

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BENZ(a)ANTHRACENE

Benz(a)anthracene (BaA) ($C_{18}H_{12}$, CAS registry number 56-55-3) is a polynuclear aromatic hydrocarbon that exists as solid, colorless plates with a greenish-yellow fluorescence at standard temperature and pressure. It occurs as a by-product of incomplete combustion of organic substances. It is a constituent in mainstream cigarette smoke, exhaust emissions from gasoline engines, charcoal-broiled foods, and crude oils (HSDB 2000).

Cancer

Benz(a)anthracene is classified by EPA in the category B2, probable human carcinogen, based on a lack of human data but sufficient data from animal bioassays (USEPA 2001).

There were no reported epidemiologic data in humans assessing the carcinogenicity of BaA, although BaA is a component of mixtures that have been associated with human cancer. These mixtures include coal tar, soots, coke oven emissions, and cigarette smoke (USEPA 2001).

In a group of male B6AF1/J mice exposed by gavage to 3 percent solutions BaA in dioctyl ester of sodium sulfosuccinic acid three times per week for 5 weeks, there was an increased incidence of pulmonary adenoma and hepatoma in treated animals versus controls (Klein 1963). After 546 days, 100 percent of treated animals had hepatomas versus 10 percent of controls. In another study, mice (strain and sex not specified) dosed with multiple gavage of 0.5 mg BaA in mineral oil (17 mg/kg for 8 or 16 treatments at 3-7 day intervals) resulted in forestomach papillomas in 2 out of 27 treated mice and none in controls (USEPA 2001).

BaA yielded positive results in tests for complete carcinogenicity and initiating activity in skin painting assays in C3H/He, CAF1, and ICR/Ha mouse strains (USEPA 1994). C3H/He mice were painted 3 times weekly with several concentrations (0.002 percent, 0.02 percent, 0.2 percent, or 1 percent) of BaA in toluene or in n-dodecane. Tumors, mostly malignant, formed after 50 weeks off treatment in the three lower concentrations (Bingham and Falk 1969).

Groups of male and female CD-1 mice received intraperitoneal injections of BaA in DMSO on days 1, 8, and 15 of age (total dosage 638 μg/mouse) (Wislocki et al. 1986). There was a statistically significant increase in the incidence of liver adenomas or carcinomas (31/39 in treated versus 2/28 in controls); however, female mice did not develop liver tumors. There was an increase in pulmonary adenomas or carcinomas in males (not statistically significant), although the incidence in female treated mice was increased significantly (6/32 in treated versus 0/31 in controls).

Subcutaneous injection of BaA in tricaprylin into C57B1 mice produced injection site sarcomas 9 months following treatment. The doses were 0.05, 0.2, 1.0, 5.0, or 10 mg, and the corresponding incidence of tumor formation was 12 percent, 26 percent, 48

percent, 34 percent, and 31 percent, respectively. A statistical analysis was not reported (USEPA 2001).

Intramuscular injection of BaA in combination with 1 or 3 percent croton oil produced injection site fibrosarcomas and hemangioendotheliomas in Strain A-derived albino mice. Of the mice injected with BaA in 1 percent croton oil, 3/24 developed tumors, and in the 3 percent croton oil administration, 1/26 developed tumors.

No studies were found that investigated the carcinogenicity of BaA via inhalation.

Mutagenicity

BaA was mutagenic to *S. Typhimurium*, *Drosophila melangaster*, and mammalian cell *in vitro* in the presence of an exogenous metabolic system. There was some evidence of clastogenic activity, as BaA yielded mostly positive results for DNA damage, mutations, chromosomal effects, and cell transformation in a variety of eukaryotic cells (USEPA 2001).

Systemic Toxicity

No studies were located that investigated the systemic toxicity of BaA in either humans or animals.

Toxicokinetics

No studies have been located on the absorption of BaA in isolation, although BaA undergoes intestinal transport by passive diffusion (Rees et al. 1971). A discussion of the toxicokinetics of PAHs as a class of compounds is presented in the polycyclic aromatic harbon section above.

Metabolism

BaA is similar to other polynuclear aromatic hydrocarbons that have a "bay-region" structure; it is metabolized by the mixed function oxidases to reactive bay-region diol epoxides (i.e., the sterically hindered cup-shaped area between carbons 1 and 12 of benzo(a)anthracene) intermediates of diol epoxide are currently considered to be the ultimate carcinogen for alternant PAHs (Jerina et al. 1980). These diol epoxides are easily converted into carbonium ions, which are alkylating agents. BaA is metabolized to all five of its dihydrodiols and a number of phenolic metabolites and conjugates. The 3,4-dihydrodiol is mutagenic in the presence of rat liver microsomal fraction, and the 3,4-dihydrodiol and 3,4-diol-1,2-epoxide are also highly tumorigenic.

Toxicity Values

EPA (USEPA 2001) has performed no quantitative estimates of risk for human exposure to benzo(a)anthracene. To derive a CPF for BaA, a relative potency factor of 0.1

(USEPA 1993) was applied to the benzo(a)pyrene slope factor to obtain an oral SF of 7.3 $\times 10^{-1}$ (mg/kg-day)$^{-1}$ for BaA. The NCEA derived an inhalation slope factor for benzo(a)pyrene of 3.1 (mg/kg/day)$^{-1}$, which has been modified by the same relative potency factor of 0.1 to 0.31 (mg/kg/day)$^{-1}$.

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BENZENE

Benzene (C_6H_6, CAS Registry Number 71-43-2) is a volatile, colorless, liquid aromatic hydrocarbon with a characteristic odor. Environmental benzene is primarily created anthropogenically, although a small amount arises from natural events (i.e., forest fires and volcanoes). Benzene is obtained primarily from petroleum, with coal being a minor secondary source. As a result of its anti-knock characteristics, it is an important component of unleaded gasoline. Benzene is also used commercially as a solvent, in the manufacture of plastics, and as an intermediate in the manufacture of a wide range of chemical products. Due to its adverse health effects in humans, however, its use as a solvent has been greatly reduced.

Carcinogenicity

Benzene has been classified as a known human carcinogen (category A) by EPA, based on several occupational epidemiology studies showing an increased incidence of nonlymphocytic leukemia associated with high workplace exposures (USEPA 2001). Supporting evidence comes from rodent studies in which benzene induced tumors in multiple species at multiple sites when administered either by inhalation or by gavage (USEPA 2001). While some studies have implicated other types of leukemia and possibly lymphoma with exposure to high concentrations of benzene, the evidence is strongest and most consistent for acute myeloid leukemia (AML) and variants of that condition (Goldstein 1988).

Mutagenicity

Benzene is not mutagenic in *in vitro* studies in bacterial (*Salmonella* and yeast) or mammalian (mouse lymphoma cells) bioassays (USEPA 2001). Administered *in vivo*, however, benzene induces chromosomal aberrations in bone marrow cells of rabbits (Kissling and Speck 1973), mice (Meyne and Legator 1980), and rats (Anderson and Richardson 1979). Several investigators have also reported positive results for benzene in the mouse micronucleus assay at high doses (ATSDR 1991). Workers exposed occupationally to high doses of benzene have shown increases in the number of chromosomal aberrations in bone marrow cells and in peripheral lymphocytes (IARC 1982).

Systemic Toxicity

The major systemic effect of high-dose, chronic exposure to benzene is hematotoxicity. Benzene exerts its toxic effects in the bone marrow, producing deficiencies of blood cells such as pancytopenia (significant decreases in all major types of blood cells, C erythrocytes, leukocytes and platelets) and aplastic anemia (which can progress to acute nonlymphocytic leukemia). Deficiencies in various types of blood cells lead to other disorders such as hemorrhagic conditions from a lack of platelets, susceptibility to infection from lack of leukocytes, and increased cardiac output from lack of erythrocytes.

Neurological abnormalities have been observed in humans with previous chronic occupational exposure to benzene (air concentrations reached 210 ppm or higher) (Baslo and Aksoy 1982). The abnormalities included distal nephropathy in patients with pre-existing aplastic anemia.

Animal responses to benzene exposure vary and may depend on factors such as species, strain, duration of exposure, and pattern of exposure (e.g., intermittent versus continuous).
Relatively high concentrations of benzene may induce fetal growth retardation or an increase in skeletal abnormalities when pregnant mice and rats are exposed via inhalation (ATSDR 1991). Fetotoxicity in animals may be a function of maternal toxicity (Tatrai et al. 1980).

Toxicokinetics

Benzene is readily absorbed through the lungs and about 30 to 50 percent is excreted unchanged in exhaled air. The 40 to 50 percent that is retained is taken up preferentially by fatty and nervous tissues, broken down into metabolites by the liver, and excreted in urine (IARC 1974). In addition, significant amounts of benzene may enter the body through the skin. When ingested, it is absorbed through the gastrointestinal tract.

Metabolism

The toxicokinetics of benzene suggest that its metabolism is complex and that toxicity and carcinogenicity may occur only after it has been biotransformed to possibly synergistic reactive metabolites (Stevens et al. 1994, Medinski et al. 1996, Lee et al. 1996, Rothman 1997). Benzene is broken down into its major metabolites (phenol, hydroquinone, and catechol) by cytochrome P4502E1 in the liver and excreted in the urine via glucuronide or sulfate conjugation (Henderson et al. 1989). It appears that catechol and phenol are formed by two distinctly different metabolic pathways. Hydroquinone is thought to result from a second passage of phenol through the mixed function oxidases.

Other putative toxic metabolites include (1) benzoquinone, resulting from the oxidation of hydroquinone (formed by the oxidation of phenol), and (2) muconic dialdehyde (also referred to as *trans, trans*-muconaldehyde), resulting from ring opening of either benzene epoxide or a nonreactive metabolic intermediate, benzene glycol, in the presence of NADPH. Muconic dialdehyde is a known hematotoxin (Latriano et al. 1986), and the percent of this metabolite formed *in vivo* increases nonlinearly with increasing dose.

Biotransformation of benzene is essential for benzene-induced bone marrow damage. There is disagreement as to whether benzene is activated in the marrow or activated elsewhere and transported to the marrow. When several of the metabolites of benzene were compared with the parent compound in hematotoxicity studies, the metabolites were less toxic than benzene. Tunek et al. (1981) suggested that perhaps the metabolites tested

may conjugate strongly in the liver and possibly other organs, and may not reach the bone marrow in amounts sufficient to produce the expected hematotoxicity.

Toxicity Values

An oral slope factor ranging from 1.5×10^{-2} to 5.5×10^{-2} per (mg/kg)/day has been extrapolated by USEPA (USEPA 2001) from the results of human occupational studies of workers who showed an increased incidence of leukemia after inhalation exposure. Expected differences in absorption by the two exposure routes were taken into account.

EPA estimates that an air unit risk ranging from 2.2×10^{-6} to 7.8×10^{-6} is the increase in risk to an individual exposed for a lifetime to $1 \mu g/m^3$ benzene in air (USEPA 2001).

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BENZO(b)FLUORANTHENE

Benzo(b)fluoranthene ($C_{20}H_{12}$, MW 252.3, CAS registry number 205-99-2) is a polynuclear aromatic hydrocarbon that exists in pure form as colorless needles. Benzo(b)fluoranthene (BbF) is virtually insoluble in water, and slightly soluble in organic media. BbF occurs in fossil fuels, and is formed by the incomplete combustion of fossil fuels. It has also been identified in cigarette smoke, engine exhaust, broiled and smoked food, edible oils and margarines, and various water bodies.

Cancer

The carcinogenicity of BbF has not been classified by EPA (USEPA 2001). Epidemiologic studies have demonstrated increased mortality due to lung cancer in humans exposed via inhalation to coke-oven emissions (Lloyd 1971; Mazumdar et al. 1975; Redmond et al. 1976), roofing-tar emissions (Hammond et al. 1976), and cigarette smoke (McLure and MacMahon 1980). Reports of skin tumors among individuals exposed to mixtures containing BbF have been documented. The earliest of these is the report by Pott (1775) of scrotal cancer among chimney sweeps. More recently, skin cancer among humans dermally exposed to shale oils has been reported (Purde and Etlin 1980). Each of these mixtures contains a number of PAHs, including benzo(a)pyrene, benzo(b)fluoranthene, chrysene, benzo(a)anthracene, benzo(k)fluoranthene, and dibenzo(a,h)anthracene, as well as other potentially carcinogenic PAHs and other potentially carcinogenic chemicals, including nitrosamines, coal tar pitch, and creosote. It is impossible to evaluate the contribution of any individual PAH to the total carcinogenicity of these mixtures in humans because of the complexity of the mixtures and the presence of other carcinogens. Therefore, epidemiologic evidence in humans regarding the potential carcinogenicity of BbF is inadequate.

A dose-response relationship for the dermal carcinogenicity of BbF has been demonstrated over an order-of-magnitude dose range in Swiss mice receiving daily topical doses throughout their lifetime (Wynder and Hoffmann 1959b). Survival was also dose-dependent. The time-to-tumor latency was short, with skin papillomas and carcinomas appearing in high dose groups after 5 months of treatment.

Mutagenicity

A single topical application of 100 μg BbF was reported to bind to DNA in mouse skin (Weyand et al. 1987). The relative extent of binding for carcinogenic PAHs was benzo(b)fluoranthene > benzo(k)fluoranthene > indeno(1,2,3-cd)pyrene (Weyand et al. 1987), which corresponds to the relative cancer potency of these PAHs.

Systemic Toxicity

No studies were located that assessed the systemic toxicity of BbF.

Toxicokinetics

PAHs have been divided into two groups, based on structural similarity and likely metabolic/detoxification pathways. These two groups have been termed "alternant" and "non-alternant" and differ in the electron density associated with the molecule. Alternant PAHs have an equally distributed electron density, whereas non-alternant PAHs have an uneven distribution of electron density from one portion of the molecule to the other and behave almost as if they were two different molecules. Structurally, alternant PAHs consist of multiple 6-membered rings, whereas non-alternant PAHs consist of several 6-membered rings, and one 5-membered ring to which many of the 6-membered rings are attached. It is the 5-membered ring that contributes substantially to the uneven distribution of electron density. Generally, alternant PAHs are more potent than non-alternant PAHs (ATSDR 1990b), and the mechanisms of carcinogenicity appear to be different between the two groups. BbF is a non-alternant PAH.

Metabolism

The mechanism of carcinogenicity of non-alternant PAHs is unclear, because non-alternant PAHs either do not have a bay region or have been shown not to be activated via a bay-region epoxide, as are alternant PAHs (Amin et al. 1985a,b). Studies of BbF metabolism indicate the major metabolites of this non-alternant PAH are 6-hydroxybenzo(b)fluoranthene and 4- or 7-hydroxybenzo(b)fluoranthene; the predominant diol formed was trans-11,12-dihydro-11,12-dihydroxybenzo(b)-fluoranthene (Amin et al. 1982, 1985b). There was no evidence to suggest that a diol epoxide is the ultimate carcinogenic metabolite of non-alternant PAHs. If non-alternants differ in their mechanism of action, then they may also differ with regard to other factors, including target organ and species sensitivity.

Toxicity Values

EPA (USEPA 2001) has not calculated quantitative risk estimates for human exposure to BbF. To derive a cancer potency factor for BbF, a relative potency factor of 0.1 (USEPA 1993) is applied to the BaP cancer potency factor to obtain an oral CPF for BbF of 7.3×10^{-1} $(mg/kg\text{-}day)^{-1}$. The NCEA derived an inhalation slope factor for benzo(a)pyrene of 3.1 $(mg/kg/day)^{-1}$, which has been modified by the same relative potency factor of 0.1 to 0.31 $(mg/kg/day)^{-1}$.

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BENZO(a)PYRENE

Aside from one study, reports of subchronic and chronic systemic toxicity of benzo(a)pyrene (C_{20}-H_{12}, CAS Registry No. 50-32-8) by any route have not been found (ATSDR, 1995). Workers in a rubber factory with measured exposures to benzo(a)pyrene and total suspended particulate matter were monitored for respiratory health (Gupta et al., 1993). Long-term employees had statistically significant loss of ventilatory function; those with highest exposures exhibited patch opacities in the lungs, prominent bronchiovascular markings, and pleural effusions, along with bloody vomit, breathing problems, chest pains, lung and throat irritation, and cough. Workers in other parts of the plant were similarly affected to a lesser degree. No attempt was made to separate the effects of exposure to benzo(a)pyrene and particulates, or to identify other potentially toxic air pollutants at the plant. Other human occupational studies (Assenato et al., 1993; Santella et al., 1993) seeking markers of BaP exposure experienced similar problems of confounding and revealed trends, but could not establish statistically significant effects. Wolff et al. (1989) tested 40 male and 40 female Fischer-344 Crl rats with nose-only exposures to a 7.7-mg/m^3 aerosol of benzo(a)pyrene 2 hrs/day, 5 days/week for 4 weeks, and found no treatment-related lesions in the lungs or nasal cavities; but no dose-response relationship could be established with this single-level study, and the rest of the respiratory tract was not examined. A few other research studies have explored some systemic pathologic endpoints in a few animals exposed either by ingestion or inhalation (Nousiainen et al., 1984; Robinson et al., 1975; Thyssen et al., 1981). However, the findings of these investigations are also judged inadequate to estimate the risks of this compound for exposed humans.

The carcinogenicity of benzo(a)pyrene has been classified as Category B2 by USEPA (2001). The effects of this chemical by skin contact, ingestion, and inhalation have been investigated. Epidemiology studies have demonstrated increased mortality due to lung cancer in humans exposed via inhalation to coke-oven emissions (Lloyd, 1971; Mazumdar et al., 1975; Redmond et al., 1976), roofing tar emissions (Hammond et al., 1976), and cigarette smoke (McLure and MacMahon, 1980). Reports of skin tumors among individuals exposed to mixtures containing PAHs have also been documented (e.g., Purde and Etlin, 1980). Each of these mixtures contained a number of PAHs including benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)anthracene, dibenz(a,h)anthracene, as well as other potentially carcinogenic chemicals including nitrosamines, coal tar pitch, and creosote. Evaluating the contribution of any individual PAH to the total carcinogenicity of these mixtures in humans is not possible, because of the complexity of the mixtures and the presence of other carcinogens. Thus, epidemiologic evidence in humans regarding the potential carcinogenicity of benzo(a)pyrene alone is inadequate.

Experimental studies in laboratory animals have shown that repeated administration of benzo(a)pyrene by any route is associated with an increased incidence of tumors. It is tumorigenic in animals via dietary administration, gavage, inhalation, intratracheal instillation, and dermal and subcutaneous applications (USEPA 2001). Mice administered daily dietary doses of up to 1000 ppm benzo(a)pyrene for 38 to 238 days

exhibited an increased incidence of stomach tumors (Rigdon and Neal, 1966). Benzo(a)pyrene induces skin tumors in mice at the point of contact with repeated dermal application (Shubik and della Porta, 1957). An inhalation study by Thyssen et al. (1981) showed that repeated exposure to benzo(a)pyrene particles induced respiratory tract tumors in Syrian golden hamsters. Benzo(a)pyrene is mutagenic in the Ames assay and in prokaryote and mammalian cell culture tests for DNA damage (USEPA 2001), providing supporting data for its possible mechanism of action as an initiator of tumorigenesis.

Benzo(a)pyrene appears to be a developmental and reproductive toxicant when administered at high doses by ingestion to pregnant mice (MacKenzie and Angevine, 1981; Rigdon and Neal, 1965) and rats (Rigdon and Rennels, 1964), although results are inconsistent among studies. In addition, the findings may be of questionable relevance to humans, since the doses administered appear to have induced maternal toxicity (MacKenzie and Angevine, 1981; Shevaleva, 1978; Rigdon and Neal, 1965).

Among the carcinogenic PAHs, benzo(a)pyrene has been the most widely studied, and is considered by some to be the most potent, although sometimes dibenz(a,h)anthracene is also acknowledged as being of similar potency. Therefore, the toxicity of benzo(a)pyrene was chosen as the relative standard against which the carcinogenic potential of other PAHs in this study were considered, and then applied to the quantitative assessment of their risk.

Toxicity Values

No toxicity values for health effects other than cancer have been derived for benzo(a)pyrene (USEPA 2001).

Carcinogenic PAHs appear to exert their effects mainly at the point of contact (dermal application results in skin tumors), or portal of entry (ingestion results mainly in forestomach tumors. Inhalation results in respiratory tract tumors and tumors of the upper digestive tract presumably due to mucociliary particle clearance and involuntary ingestion of particles). Tumors distant from the point of application have also been observed. Among the PAHs, benzo(a)pyrene (BaP) has the ability to elicit cancer, and it is the most potent carcinogen.

For ingestion of BaP, a SF of 7.3 $(mg/kg\text{-}day)^{-1}$ was estimated by USEPA (2001). This SF is the geometric mean of four slopes (ranging from 4.5 to 11.7 $(mg/kg\text{-}day)^{-1}$) derived by different modeling procedures from the studies of Neal and Rigdon (1967), Rabstein et al. (1973), and Brune et al. (1981).

Few studies have evaluated the carcinogenic effects of inhalation exposure to BaP. Rats exposed chronically to combustion gases of a coal-burning furnace enriched with BaP developed lung tumors. However, these gases contained other PAHs, as well as other potentially carcinogenic compounds, so that a direct association between BaP exposure and lung tumors cannot be made (Heinrich et al., 1986).

A provisional inhalation toxicity value for benzo(a)pyrene has been developed by USEPA National Center for Environmental Assessment, based on a hamster inhalation study. The inhalation slope factor is 3.1 $(mg/kg\text{-}day)^{-1}$ and the inhalation unit risk is 0.88 $(mg/m^3)^{-1}$.

As per USEPA (1989) guidance, it is not recommended to derive a dermal SF from oral data for the carcinogenic PAHs.

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BIS(2-ETHYLHEXYL)PHTHALATE

Potential targets of bis(2-ethylhexyl) phthalate [di(2-ethylhexyl)phthalate, or DEHP] toxicity include the liver (altered morphology, physiology, and biochemistry), gastrointestinal tract, cardiac, hematological, renal, and reproductive systems. With oral administration of di(2-ethylhexyl)phthalate for subchronic exposure durations of 2 weeks to 1 year, animal LOAELs ranging from 50 to 2000 mg/kg/day have been observed; the lowest reported animal NOAEL is 5 mg/kg/day for liver toxicity (ATSDR 1993). With chronic oral exposures, typical animal LOAELs and NOAELs range from 10 to 2000 mg/kg/day, depending on the species, the route of administration, target organ toxicity, and duration of exposure (ATSDR 1991). Phthalate esters are reported to induce hepatic peroxisome proliferation in rodents, although the relevance of these cellular changes to target organ disease or injury is not certain.

EPA considers di(2-ethylhexyl)phthalate to be a probable human carcinogen, based on the results of an NTP (1982) cancer bioassay. Male and female rats and mice were exposed to di(2-ethylhexyl)phthalate in their diet for 2 years. No clinical signs of toxicity were observed in either rats or mice. However, a statistically significant increase in the incidence of hepatocellular carcinomas and in the combined incidences of hepatocellular carcinomas and adenomas was observed in female rats and both sexes of mice.

Toxicity values EPA (USEPA 1999) has established a oral chronic RfD for di(2-ethylhexyl)phthalate of 0.02 mg/kg/day, based on the finding of increased relative liver weights in guinea pigs exposed to di(2-ethylhexyl)phthalate in the diet for one year (Carpenter *et al.* 1953). The LOAEL in this study was 19 mg/kg/day (equivalent to 0.04% of the diet), and uncertainty factors totaling 1000 (species-to-species extrapolation (10-fold), variability among human subpopulations (10-fold), and the use of subchronic data to derive a chronic toxicity value (10-fold) were applied. The subchronic oral RfD is 0.2 mg/kg/day. Dermal RfDs were estimated using the default oral absorption factor of 0.5 for semi-volatile organic compounds (USEPA Region IV 1996). EPA has not derived inhalation RfCs for DEHP.

EPA (USEPA 1999) has calculated an oral SF for bis(2-ethylhexyl)phthalate of 1.4×10^{-2} $(mg/kg\text{-}d)^{-1}$. The oral SF is based on the increased incidence of combined hepatocellular carcinoma and adenoma in male mice from the NTP (1982) feeding study, using standard food consumption rates for rodents. The dermal SF is derived from the oral SF using a default oral absorption efficiency factor of 0.5 for semivolatile organic compounds (EPA Region IV 1996). No inhalation SF has been determined for DEHP by EPA.

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CHLOROFORM

Chloroform ($CHCl_3$; CAS Registry No. 67-66-3) is a colorless, heavy liquid with a pleasant, non-irritating, etheric odor. It has been used in the past in the manufacture of the refrigerant HCFC-22 (monochlorodifluoromethane); as a raw material for polytetrafluoroethylene plastics; as a solvent for fats, oils, alkaloids, and waxes; as a cleansing agent; as an insecticidal fumigant for stored grain; as an ingredient in toothpaste, cough syrup, linaments; in human and veterinary medicine; and in the rubber industry (HSDB 2000).

Cancer

Chloroform is classified as a probable human carcinogen (Category B2), based on increased incidence of several tumor types in rats and three strains of mice, but inadequate human data (EPA 2001).

Mutagenicity

The majority of tests for genotoxicity of chloroform (including covalent binding to DNA, mutation in Salmonella, a Drosophila sex-linked recessive test, tests for DNA damage, a micronucleus test, and transformation of BHK cells) have been negative; however, it did cause mitotic recombination in Saccharomyces (Callen et al., 1980) and sister chromatid exchange in cultured human lymphocytes and in mouse bone marrow cells exposed in vivo (Morimoto and Koizumi 1983) (EPA 2001).

Systemic Toxicity

Adverse effects resulting from chloroform ingestion have been widely studied. Effects of subchronic and chronic exposure to chloroform range from mortality through various systemic (respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, and renal systems), immunological, neurological, developmental, reproductive, and carcinogenic effects (ATSDR 1993). Typical animal LOAELs and NOAELs for these varied systems range from 30 to 600 mg/kg-day chloroform for subchronic and chronic exposure durations (ATSDR 1993). Cancer in laboratory animals is reported from various studies to occur with ingestion of approximately 60 to 200 mg/kg-day chloroform for 78 to 180 weeks (ATSDR 1993).

Chloroform was first recognized for its ability to depress central nervous system function, hence its use as a general anesthetic (Simpson 1847). Much as with oral exposure, inhalation exposure to chloroform may bring about death, systemic effects (respiratory, gastrointestinal, hematological, hepatic, renal), neurological effects, and cancer. Human and laboratory animal LOAELs and NOAELs for systemic or neurological toxicity occur largely in the range of 10 to 100 ppm exposures (ATSDR 1993).

Neither ATSDR (1993) nor EPA (USEPA 2001) have reviewed, in their toxicological assessments of chloroform, studies relating subchronic or chronic dermal exposure to

chloroform with adverse health effects in rodents or humans. Acute exposure NOAELs for hepatic, dermal, or renal effects in rabbits are observed with topical application of solutions containing 1000 to 4000 mg chloroform per kilogram body weight (ATSDR 1993).

Toxicity Values

EPA has established an oral RfD of 0.01 mg chloroform ingested per kilogram body weight per day, based on formation of fatty cysts in the liver of chronically-exposed dogs (Heywood et al. 1979) (USEPA 2001). This RfD is based upon a LOAEL of 15 mg/kg-day, adjusted to 12.9 mg/kg-day to reflect a 7-day per week dosing schedule, and an aggregate uncertainty factor of 1000. The uncertainty factor incorporates use of a LOAEL, interspecies conversion, and human sensitivity (10-fold each). No inhalation RfC has been derived for chloroform.

EPA has calculated an oral slope factor (a plausible upper-bound estimate of the probability of a carcinogenic response per unit intake of the chemical over a lifetime) for chloroform of 0.0061 $(mg/kg\text{-}day)^{-1}$ (USEPA 2001). The slope factor calculation was based on kidney tumors in male Osborne-Mendel rats administered chloroform via drinking water (Jorgenson et al. 1982).

EPA has calculated an inhalation unit risk concentration (risk per unit concentration of chloroform in the air) of 2.3×10^{-5} $(\mu g/m^3)^{-1}$, which is the geometric mean of separate male and female slope factors (USEPA 2001). The critical study (NCI 1976) for this derivation reported hepatocellular carcinoma in female $B6C3F_1$ mice and kidney tumors in male rats administered chloroform by gavage. Studies by Roe et al. (1979) are generally supportive of the risk estimate.

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COPPER

Copper (Cu; CAS Registry No. 7440-50-8) is a ubiquitous, nutritionally essential trace element required for hemoglobin synthesis and for varied oxidative enzymes; copper also has various industrial uses.

Cancer

At present, EPA considers copper to be unclassifiable as a human carcinogen (Category D), based on an absence of human carcinogenicity data, inadequate evidence of carcinogenicity in animals, and equivocal mutagenicity data (USEPA 2001).

EPA (USEPA 2001) summarized the inadequacy of the animal carcinogenicity data, as follows:

Bionetics Research Labs (1968) studied the carcinogenicity of a copper-containing compound, copper hydroxyquinoline, in two strains of mice (B6C3F1 and B6AKF1). Groups of 18 male and 18 female 7-day-old mice were administered 1,000 mg/kg copper hydroxyquinoline (180.6 mg Cu/kg) suspended in 0.5 percent gelatin daily until they were 28 days old, after which they were administered 2,800 ppm (505.6 ppm Cu) in the feed for 50 additional weeks. No statistically significant increases in tumor incidences were observed in the treated 78-week-old animals.

In the same study, Bionetics Research Labs (1968) administered a single subcutaneous injection of gelatin (control) or 1,000 mg/kg of copper hydroxyquinoline (180.6 mg Cu/kg) suspended in 0.5 percent gelatin to groups of 28-day-old mice of both strains. After 50 days of observation, the male $B6C3F_1$ had an increased incidence of reticulum cell sarcomas compared with controls. No tumors were observed in the treated male $B6AKF_1$ mice, and a low incidence of reticulum cell sarcomas was observed in the treated female mice of both strains.

Gilman (1962) administered intramuscular injections containing 20 mg of cupric oxide (16 mg Cu), cupric sulfide (13.3 mg Cu), and cuprous sulfide (16 mg Cu) into the left and right thighs of 2- to 3-month-old Wistar rats. After 20 months of observations, no injection-site tumors were observed in any animals, but other tumors were observed at very low incidence in the animals receiving cupric sulfide (2/30) and cuprous sulfide (1/30). As the relevance of the organic copper compound to the observation of sarcoma induction is uncertain and the incidence of tumors in rats treated *i.m.* with inorganic copper was very low, data are considered inadequate for classification.

Mutagenicity

Moriya et al. (1983) reported no increase in mutations in *E. coli* and *S. typhimurium* strains TA98, TA1535, TA1537 and TA1538 incubated with up to 5 mg copper quinolinolate per plate and in *S. typhimurium* TA98 and TA100 incubated with up to 5 mg copper sulfate per plate. Demerec et al. (1951) reported dose-related mutagenic effects in *E. coli* with 2-10 ppm copper sulfate in a reverse mutation assay. Negative results were obtained with copper sulfate or copper chloride in assays using *S. cerevisiae* (Singh 1983) and *Bacillus subtilis* (Nishioka 1975, Matsui 1980, Kanematsu et al. 1980). Errors in DNA synthesis from poly(c)templates have been induced in viruses incubated with copper chloride or copper acetate (Sirover and Loeb 1976). Chromosomal aberrations were induced in isolated rat hepatocytes when incubated with copper sulfate (Sina et al. 1983). Casto et al. (1979) showed enhanced cell transformation in Syrian hamster embryo cells infected with simian adenovirus with the addition of cuprous sulfide and copper sulfate. High concentrations of copper compounds have been reported to induce mitosis in rat ascites cells and recessive lethals in *Drosophila melanogaster*. Law (1938) reported increases in the percent lethals observed in *Drosophila* larvae and eggs when exposed to copper by microinjection (0.1 percent copper sulfate) or immersion (concentrated aqueous copper sulfate), respectively.

Systemic Toxicity

Because of its role as a nutritionally essential element, copper is subject to homeostatic mechanisms that attempt to maintain a physiologically balanced supply of the nutrient. Thus, altered copper status can create problems of both deficiency and excess. Copper deficiency is primarily a topic for nutritionists, and only copper excess (toxicity) will be considered here. An additional consideration is the chemical speciation of the nutrient. Generally, the element must be in a biologically available form (e.g., a soluble salt) to exert toxic effects; insoluble forms usually preclude any significant absorption into an organism.

As a dietary requirement, oral exposure to copper has been well studied in animals and humans. Toxic effects can be noted in a variety of organ systems with excess ingestion of copper: cardiovascular, hematological, musculoskeletal, hepatic, renal, neurological, developmental, reproductive, as well as mortality (ATSDR 1989). In laboratory animals exposed to copper for subchronic or chronic durations, LOAELs and NOAELs for ingested copper typically range from 3 to 150 mg/kg-day (ATSDR 1989).

Inhalation exposure to copper aerosols or fumes for subchronic or chronic durations have been much less studied. Changes in alveolar type II cells have occurred in rabbits exposed to 0.6 mg Cu/m^3 for 6 hr/day, 5 day/wk, for 4 to 6 weeks (Johansson et al. 1984; Lundborg and Camner 1984). This value is reported as a NOAEL by ATSDR (1989).

Neither ATSDR (1989a) nor EPA (USEPA 2001) has reviewed, in their toxicological assessments of copper, studies relating subchronic or chronic dermal exposure to copper with adverse health effects in rodents or humans. A few acute effects are, however, reported by ATSDR (1989), including dermal irritation, and hematological and immunological effects.

Toxicity Values

A subchronic and chronic oral RfD for copper was reported as 1.3 mg/L (3.7×10^{-2} mg/kg-day), which is the current maximum contaminant level (MCL) standard for copper (USEPA 1991). EPA has not set an oral RfD (USEPA 2001).

The EPA weight-of-evidence classification for the carcinogenicity of copper is Category D (not classifiable as to human carcinogenicity), and no slope factors have been calculated (USEPA 2001).

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1,2-DICHLOROETHANE

1,2-Dichloroethane (CAS No. 107-06-2) is used primarily in the manufacture of vinyl chloride, as well as in the synthesis of tetrachloroethylene, trichloroethylene, 1,1,1-trichloroethane, vinylidene chloride, aziridines, and ethylenediamines (U.S. Air Force 1989, ATSDR 1992). It is added to gasoline as a lead-scavenging agent, and, in the past, has been used as a metal degreasing agent; a solvent; and a fumigant for grain, upholstery, and carpets. It has also been used in paints, coatings, adhesives, varnishes, finish removers, soaps, and scouring agents (U.S. Air Force 1989, ATSDR 1992).

Toxicokinetics

1,2-Dichloroethane is absorbed through the lungs, gastrointestinal system, and skin (ATSDR 1992). It is distributed throughout the body but may be concentrated in adipose tissue. The compound can also accumulate in breast milk (Urusova 1953) and may cross the placenta (Withey and Karpinski 1985, Vozovaya 1977). Metabolism of 1,2-dichloroethane most likely involves conjugation with glutathione (ATSDR 1992). Excretion occurs primarily through elimination of soluble urinary metabolites (Reitz et al. 1982, Spreafico et al. 1980).

Systemic Toxicity

Bronchitis, hemorrhagic gastritis and colitis, hepatocellular damage, renal tubular necrosis, central nervous system depression, and histopathological changes in the brain have been reported in cases of acute oral poisoning of humans (ATSDR 1992, NIOSH 1976). Animal data indicate that short-term exposures may produce immune system deficiencies (Munson et al. 1982), and subchronic or chronic oral exposures may affect the liver or kidney (NTP 1991, Alumot et al. 1976).

Acute inhalation exposures to 1,2-dichloroethane can result in irritation of the eyes, nose and throat, dizziness, nausea, vomiting, increasing stupor, cyanosis, rapid pulse, delirium, anesthesia, partial paralysis, loss of tactile sense, degenerative changes in the myocardium, abnormal EEG, liver and kidney damage, pulmonary edema, and hemorrhages throughout the body (NIOSH 1976, CEC 1986, ATSDR 1992, Nouchi et al. 1984). Short-term exposures to animals have resulted in central nervous system depression (inactivity or stupor, tremors, uncertain gait, narcosis); pulmonary congestion; renal tubular degeneration; fatty degeneration of the liver and, less commonly, necrosis and hemorrhage of the adrenal cortex; chronic splenitis; fatty infiltration of the myocardium; and immuno-deficiency (Spencer et al. 1951, Heppel et al. 1946, Storer et al. 1984, Sherwood et al. 1987). Chronic occupational exposure to 1,2-dichloroethane may result in central nervous systems effects including irritability, sleeplessness, and decreased heart rate; loss of appetite; nausea; vomiting; epigastric pain, as well as irritation of the mucous membranes; and liver and kidney impairment (NIOSH 1976). Subchronic or chronic inhalation exposures to animals resulted in pathological lesions in the kidney, liver, heart, lungs, and testes (Heppel et al. 1946, Spencer et al. 1951, Cheever et al. 1990).

Carcinogenicity

1,2-Dichloroethane is classified EPA weight of evidence group B2 as a probable human carcinogen, based on evidence for the induction of several types of tumors in rats and mice. Male rats treated by gavage with 1,2-dichloroethane exhibited increased incidences of fibromas of the subcutaneous tissue; hemangiosarcomas of the spleen, liver, pancreas, and adrenal gland; and squamous-cell carcinomas of the forestomach. Female rats treated by gavage developed mammary adenocarcinomas. Increased incidences of hepatocellular carcinomas and pulmonary adenomas were observed in male mice treated by gavage, and increased incidences of mammary adenocarcinomas, pulmonary adenocarcinomas, and endometrial polyps and sarcomas were observed in female mice (NCI 1978). Mice treated by topical application of 1,2-dichloroethane exhibited an increased incidence of lung papillomas (Van Duuren et al., 1979).

Toxicity Values

EPA has established an oral reference dose (RfD) of 0.03 mg/kg-day (USEPA 1993). An inhalation RfD of 0.0014 mg/kg-day has been determined.

EPA has established an oral cancer slope factor of 0.091 per mg/kg/day (USEPA 2001), based on hemangiosarcomas in rats (NCI 1978). An inhalation cancer slop factor of 0.0014 per mg/kg-day has been determined (USEPA 2001).

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DIBENZ(a,h)ANTHRACENE

Reports of the subchronic or chronic systemic toxicity of dibenz(a,h)anthracene by any route have not been found (ATSDR, 1995). A few research studies have explored some systemic pathologic endpoints in a few animals exposed either by ingestion or injection (ATSDR, 1995). However, the findings of these investigations are judged inadequate to estimate the risks of dibenz(a,h)anthracene for exposed humans.

Dibenz(a,h)anthracene induced skin tumors following intermediate or long-term dermal exposure (Platt et al., 1990; Van Duuren et al., 1967; Wynder and Hoffmann, 1959a). The carcinogenicity of this chemical has been classified as Category B2 by USEPA. The carcinogenicity of ingested dibenz(a,h)anthracene by single dose and repeated dose was investigated in one study (Berenblum and Haran, 1955); however, the findings are inconclusive because of methodologic limitations. Carcinogenicity occurred in two intermediate-duration oral studies (Biancifiori and Caschera, 1962; Snell and Stewart, 1963), but one used no controls and the other was confounded by the vehicle.

No studies investigating the carcinogenicity of inhaled dibenz(a,h)anthracene have been conducted.

Toxicity Values

No toxicity values for health effects other than cancer have been derived for dibenz(a,h)anthracene.

Insufficient data are available to derive a SF for dibenz(a,h)anthracene. Based on USEPA Region IV guidance (USEPA 1996a), the oral SF for benzo(a)pyrene was used as a surrogate because the cancer potency of benzo(a)pyrene was considered to be representative of the potential for dibenz(a,h)anthracene to cause cancer.

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DIBENZOFURAN

Dibenzofuran ($C_{12}H_8O$; CAS Registry No. 132-64-9) is a colorless-to-white crystalline solid (leaves or needles) with a weak, characteristic odor. It is a component of diesel fuel, tobacco smoke, and heat-transfer oils; an intermediate in the production of dyes, plastics, and antioxidants in plastics; and a carrier for dyeing and printing textiles. Human exposure occurs occupationally to those working around coal tar and creosote and to the general population through tobacco smoke, other combustion sources, and contaminated food and drinking water (HSDB 2000).

Cancer

EPA has placed dibenzofuran in cancer category D (not classifiable as to human carcinogenicity). No data on this compound alone exist for humans; all studies have been of exposures to complex mixtures, and the contribution of individual components to cancer mortality cannot be determined. No animal carcinogenicity data is currently available. (USEPA 2001).

Mutagenicity

In an Ames assay, dibenzofuran was not mutagenic with or without metabolic activation in several strains of *Salmonella typhimurium* (Schoeny 1982).

Systemic Toxicity

No information has been found.

Toxicokinetics

No information has been found.

Metabolism

No information has been found.

Toxicity Values

EPA has calculated no references doses or slope factors for dibenzofuran (USEPA 2001). EPA NCEA has provided a provisional RfD of 0.004 mg/kg/day that has been used for this document.

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1,4-DICHLOROBENZENE

1,4-Dichlorobenzene (CAS 106-46-7), also referred to as para-DCB, p-DCB, paracide, Paramoth®, Parazene®, PDB, and Santochlor®, has a benzene ring with two chlorine atoms attached at the 1 and 4 carbon atoms. It is a white crystalline chemical with a penetrating, camphoraceous odor. It has a formula of $C_6H_4C_{12}$, a molecular weight of 147.01, a melting point of 53.1C, and a vapor pressure of 64 torr at 20OC (Budavari et al. 1989). The odor threshold has been reported to be as low as 0.18 ppm (Amoore and Hautala 1983), and it does not occur naturally (ATSDR 1993). 1,4-Dichlorobenzene is a chemical used to control moths, molds, and mildew, and to deodorize restrooms and waste containers. It is the vapor that acts as a deodorizer or insect killer. Most people recognize the odor as the smell of mothballs, and can smell p-DCB in the air at very low levels. Most p-DCB in our environment comes from its use in moth repellent products and in toilet deodorizer blocks.

Noncarcinogenic Health Effects

A single gavage dose of 1000 mg/kg in rats and 1600 mg/kg in guinea pigs did not induce death; however, 4000 mg/kg in rats and 2800 mg/kg in guinea pigs resulted in 100% mortality (Hollingsworth et al. 1956). Oral LD_{50} values in adult rats were reported by Gaines and Linder (1986) to be 3900 and 3800 mg/kg for males and females, respectively. Administration of gradually increasing doses up to 770 mg/kg/day for 5 days produced high urinary excretion of porphyrin, coproporphyrin, uroporphyrin, porphobilinogen, and delta-aminolevulinic acid (ALA) (Rimington and Ziegler 1963). Liver concentrations of uroporphyrin and protoporphyrin were elevated, while concentrations of coproporphyrin were not. Rats gavaged with 250 mg/kg/day for 3 days had increased microsomal protein content and no change in the cytochrome P450 content. Several hepatic microsomal xenobiotic metabolic systems increased at low doses of 20 and 40 mg/kg/day. Protein droplet formation was noted in males but not females administered 1,4-dichlorobenzene by gavage with 7 daily doses of 120 or 300 mg/kg/day (Charbonneau et al. 1989). A single dose (500 mg/kg, gavage) of ^{14}C-1,4-dichlorobenzene produced a similar protein droplet formation, and the ^{14}C was reversibly associated with -2μ-globulin.

A pregnant woman (21 years old) consumed 1-2 blocks of 1,4-dichlorobenzene toilet air freshener per week throughout her pregnancy and developed severe hypochromic, microcytic anemia with excessive polychromasia, marginal nuclear hypersegmentation of the neutrophils, and a small number of red blood cells with Heinz bodies (Campbell and Davidson 1970). Her consumption of 1,4-dichlorobenzene ceased at about 38 weeks of gestation, and her hemoglobin concentrations increased.

Four 14-day gavage studies have reported deaths from 1,4-dichlorobenzene at the following doses: no deaths in male rats administered up to 1000 mg/kg/day, 80% (4/5) in female rats at 1000 mg/kg/day, 100% in male and female rats at 2000 mg/kg/day and higher, 70% in mice at 1000 mg/kg/day, and 100% in mice at 4000 mg/kg/day (NTP 1987). In 13-week gavage studies, mortality percentages were 0% for mice administered

900 mg/kg/day, 40% (8/20) for mice receiving 1500 mg/kg/day, and 85% (17/20) for rats dosed with 1500 mg/kg/day (NTP 1987).

In the NTP study (1987), F344/N rats were dosed with 0, 300, 600, 900, 1200, and 1500 mg/kg for 13 weeks, and $B6C3F_1$ mice were dosed with 0, 600, 900, 1200, and 1800 mg/kg for 13 weeks. The findings encompassed four systems: digestion, liver, blood, and kidney. Gastrointestinal irritation, described as epithelial necrosis and villar bridging of the mucosa of the small intestine, was observed in rats dosed with 1200 mg/kg/day. Decreased hematocrit levels, red blood cell counts, and hemoglobin concentrations were present in male rats dosed with 300 mg/kg/day. Mice dosed with 600 to 1800 mg/kg/day demonstrated 34 to 50% lower white cell counts. Rats displayed degeneration and necrosis of hepatocytes at 1200 and 1500 mg/kg/day. Serum cholesterol concentrations were elevated in the male rats receiving 600 mg/kg/day and in the female rats receiving 900 mg/kg/day. The serum triglycerides and protein concentrations were reduced in male rats dosed with 300 mg/kg/day. Both sexes of rats demonstrated increased urinary porphyrins at 1200 mg/kg/day. Male mice had increased serum cholesterol concentrations at doses of 900 mg/kg/day and serum protein and triglyercide concentrations at doses of 1500 mg/kg/day. Hepatocellular cytomegaly was present in mice administered 600 mg/kg/day. Hepatocellular degeneration was noted in male and female mice administered 600-1800 mg/kg/day. Male rats exhibited renal tubular degeneration, ranging from slight to severe at 300 mg/kg/day and moderate at the 600 mg/kg/day. Mice, however, did not exhibit renal effects at dose ranges of 600-1000 mg/kg/day or 80-900 mg/kg/day.
In another 13-week study, male rats dosed with 75-600 mg/kg/day were observed to have renal hyaline droplets in all males from the 150 mg/kg/day group and above (Bomhard et al. 1988). During a 13-week study (Carlson and Tardiff 1976), rats dosed with 20 and 40 mg/kg/day were observed to have increased O-ethyl-O-p-nitrophenyl phenylphosphorothionate detoxification and benzpyrene hydroxylase and azoreductase activities, suggesting hepatic enzyme induction. At the end of a 30-day recovery period, the benzpyrene hydrolase and azoreductase activities were still elevated. No effect on the hemoglobin or hematocrit concentrations of rats receiving 40 mg/kg/day was observed. Carlson (1977) reported increased liver porphyrins in rats dosed by gavage with 50 to 200 mg/kg/day for up to 120 days. Males may have been more sensitive than the females.

No respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, dermal, or ocular effects were noted after 2 years of gavaging at 300 and 600 mg/kg/day in female rats and male and female mice and at 150 and 300 mg/kg/day in male rats (NTP 1987). Increased incidences of alterations in cell size (cytomegaly and karyomegaly), hepatocellular degeneration, and individual cell necrosis were observed in mice gavaged with 300 and 600 mg/kg/day. Nephropathy, epithelial hyperplasia of renal pelvis, mineralization of the collection tubules in the renal medulla, and focal hyperplasia of renal tubular epithelium were noted in male rats receiving 150 and 300 mg/kg/day. Female rats gavaged with 300 and 600 mg/kg/day demonstrated an increased incidence of nephropathy and minimal hyperplasia of the renal pelvis or tubules.

Pregnant female rats gavaged with 500 or 1000 mg/kg/day on gestation days 6-15 presented fetuses with a dose-related incidence of an extra rib, fetal weight loss at 1000

mg/kg/day, and reduced maternal weight gain at 500 and 1000 mg/kg/day (Giavini et al. 1986). Another study dosing rats with 250, 500, 750, or 1000 mg/kg/day on gestation days 6-15 resulted in similar findings at 500 mg/kg/day. Additionally, the number of skeletal variations increased at 750 and 1000 mg/kg/day (Ruddick et al. 1983).

Information on the acute toxicity of 1,4-dichlorobenzene in humans or animals following inhalation exposure was not available.

A husband (60 years old) and wife were exposed to supposedly high concentrations of 1,4-dichlorobenzene in their house for a period of 3 to 4 months. Clinical signs included severe headaches, diarrhea, numbness, clumsiness, burning sensation in the man's legs, slurred speech, weight loss (50 pounds in 3 months for the man), and jaundice; both individuals died. The woman died within a year of the initial exposure, and the man died within a few months of his wife. Both had acute yellow atrophy of the liver (also known as massive hepatic necrosis or fulminant hepatitis). No additional information was provided regarding confounding factors (Cotter 1953).

A 69-year-old man exposed in his chair at home for approximately 3 weeks to 1,4-dichlorobenzene developed petechiae, purpura, and swelling of his hands and feet (Nalbandian and Pearce 1965). An indirect basophil degranulation test demonstrated a strong positive reaction suggesting a sensitivity to 1,4-dichlorobenzene. A 36-year-old woman using 1,4-dichlorobenzene as a moth killer in her home reported intense headaches. A 34-year-old woman who demonstrated the use of 1,4-dichlorobenzene in an enclosed booth in a department store complained of headaches, nausea, and vomiting (Cotter 1953).

Male rats, female guinea pigs, and one female rabbit exposed for 16 days to 173 ppm developed pulmonary interstitial edema and congestion and alveolar hemorrhage (Hollingsworth et al. 1956). After 12 weeks of exposure to 798 ppm, two rabbits displayed lung congestion and emphysema. Two rabbits, exposed for 12 weeks to 798 ppm, developed ocular effects described as reversible [nonspecific eye ground changes (changes in the fundus or back of the eye)]. Guinea pigs exposed to 341 ppm for a comparable duration exhibited focal necrosis, slight cirrhosis, and hepatocyte swelling and degeneration. Rats exposed to 96-341 ppm intermittently for 5-7 months had cloudy swelling and degeneration of hepatic parenchymal cells in the central zone of the liver and increased liver weights in the male and/or females above 96 ppm. No changes were observed at 96 ppm. Kidney weights in the male rats (not the females) increased slightly after 5-7 months of intermittent exposure to 158 or 341 ppm. These findings (Hollingsworth et al. 1956) must be viewed with the understanding that several inconsistent variables were employed in the study design, such as species, numbers, sex of the animals per species, observations on controls, and duration of exposure at each exposure level. During a two-generation study, adult rats exposed to 538 ppm (6 hours/day and 7 days/week for 10 weeks) exhibited tremors, ataxia, hyperactivity, decreased grooming behavior, and an unkempt appearance (Tyl and Neeper-Bradley 1989).

For 12-15 years on a weekly basis, a 53-year-old woman had been inhaling 1,4-dichlorobenzene crystals that were scattered on the carpet and furniture (Weller and Crellin 1953). Pulmonary findings included lung parenchyma distorted by fibrosis, a thickening of the alveolar walls, and a marked infiltration of lymphocytes, mononuclear phagocytes, some thickening of the muscular walls of the small arteries, and focal fibrous thickening of the pleura. The authors attributed these findings to the physical interaction of the crystals with the lung tissue rather than chemical toxicity.

Carcinogenicity

In a 2-year study, female rats and male and female mice were gavaged with 300 or 600 mg/kg/day and male rats were gavaged with 150 or 300 mg/kg/day (NTP 1987). Renal tubular adenocarcinomas in the male rats were increased in a dose related manner (controls, 2%; low dose, 6%; high dose, 14%). A marginal increase in mononuclear cell leukemia was also observed in all dose groups (controls, 10%; low dose, 14%; high dose, 22%). The male and female mice in the 600 mg/kg/day group had increased incidences of hepatocellular carcinomas [(male - controls, 28%; low dose, 22.5%; high dose, 64%) (female - controls, 10%; low dose, 10.4%; high dose, 38%)]. Hepatocellular adenomas were also noted in male mice from the 300 and 600 mg/kg/day groups (controls, 10%; low dose, 26.2%; high dose, 32%) and in female mice from the 600 mg/kg/day group (controls, 20%; low dose, 12.5%; high dose, 42%).
Hepatoblastomas (a rare form of hepatocellular carcinoma) were observed in 4/50 males of the 600 mg/kg/day group. Male mice exhibited an increase in thyroid gland follicular cell hyperplasia, and the female mice had a marginal positive trend in the incidence of follicular cell adenomas of the thyroid gland. The male mice in the 600 mg/kg/day group had an increased incidence of adrenal pheochromocytomas. Adrenal gland medullary hyperplasia and focal hyperplasia of the adrenal gland capsule incidences were higher in the male mice. Renal tumors occurring in only male rats may have been the result of an $_{2\mu}$-globulin increase. In this NTP study, the liver tumor incidence in female controls was higher than the historical control numbers. Additionally, no mutagenicity in microbial or mammalian systems was demonstrated. In both the male and female gavaged mice, hepatocellular degeneration with resultant initiation of tissue repair was present. These findings resulted in a speculation by NTP (1987) that 1,4-dichlorobenzene was acting as a tumor promotor for liver tumors in male and female mice.

No evidence of carcinogenicity was observed in rats exposed to 500 ppm for 76 weeks (Riley et al. 1980). However, organ toxicity may not have been achieved suggesting that a maximum tolerated dose was not obtained. In addition, the exposure length was less-than-lifetime.

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1,1-DICHLOROETHENE

1,1-Dichloroethene ($C_2H_2Cl_2$, CAS Registry Number 75-35-4), also known as dichloroethylene or vinylidene chloride, is an organic solvent used in chemical manufacturing of plastics, flame-retardant fabrics, and as a precursor to other chemical syntheses.

The biomedical effects of dichloroethene may be manifest as a multitude of effects under varied exposure route, concentration, and duration scenarios. Evidence reviewed by the EPA for inclusion into the IRIS database leads to the Agency's suggestion that the liver is the most sensitive target organ for 1,1-dichloroethylene toxicity, and that of typical laboratory study organisms, the rat is the most sensitive model for toxic effects (EPA, 1999). In ATSDR's toxicological profile of dichloroethene, the agency identifies a variety of studies reporting hematological, hepatic, renal, and reproductive effects in laboratory animals chronically-exposed to dichloroethene (ATSDR, 1989). In these studies, NOAELS and LOAELs for laboratory animals are all at oral exposures of 10 ppm dichloroethene. Other adverse biomedical effects are reported for 1,1-dichloroethylene; for example, 1,1-dichloroethylene has been shown to be fetotoxic, but not teratogenic to rodents after exposure in drinking water or by inhalation (Short *et al.*, 1977; Murray *et al.*, 1979). However, the EPA emphasizes hepatotoxicity in the Agency's derivation of oral reference concentrations (EPA, 1999).

Toxicity values EPA has established an oral reference dose (RfD) of 0.009 mg 1,1-dichloroethene ingested per kilogram of body weight per day, based on hepatic lesions in a rat chronic oral bioassay (Quast *et al.*, 1983). This RfD is based upon a LOAEL of 50 ppm [9 mg kg^{-1} d^{-1}] adjusted by application of an aggregate uncertainty factor of 1000. The uncertainty factor accounts for inter- and intra-species extrapolations (10-fold each) and for extrapolation from a LOAEL to a NOEAL (10-fold) (EPA 1999).

The critical study used by EPA for 1,1-dichloroethene toxicity reference dose estimation was a 2-year study by Quast et al. (1983) exposing rats and a 97-day study exposing beagle dogs to 1,1-dichloroethene in drinking water (EPA 1999). For the rat study, 48 male and 48 female Sprague-Dawley rats (Spartan substrain) were administered 50, 100, or 200 ppm 1,1-dichloroethene in the drinking water, with concurrent control groups of 80 animals per sex. Daily intake was estimated as 7, 10, or 20 mg 1,1-dichloroethene per kg body weight per day for males and 9, 14, or 30 mg 1,1-dichloroethene per kg body weight per day for females. Hepatic lesions were the sole endpoint where statistically significant dose-related changes were observed. There were no treatment-related effects on mortality, body or organ weight, clinical chemistry, urinalysis, hematology, or numbers of tumors. Hepatic lesions were detected consistently in all 1,1-dichloroethene-exposed females; lesions were detected at statistically significant amounts only in the 200 ppm exposure group for males, but a non-significant increase was also observed in the 100 ppm exposure-group. The lesions generally consisted of minimal mid-zonal hepatocellular fatty change and hepatocellular swelling.

In the companion beagle dog study, 4 male and 4 female dogs per treatment group were administered 6.25, 12.5, or 25 mg 1,1-dichloroethene in gelatin capsules. This 97-day exposure had no measured adverse effects.

1,1-Dichloroethene has been shown to be fetotoxic, but not teratogenic to rodents after exposure by inhalation (Short *et al.*, 1977; Murray *et al.*, 1979). However, EPA emphasizes hepatotoxicity in the agency's derivation of an oral reference concentration (USEPA 2001). Chronic inhalation studies in laboratory rodents indicate a variety of respiratory, hematological, hepatic, and renal effects, including carcinogenesis, at LOAELs and NOAELs of ≥10 ppm exposures (ATSDR, 1989). EPA has calculated an oral RfD of 0.009 mg/kg-day (USEPA 2001).

Studies that utilize a dermal route of chronic exposure were not located by EPA or by ATSDR in their reviews of the toxicity of 1,1-dichloroethene (ATSDR, 1989; USEPA, 2001).

1,1-Dichloroethene is classified by EPA as a Group C (possible human) carcinogen (USEPA 2001). This conclusion is based on positive results from one study using one mouse strain following inhalation exposure, as well as the compound's mutagenic potential, the alkylating capacity of a metabolite, and the structural relatedness to the known human carcinogen, vinyl chloride. An epidemiological study (Ott *et al.*, 1976), which found no carcinogenic effect associated with 1,1-dichloroethene exposure, was considered as inadequate to reach a conclusion regarding human carcinogenicity because of inadequate statistical power. The supporting animal evidence is based primarily upon one inhalation study (of a total of 11 inhalation studies) in which 1,1-dichloroethene was found to be a complete carcinogen (Maltoni *et al.*, 1985), although none of the inhalation studies were conducted for a rodent lifetime (all were ≤12 months). Of eighteen animal studies reporting information about the carcinogenic potential of 1,1-dichloroethene, EPA judged most as not designed for maximum sensitivity to detect carcinogenic effects.

Dichloroethene did not act as a complete carcinogen when applied topically or subcutaneously to Ha:ICR mice, but did serve as an initiator when followed by phorbol myristate acetate treatment (Van Duuren *et al.*, 1979).

EPA has calculated an oral slope factor (a plausible upper-bound estimate of the probability of a carcinogenic response per unit intake of the chemical over a lifetime) of 0.6 $(mg/kg\text{-}d)^{-1}$. The slope factor calculation was based on four gavage studies and one drinking water study (Maltoni *et al.*, 1985; Quast *et al.*, 1983; Humiston *et al.*, 1978; NTP, 1982; Ponomarkov and Tomatis, 1980). The greatest slope factor (0.6 per mg/kg-d) was based on male rat adrenal tumors from the NTP (1982) bioassay. EPA acknowledges that there were not statistically significant increases in tumor incidence in this study, but uses this value nonetheless, since the derived slope factor is within a factor of 2 of the slope factor based on data from the inhalation study of Maltoni *et al.* (1977, 1985).

EPA has calculated an inhalation unit-risk concentration (risk per unit concentration of dichloroethene in the air) of 5.0×10^{-5} $(:g/m^3)^{-1}$. The critical study for this derivation was Maltoni *et al.* (1985), in which both sexes of Swiss mice were exposed to 10 and 25 ppm (MTD) for 4-5 days/week for 12 months. Male mice developed a statistically significant increase in kidney adenocarcinoma, female mice had significantly increased mammary carcinomas, and both sexes had increased incidence of pulmonary adenomas. The dose-response relationships, however, were not consistent. Similar confounding dose-response relationships were observed in a second study by the same author.

Dichloroethene is mutagenic in *Salmonella typhimurium* assays, when included with metabolic activating microsomal enzymes (USEPA 1993), has effected positive results in conventional and host-mediated assays of *Saccharomyces cerevisiae* mitotic gene conversion (Bronzetti *et al.*, 1981), and alkylates DNA *in vivo* (McKenna *et al.*, 1977; Reitz *et al.*, 1980). Dichloroethene was not mutagenic in V79 cells exposed to vapor *in vitro* (Drevon and Kuroki, 1979), did not produce chromosomal aberrations in bone marrow cells of ICR mice given single or repeated intraperitoneal treatment *in vivo* (Cerna and Kypenova, 1977), and did not induce dominant lethal mutations in mice (Anderson *et al.*, 1977) or rats (Short *et al.*, 1977).

Dichloroethene is rapidly and almost completely absorbed via ingestion or inhalation, but because of its limited solubility, dichloroethene is not stored in body tissues (Haley and Berndt, 1987). Dichloroethene widely distributes to body tissues, and causes hepatic midzonal necrosis, damaged parenchyma, and thrombosis, but no fatty infiltration (Haley and Berndt, 1987). Dichloroethene seemingly accumulates preferentially in the kidney, liver, and lung (ATSDR, 1989). Elimination of dichloroethene and metabolites is similar, whether exposure was by oral or inhalation routes; elimination is rapid, with the bulk of absorbed dichloroethene being eliminated as urinary metabolites, and very little (<1% of the administered dose of the parent compound) is eliminated unchanged in the expired air at low levels of exposure (McKenna *et al.*, 1978).

Metabolism of dichloroethene has not been studied in humans, but has been widely investigated in laboratory rodents (summarized in ATSDR, 1989). Results from these studies indicate that dichlorethene undergoes biotransformations to a variety of metabolites, the first of which is probably the formation of dichloroethene epoxide. The primary biotransformation pathways for dichlorethene in the rats are suggested to involve conjugation with glutathione (with either the dichloroethene epoxide or following molecular rearrangement of the epoxide to chloroacetylchloride) and subsequent hydrolysis to monochloroacetic acid. Physiologically-based pharmacokinetic modeling has indicated that dichloroethene metabolism is sensitive to the absorption rate, because of the parent compound's low blood:air partitioning coefficient and because metabolism of dichloroethene is a saturable phenomena ((D'Souza and Anderson, 1988). Additionally, metabolism variability between rats and mice seems largely a product of differing rates of similar metabolic pathways (Jones and Hathway, 1978).

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1,2-DICHLOROETHENE MIXED ISOMERS

cis-1,2-Dichloroethene, or *cis*-1,2-DCE ($C_2H_2Cl_2$, CAS Registry No. 156-59-2), is a colorless, volatile liquid with a slightly acrid, ether-like odor. *trans*-1,2-DCE ($C_2H_2Cl_2$, CAS Registry No. 156-60-5) is also a light, colorless liquid with a sweetish odor. In commerce, the two isomers are often found combined as mixed 1,2-DCE, which is used in perfumes, dyes, gums and waxes, oils, lacquers, thermoplastics, phenols, and camphor as a direct solvent; as an intermediate for chlorinated compounds; and as a fermentation-retarding agent. Mixed 1,2-DCE has been used in the extraction process for caffeine, fat, and natural rubber; as a dry-cleaning solvent; in food packaging adhesives and germicidal fumigants; and as a veterinary and human anesthetic (ACGIH 1991-1992).

EPA has established an RfD of 0.01 mg/kg-day. EPA has not established an RfC for 1,2-DCE, and has given it the carcinogenicity classification D (not classifiable as to human carcinogenicity (USEPA 2001), based on a lack of human and animal data and generally negative results in mutagenicity tests.

The greater part of 1,2-DCE is excreted directly from the lungs after inhalation. Once absorbed, the remainder distributes rapidly to tissues based on their lipid content and individual tissue/blood partition coefficients, with highest concentrations appearing in the fat, brain, and blood. Peak blood levels occur soon after inhalation exposure ceases (Sullivan and Krieger 1992). In rats, both isomers (*cis*- and *trans*-) are transformed to the same metabolites, dichloroacetic acid and dichloroethanol (Anonymous 1984).

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DIELDRIN

Dieldrin (CAS No. 60-57-1) was commonly used as an insecticide. Pure dieldrin is a white powder. Dieldrin can slowly evaporate in the air (ATSDR 1993). Dieldrin is no longer used. From the 1950s until 1970, dieldrin was used extensively as insecticides on crops such as corn and cotton. The U.S. Department of Agriculture canceled all uses of dieldrin in 1970. In 1972, however, EPA approved dieldrin for killing termites. Use of aldrin and dieldrin to control termites continued until 1987. In 1987, the manufacturer voluntarily canceled the registration for use in controlling termites (ATSDR 1993).

Systemic Toxicity

Exposure to very high levels of dieldrin for a short time causes convulsions or kidney damage. One very young child died from drinking a solution containing a very high level of dieldrin. Exposure for a long time to somewhat lower levels of dieldrin also causes convulsions. Exposure to moderate levels of dieldrin for a long time also causes headaches, dizziness, irritability, vomiting, or uncontrollable muscle movements.

Animal studies show effects of dieldrin on the nervous system and on the kidneys similar to those seen in people. Results from animal studies also show additional effects. Animal studies show that dieldrin cause increases in liver enzymes and liver weight. Studies in animals give conflicting information about whether dieldrin causes birth defects. Some studies show that dieldrin may damage sperm. Studies in animals show that mice that eat dieldrin develop liver tumors.

Carcinogenicity

Dieldrin is classified with an EPA weight of evidence B2. Dieldrin is carcinogenic in mice when administered orally. In addition, dieldrin is structurally related to aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid, which produce tumors in rodents (USEPA 2001). At different dose levels the effects range from benign liver tumors, to hepatocarcinomas with transplantation confirmation, to pulmonary metastases.

Dieldrin causes chromosomal aberrations in mouse cells (Markaryan, 1966; Majumdar et al., 1976) and in human lymphoblastoid cells (Trepanier et al., 1977), forward mutation in Chinese hamster V79 cells (Ahmed et al., 1977), and unscheduled DNA synthesis in rat (Probst et al., 1981) and human cells (Rocchi et al., 1980).

Toxicity Values

EPA has established an oral and inhalation RfD for dieldrin of 0.00005 mg/kg-day (USEPA 2001), based on liver lesions in rats in a chronic study (Walker 1969). An uncertainty factor of 100 was incorporated in the calculations (10 for species extrapolation, and 10 for human sensitivity variations) (USEPA 2001).

EPA has established an oral and inhalation cancer slope factor for dieldrin of 16 per mg/kg/day (USEPA 2001), based on liver carcinoma in mice.

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alpha-BENZENEHEXACHLORIDE (*alpha*-HCH)

alpha-Benzenehexachloride, or *alpha*-hexachlorocyclohexane ($C_6H_6Cl_6$, CAS Registry No. 319-84-6) and the seven other isomers of BHC are separated by fractional crystallization from organic solvents. Formerly used as an insecticide, technical BHC (which contained several of the isomers) is no longer produced in the U.S., and only the *gamma* isomer (lindane) is allowed in commercial use here as a dust, powder, liquid, or concentrate for insecticides, and as an ingredient in lotions, creams, or shampoos to control head lice and scabies. Isomers of BHC have been found in the soil and water at hazardous waste sites, with the *gamma* isomer persisting in air for up to 17 weeks. Decomposition products include carbon monoxide and hydrochloric acid (HSDB, 1999).

Plant foods can be contaminated with BHC isomers that enter the body when eaten, or BHC can be inhaled from the air. The isomers and their byproducts can be temporarily stored in body fat and are excreted in the urine, feces, and exhaled air. Two harmful metabolites are pentachlorophenol and chlorinated hydrocarbon epoxide. In the past, pesticide manufacturing workers breathing toxic amounts of *delta*-BHC have experienced headaches, dizziness, blood disorders, and possible changes in the levels of sex hormones in the blood. Repeated high exposures can lead to seizures. All BHC isomers produce liver and kidney effects.

delta-BHC fed to male DD mice and Wistar rats at concentrations of 100-1000 ppm for 24 weeks did not produce noticeable neoplastic or nonneoplastic effects in the livers of a small number of animals (Ito et al., 1973a,b, 1975; Nagasaki et al., 1972). On the other hand, a 600-ppm mixture of *delta*- and *epsilon*-BHC in the diet of ICR-JCL mice for 26 weeks produced an increased incidence of benign and malignant hepatomas (Goto et al., 1972).

Toxicity Values

EPA has not established oral or inhalation RfDs for *alpha*-BHC. Although human cancer data is inadequate, *alpha*-BHC in the diet has caused increased incidences of tumors in 5 strains of mice and in Wistar rats (Ito et al., 1973a,b, 1976; Nagasaki et al., 1972, 1975; Hanada et al., 1973; Goto et al., 1972; Schulte-Hermann and Parzefall, 1981). EPA's cancer classification for alpha-BHC is thus B2 (probable human carcinogen). An oral slope factor of 6.3 $(mg/kg\text{-}d)^{-1}$ has been calculated. EPA has also established an inhalation unit risk of 1.8E-3 per ($\mu g/m^3$) based on the oral data for *alpha*-BHC.

The Department of Health and Human Services has also determined that BHC may be reasonably anticipated to cause cancer in humans (ATSDR 1997), and IARC ranks it as a 2B (possible human carcinogen) (ATSDR 1997).

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HEPTACHLOR EPOXIDE

Heptachlor epoxide ($C_{10}H_5Cl_7O$, CAS Registry No. 1024-57-3) is a natural degradation product of heptachlor, and is also the primary metabolite of heptachlor. Other metabolites of heptachlor epoxide have been observed, but it has a relatively long half-life.

Carcinogenicity

Heptachlor epoxide have been given the carcinogenicity weight-of-evidence classification of B2 (probable human carcinogen) in the absence of conclusive human data (USEPA 2001).

Systemic Toxicity

Chronic oral exposure to heptachlor epoxide affects the liver, with heptachlor also affecting renal and neurological systems. Liver effects have included alterations in liver weights with minimal pathological influences on tissues. Kidney effects were altered weights, and an increase in blood urea (this may indicate kidney inefficiency in protein clearance). Neurological long-term effects may include tremors, muscle spasms, convulsions. Heptachlor epoxide was negative in the dominant lethal test in rodents after oral ingestion.

Minimal information on long-term effects after inhalation was available. Most inhalation studies were of acute occupational exposures and affected the same target organs as oral exposures.

No chronic studies of dermal exposure to heptachlor epoxide were located.

Toxicokinetics

Heptachlor epoxide is absorbed in the intestine, and tends to bioaccumulate primarily in tissues with high fat content, then in liver, kidney, and brain (HSDB 2000)..

Toxicity Values

The oral RfD for heptachlor epoxide is 0.000013 mg/kg/day (USEPA 2001), based on increased liver-to-body-weight ratios in both male and female dogs in a 14-month study (Dow Chemical 1958). An uncertainty factor of 1000 allows for intra- and interspecies differences and the failure to demonstrate a NOEL (USEPA 2001). No inhalation RfC has been calculated for either compound (USEPA 2001).

The oral cancer slope factor for heptachlor epoxide is 9.1 per (mg/kg)/day, based on the geometric mean of four slope factors from studies producing hepatocellular carcinomas in two strains of mice. Davis (1965) fed the compound in the diet for 2 years and Velsicol (1973) for 18 months, respectively, and data were reinterpreted by Reuber

(1977) (USEPA 2001). An early two-year study in CFN rats (Witherup 1959) provides supporting evidence that, although rats were less sensitive than the mice, female rats demonstrated a statistically significant increase in hepatic carcinomas.

EPA has calculated an inhalation unit risk value of 0.0013 per ($\mu g/m^3$) for heptachlor and 0.0026 per ($\mu g/m^3$) for heptachlor epoxide from the oral data, using standard conversion factors for route-to-route extrapolation (USEPA 2001).

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INDENO(1,2,3-C,D)PYRENE

Studies investigating the subchronic or chronic systemic toxicity of indeno(1,2,3-cd)pyrene by skin contact, ingestion, or inhalation have not been conducted (ATSDR, 1995).

Indeno(1,2,3-c,d)pyrene induced skin tumors following intermediate or long-term dermal exposure.

Toxicity values

No toxicity values for health effects other than cancer have been derived for indeno(1,2,3-c,d)pyrene.

Insufficient data are available to derive a SF for indeno(1,2,3-c,d)pyrene. Based on USEPA Region IV guidance (USEPA 1996), the oral SF for benzo(a)pyrene was used as a surrogate. The oral SF is multiplied by 0.1 to obtain a SF of 7.3×10^{-1} $(mg/kg\text{-}day)^{-1}$ for indeno (1,2,3-c,d)pyrene.

USEPA (1989) guidance recommends against deriving a dermal SF from oral data for the carcinogenic PAHs.

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IRON

Iron (Fe, CAS registry number 7439-89-6) is the fourth most abundant element in the earth's crust and the second most abundant metal. It comprises approximately 5 percent of the continental crust. Its concentration in ground water ranges from 0.5 mg/L to 10 mg/L; its concentration in soil is between 0.7 and 4.2 percent (NRC 1979). In the late 1970s, world production of iron was over 500 million metric tons, with the U.S. producing roughly 20 percent of the world total (NRC 1979). Since iron is an essential nutrient, some amount of iron is needed in the diet.

Iron has not been reported to be mutagenic (NRC 1979). It has not been classified with respect to carcinogenicity, given the paucity of animal cancer bioassays and human cancer studies. Iron overload may be associated with carcinoma of the liver; however the data are poor and inconclusive (NRC 1979).

The acute effects of iron toxicity in humans are well characterized and consist of gastrointestinal, cardiovascular, metabolic, neurological and hepatic alterations (Bothwell et al. 1979; Banner and Tong 1986; Engle et al. 1987; and Mann et al. 1989, all as cited in EPA 1993). Acute effects are based mostly on observations of children who accidentally ingest therapeutic iron supplements; they are rarely, if ever, associated with ingestion of naturally occurring or other commercially produced substances (NRC 1979). Gastrointestinal toxicity is characterized by vomiting, diarrhea, and abdominal pain, caused by the direct caustic effect of iron on the mucosa of the stomach and small intestine. Gastrointestinal toxicity can progress to gastric/intestinal hemorrhage and/or necrosis and, in rare cases, to stenosis in the stomach outlet and small intestine. Cardiovascular iron toxicity is marked by severe hemodynamic alterations and can lead to shock and cardiac failure; neurological toxicity ranges from lethargy to coma. Although a rare occurrence, hepatic toxicity from iron can range from cloudy swelling of hepatocytes to necrosis. The average human lethal dose is 200B250 mg/kg body weight (NRC 1979). Thus, the average adult male would have to ingest 14 grams of elemental iron for it to be lethal; the average 2 year old, 3 grams (NRC 1979).

Chronic iron toxicity has been noted in individuals with various genetic and/or metabolic disorders, including hemochromatosis (massive iron overload together with cirrhosis and/or other tissue damage due to iron), thalassemia, and sideroblastic anemia, as well as in individuals who receive frequent blood transfusions (Jacobs 1977, and Bothwell et al. 1979, both as cited in EPA 1993). Excessive intake of iron attributed to consumption of home-brewed Kaffir beer has resulted in chronic hemochromatosis among the South African Bantu population (NRC 1979; and Bothwell and Bradlow 1960; and Bothwell et al. 1964, both as cited in EPA 1993). Pathologic findings associated with hemochromatosis include: 1) fibrosis in heavily siderotic organs, particularly the liver, 2) cirrhosis, 3) testicular atrophy, and 4) osteoporosis (NRC 1979).

Though chronic iron toxicity can occur in individuals with genetic/metabolic disorders, it is debatable whether a chronic overload via ingestion is possible in individuals with a normal ability to control iron absorption. Using values obtained from the second

National Health and Nutrition Examination Survey (NHANES II), Looker et al. (1988, as cited in EPA 1993) compared dietary iron intake with biochemical indices of iron status. NHANES II consisted of a 1976B1980 sample of the U.S. population aged 6 months to 74 years. Observed intake levels of 0.15B0.27 mg/kg-day iron were found to be both great enough to prevent iron deficiency and insufficient to cause the toxic effects of iron overload (Elinder 1986; Cook 1991; Hillman and Finch 1985, all as cited in EPA 1993). Lauffer (1991, as cited in EPA 1993) and Sullivan (1992, as cited in EPA 1993) suggest that iron overload elevates the risk of acute myocardial infarction by promoting oxidation of low density lipoprotein (LDL). A 1992 Finnish study of 1,931 randomly selected men aged 42B60 years by Salonen et al. lends support to this theory in that it found that high serum ferritin concentration and high dietary iron intake were risk factors for myocardial infarction.

Animal studies attempting to model hemochromatosis have been mostly negative, as have animal studies involving parenteral administration of iron (Bothwell et al. 1979, as cited in EPA 1993; and NRC 1979).

Ingestion of iron supplements during pregnancy has not been correlated to adverse developmental effects in humans, although some women ingesting large quantities of iron (>1.2 gram) during pregnancy experienced nausea, vomiting, hematoemesis, abdominal pain, and/or diarrhea (NAS 1989, as cited in EPA 1993). No teratogenic effects have been associated with iron (NRC 1979).

No treatment-related teratogenic or embryotoxic effects were observed in rats given 2.7 mg/kg-day iron on gestation days 6-15 or rats/mice given 24-76 mg/kg-day iron for 6 days (Nolen et al. 1972; Tadokoro et al. 1979, as cited in EPA 1993).

This essential nutrient is found primarily in the form of hemoglobin in the body. The concentration of iron in the body at any given point is regulated largely through changes in the amount of iron absorbed by the gastrointestinal mucosa. The following factors influence the absorption of iron: 1) body stores, 2) the amount and nature of iron in ingested food, and 3) dietary factors that may increase or decrease the availability of iron for absorption (NRC 1979). Although the body is generally effective in regulating iron levels, it is incapable of excreting large amounts of iron following excessive accumulation resulting from acute or chronic ingestion of high levels of iron (NAS 1989, as cited in EPA 1993).

Toxicity values

Using values obtained from the second National Health and Nutrition Examination Survey (NHANES II), Looker et al. (1988, as cited in EPA 1993) compared dietary iron intake with biochemical indices of iron status. NHANES II consisted of a 1976-1980 sample of the U.S. population aged 6 months to 74 years. Observed intake levels of 0.15-0.27 mg/kg-day iron were found to be both great enough to prevent iron deficiency and insufficient to cause the toxic effects of iron overload (Elinder 1986; Cook 1991; and Hillman and Finch 1985, as cited in EPA 1993). EPA (1993) has proposed a provisional

chronic oral RfD of 0.3 mg/kg-day based on a NOAEL of 0.27 mg/kg-day (i.e., the upper-bound value in the range of mean dietary iron intakes from the NHANES II data base) and an uncertainty factor of 1. An uncertainty factor of 1 was used since: 1) iron is an essential nutrient, 2) the NHANES II data base comprised a relatively large sample size, and 3) humans exert an efficient homeostatic control over iron such that body burdens are kept constant with variations in diet. EPA's confidence in the critical study is high, while confidence in the data base is medium, resulting in medium confidence in the RfD. EPA (1993) suggests that the RfD may not be protective of people with inherited disorders of iron metabolism. In addition, EPA states that the RfD could be conservative if applied to exposure scenarios involving forms of iron with low bioavailability. The NCEA has established a provisional chronic RfD of 0.3 (mg/kg/day) (USEPA 1996, 1998). Iron has not been classified with respect to carcinogenicity. Slope factors are not available for iron.

The TLV-TWA for iron salts is 1 mg/m^3 (ACGIH 1995).

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LEAD

Lead (Pb; CAS Registry No. 7439-92-1) and lead compounds are present in the environment both naturally and as a result of anthropogenic activities. Lead compounds have many industrial applications, such as in the plates of electric batteries and accumulators; as compounding agents in rubber manufacturing; as ingredients in paints, glazes, glass, and pigments; and in the chemical industry. Exposures to lead dust may occur during the manufacture of these product, or during mining, smelting, and refining; and exposures to lead fumes may occur during such high-temperature operations as welding or spray-coating of metals with molten lead, or through waste incineration. Lead may occur naturally in drinking water and unnaturally in water delivered through lead pipes to residents of older homes in soft-water areas. Inadequately glazed earthenware or china may contain lead that leaches out on contact with heated or acidic foods. It occurs in the house dust of older homes that were once painted with lead paint. The level of exposure resulting from contact is highly variable. Children with pica for paint chips or for soil may have elevated blood lead levels ranging from marginal to high enough to cause clinical illness (HSDB 2000).

The majority of the toxic effects of lead have been observed in humans. Effects of lead on humans are typically discussed in terms of their correlating absorbed internal body dose, expressed as a blood lead level (ug/dL). In many instances, internal doses are actually the result of multiple routes of exposure.

Cancer

Lead at very high doses has been shown to cause some tumors in animals (Azar *et al.* 1973; Koller *et al.* 1985; Van Esch and Kroes 1969). However, epidemiology studies, despite large exposures among workers, have failed to indicate that lead may cause cancer in humans (Kang *et al.* 1980; Cooper *et al.* 1985; Selevan *et al.* 1985; Baker *et al.* 1980). EPA has classified lead as a Category B2 carcinogen, due to sufficient evidence from animal bioassays (renal tumors in rats ingesting lead for a lifetime) but inadequate evidence in humans (USEPA 2001). The data are considered inadequate to develop a slope factor for lead. Lead appears to be more potent as a systemic toxicant to the hemopoietic and neurologic systems than as a cancer-causing agent.

Mutagenicity

According to EPA, "Lead acetate induces cell transformation in Syrian hamster embryo cells (DiPaolo et al. 1978) and also enhances the incidence of simian adenovirus induction. Lead oxide showed similar enhanced adenovirus induction (Casto et al. 1979). Under certain conditions lead compounds are capable of inducing chromosomal aberrations in vivo and in tissue cultures. Grandjean et al. (1983) showed a relationship between SCE and lead exposure in exposed workers. Lead has been shown, in a number of DNA structure and function assays, to affect the molecular processes associated with the regulation of gene expression (USEPA 1986)" (USEPA 2001).

Systemic Toxicity

The predominant systems affected by chronic lead inhalation or ingestion by humans are the hematologic, neurologic, cardiovascular, and renal systems (ATSDR 1993). The basis for lead toxicity is its ability to bind to ligating groups in physiologically critical biomolecules, which may then disrupt their function by competing with essential metal ions for binding sites, inhibiting enzyme activity, and altering ion transport (Calabrese and Kenyon 1991). Toxic hematologic effects include inhibition of several enzymes involved in heme synthesis, with resultant depression of hemoglobin synthesis and anemia. The primary cardiovascular effect is elevation of blood pressure, a dose-dependent effect that also lacks any apparent threshold (Harlan 1988). Neurologic effects typically manifest themselves as neurobehavioral effects and alterations of the peripheral nervous system. Consequences can include weakness in the limbs (Zimmerman-Tansella et al. 1983), fatigue (Hänninen et al. 1979; Baker et al. 1979), loss of memory function (Hänninen et al., 1979), significant impairment in learning capacity (Wang et al. 1989; Rummo et al. 1979; Ernhart et al. 1981), limb tremors (Baker et al. 1979), and encephalopathy at higher blood levels (Rummo et al. 1979). High blood lead levels have also been correlated with nephropathy, gastrointestinal effects, and electrocardiographic abnormalities (ATSDR 1993).

Exposure to environmental lead levels which translate to relatively low blood lead levels has been shown to have adverse effects upon several physiological parameters. These include the alteration of several of the enzymes involved in the synthesis of the heme moiety, an essential component of substances like hemoglobin (the oxygen transporter in red blood cells), cytochromes P-450 (major xenobiotic detoxification mechanisms) and respiratory cytochromes (major source of metabolic energy reserves). In addition, neurologic effects upon behavior, memory, learning capacity, and emotion have been established. Increases in blood pressure in the middle-aged have also been observed. These effects manifest themselves at all measured blood lead levels, with no known threshold in humans. However, animal studies have been performed to evaluate the toxic endpoints of low level lead exposure, and doses at which there are no or minimal apparent effects have been achieved. The existence of neurologic impairments from serum levels of lead below 10 μg/dL is currently being debated in the scientific community. Because infants and young children appear to be particularly sensitive to the neurologic effects of lead, no threshold dose has been established for either oral or inhalation exposure.

Toxicokinetics

The absorption rate of deposited lead depends on various factors, particularly on the physicochemical form of lead in particles. No evidence of lead accumulation in the lungs exists (Friberg et al. 1986). About 90% of lead particles in ambient air that are deposited in the lungs are small enough to be retained and are absorbed through alveoli into the bloodstream. Larger particles are cleared from the repiratory tract and swallowed. (Doull et al. 1986).

Absorbed lead is transported by blood to various organs and tissues, particularly the liver and kidney. It is then gradually redistributed to form an exchangeable compartment between blood and soft tissues and a storage compartment in bone, teeth, and hair. In humans subjects with low lead exposure, about 90% of the total body burden is found in bone. Lead in bone builds up throughout life. In soft tissues it stabilizes early in adult life and may decrease with age. Lead circulating in the blood is mainly bound to erythrocytes where its concentration is about 16 times higher than in plasma. Lead also has an affinity for cell membranes and mitochondria, but not for lysosomes. (Friberg et al. 1986; Gilman et al. 1985).

Placental transfer of lead has been demonstrated. There is fairly good correlation between lead concentrations in the blood of mothers and newborn infants, and the distribution in fetal tissues is similar to the distribution in the adult (Friberg et al. 1986; Doull et al. 1986).

Metabolism

About 90% of ingested lead is unabsorbed and eliminated through feces. Absorbed lead is excreted primarily in urine (about 76%); about 16% is eliminated through gastrointestinal secretions, and less than 8% through hair, nails, and sweat. The rate of biliary excretion in man is not known. The mechanism of excretion appears to be essentially glomerular filtration. Lead is also excreted in human milk in concentrations up to 12 μg/L.

Lead excretion varies considerably among animal species. The main route of excretion is via the urine in baboons; but in rats and sheep, biliary and transmucosal excretion may be higher than urinary excretion (Friberg et al. 1986).

Toxicity Values

The derivation of a toxicity value for an allowable lifetime exposure hinges upon the concept of a threshold under which the critical toxic effect will not be manifested during the average person's lifetime and over which the critical effect, as well as additional toxic effects, may occur. However, certain toxic effects have been observed to occur at any measured blood levels of lead.

No lead toxicity values have been formally adopted by regulatory agencies. There are two major complicating factors in assigning a toxicity value to lead and predicting subsequent risks associated from exposure. These factors include the complex nature of the kinetic modeling of lead distribution within the human body, and the lack of a threshold level for certain adverse health effects. Inherent in reaching any conclusion with respect to the toxic effects of lead, or any compound, is a knowledge of the internal levels of exposure as well as the relationship between the exposure level and the critical or toxic endpoint. Once lead is internalized, it distributes among blood, soft tissue, and bone. The complication arises due to the fact that lead in bone, while remaining complexed there for very long time periods (decades), can be redistributed throughout the

body, thus giving additive exposures beyond the initial exposure. In this way, a cumulative toxic effect can occur, which may not have initially been expressed.

A blood lead level of 10 μg/dL has been identified as a level at which toxic effects in humans can be manifested (e.g., decreased intelligence, impaired neurobehavioral development, elevated blood pressure) (CDC 1991).

Currently, lead is not regulated as a carcinogen because it appears to be more potent as a toxicant to the hemopoietic system by inhibiting heme synthesis.

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MANGANESE

Manganese (Mn; CAS Registry No. 7439-96-5) is a pinkish gray, lustrous and brittle metal that is used in rock crushers and in the manufacture of ceramics, matches, glass, dyes, welding rods. It is a component of steel and metal alloys. (HSDB 2000).

Cancer

EPA considers manganese to be unclassifiable as a human carcinogen (Category D), based on an absence of human carcinogenicity data, inadequate evidence of carcinogenicity in animals, and inadequate genotoxicity data (USEPA 2001).

Mutagenicity

Few genotoxicity assays of manganese have been conducted. No studies were located regarding genotoxic effects in humans (ATSDR 1991). Treatment of male rats with manganese at repeated oral doses of 0.014 mg/kg-day manganese for 80 days did not produce any significant chromo-somal damage in bone marrow or sperm cells (Dikshith and Chandra 1978). Results of *in vitro* genotoxicity assays have been mixed. The available data indicate that manganese may have genotoxic potential, but they are not sufficient to evaluate the genotoxic risk of manganese to humans (ATSDR 1991).

Systemic Toxicity

Humans exert an efficient homeostatic control over manganese so that body burdens are kept constant with variations in diet. Manganese is an essential element, being required for normal human growth and maintenance of health. Children may be less susceptible to manganese intoxication and may require slightly higher levels of manganese than do adults (USEPA 2001).

The World Health Organization (WHO 1973) reviewed several investigations of adult diets and reported the average daily consumption of manganese to range from 2.0 to 8.8 mg/day. Higher manganese intakes are associated with diets high in whole-grain cereals, nuts, green leafy vegetables, and tea. Depending on individual diets, a normal intake may be well over 10 mg Mn/day, especially from a vegetarian diet. While the actual intake is higher, the bioavailability of manganese from a vegetarian diet is lower, thereby decreasing the actual absorbed dose. From manganese balance studies, the WHO concluded that 2-3 mg/day is adequate for adults and that 8-9 mg/day is "perfectly safe" (WHO 1973).

An epidemiologic study of manganese in drinking water was performed by Kondakis et al. (1989). Three areas in northwest Greece were chosen for this study, with manganese concentrations in natural well water of 3.6-14.6 ug/L in area A, 81.6-252.6 ug/L in area B, and 1,600-2,300 ug/L in area C. The total population of the 3 areas being studied ranged from 3,200 to 4,350 people. Although the amount of manganese in the diet was not reported, the authors indicated that most of the food was purchased from markets.

The individuals chosen were submitted to a neurologic examination, the score of which represents a composite of the presence and severity of 33 symptoms (e.g., weakness/fatigue, gait disturbances, tremors, dystonia). Whole blood and hair manganese concentrations were also determined. The authors indicate that the difference in mean scores for area C versus A was significantly increased for both sexes combined. In a subsequent analysis, logistic regression indicated that there is a significant difference between areas A and C, even when both age and sex are taken into account (Kondakis 1990). The NOAEL identified in this epidemiological study was 0.005 mg/kg-day (USEPA 2001).

The major toxic effects of inhaled manganese are primarily neurological. A syndrome called "manganism" has been observed only in workers exposed to chronic, high levels of manganese. It is characterized by preliminary general weakness, anorexia and muscle pain, with psychological signs such as apathy and dullness, as well as impotence. Advanced stages include difficulty in walking, muscle tremor, and behavioral disturbances. This syndrome has not been observed for low level, chronic or sporadic exposures, nor has it been observed in studies with animals (ATSDR 1991).

Roels et al. (1992) conducted a cross-sectional study of 92 male workers exposed to manganese dioxide (MnO_2) dust in a Belgian alkaline battery plant. A control group of 101 male workers was matched for age, height, weight, work schedule, coffee and alcohol consumption, and smoking; educational level was slightly higher in the control group. The manganese-exposed group had been exposed to MnO_2 for an average of 5.3 years (range: 0.2-17.7 years). The geometric means of the workers' TWA airborne manganese concentrations, as determined by personal sampler monitoring at the breathing zone, were 0.215 mg/m^3 for respirable dust and 0.948 mg/m^3 for total dust. The authors noted that the personal monitoring data were representative of the usual exposure of the workers because work practices had not changed during the last 15 years of the operation of the plant.

Geometric mean concentrations of blood manganese (MnB) (0.81 ug/dL) and urinary manganese (MnU) (0.84 ug/g creatinine) were significantly higher in the Mn-exposed group than in the control group, but on an individual basis no significant correlation was found between either MnB or MnU and various external exposure parameters. A self-administered questionnaire focused on occupational and medical history, neurological complaints, and respiratory symptoms. Responses to the questionnaire indicated no significant differences between groups in either respiratory or neurological symptoms, nor were spirometric, hormonal, or calcium metabolism measurements significantly different for the two groups (Roels et al 1992).

Of particular note, manganese workers performed worse than controls on several measures of neurobehavioral function. Visual reaction time was consistently and significantly slower in the manganese-exposed workers measured in four 2-minute periods, with more pronounced slowing over the total 8-minute period and significantly greater variability in reaction times for the exposed group. Abnormal values for mean reaction times (defined as greater than or equal to

the 95th percentile of the control group) also were significantly more prevalent in the exposed group during three of four 2-minute intervals of the 8-minute testing period. Five measures of eye-hand coordination (precision, percent precision, imprecision, percent imprecision, and uncertainty) reflected more erratic control of fine hand-forearm movement in the exposed group than in the controls, with mean scores on all five measures being highly significantly different for the two groups. There was also a significantly greater prevalence of abnormal values for these five measures in the manganese-exposed group. The hole tremormeter test of hand steadiness indicated a consistently greater amount of tremor in the exposed workers, with performance for two of the five hole sizes showing statistically significant impairment (Roels et al. 1992).

A LOAEL may be derived from the Roels et al. (1992) study by using the IRD concentration of MnO_2, expressed as mg/m^3 years (based on 8-hour TWA occupational exposures for various job classifications, multiplied by individual work histories in years). Dividing the geometric mean IRD concentration (0.793 mg/m^3 years) by the average duration of the workers' exposure to MnO_2 (5.3 years) yields a LOAEL of 0.15 mg/m^3. Adjusted for continuous exposure, the LOAEL is 0.05 mg/m^3.

Roels et al. (1987) conducted a cross-sectional study in 141 male workers exposed to MnO_2, manganese tetroxide (Mn_3O_4), and various manganese salts (sulfate, carbonate, and nitrate). A matched group of 104 male workers was selected as a control group. The two groups were matched for socioeconomic status and background environmental factors; in addition, both groups had comparable work-load and work-shift characteristics. Significant differences in mean scores between manganese-exposed and reference subjects were found for objective measures of visual reaction time, eye-hand coordination, hand steadiness, and audio-verbal short-term memory. The prevalence of abnormal scores on eye-hand coordination and hand steadiness tests showed a dose-response relationship with blood manganese levels; short-term memory scores were related to years of manganese exposure but not to blood manganese levels. The prevalence of subjective symptoms was greater in the exposed group than in controls for 20 of 25 items on the questionnaire, with four items being statistically significant: fatigue, tinnitus, trembling of fingers, and irritability. Based upon the findings of impaired neurobehavioral function in workers whose average Mn exposure was estimated by the geometric mean TWA of total airborne manganese dust at the time of the study, a LOAEL of 0.97 mg/m^3 was identified, which, when adjusted for continuous exposure, is equivalent to a LOAEL of 0.34 mg/m^3. This LOAEL is based on total manganese dust of mixed forms, whereas the LOAEL from Roels et al. (1992) study is based on the measured respirable dust fraction of MnO_2 only.

Minimal information regarding manganese and the dermal exposure route could be located. It is generally regarded that manganese uptake across intact skin is very limited, as is the case for most inorganic forms of metal ions (ATSDR 1991).

Toxicokinetics

Exposure to manganese mainly occurs via ingestion and inhalation. The extent to which manganese is absorbed across the intestine is approximated at 3-5 percent, and does not appear to be substantially influenced by the carrier medium (i.e., water versus food). Similar extents of absorption have been noted in animals as well, with typical amounts equal to 2.5-5.5 percent. Manganese distributes to various tissues following ingestion, and serves as a normal tissue constituent. Tissue levels may be somewhat higher in animal tissues than in their human tissue counterparts. Manganese which is inhaled, typically in particle form, is absorbed to some unknown extent across the lungs, and a certain percentage of inhaled manganese particles are subsequently swallowed and ingested as well (ATSDR 1991).

Metabolism

Manganese is not known to be metabolized or biotransformed, and behavior within the body would be essentially limited to absorption, distribution, potential sequestration, and excretion. The valence state of manganese is thought to undergo changes within the body (alterations in oxidation state), which may influence its ability to form complexes or serve as a co-factor for certain proteins (ATSDR 1991).

The information used to determine the RfD for manganese in food was taken from many large populations consuming normal diets over an extended period of time with no adverse health effects (WHO 1973; NRC 1989; Schroeder et al. 1966). A NOAEL of 0.14 mg/kg-day (corresponding to 10 mg/day for a 70 kg adult) is based on a composite of data from all three references.

Toxicity Values

The information used to determine the RfD for manganese in food was taken from many large populations consuming normal diets over an extended period of time with no adverse health effects (WHO 1973; NRC 1989; Schroeder et al. 1966). A NOAEL of 0.14 mg/kg-day (corresponding to 10 mg/day for a 70 kg adult) is based on a composite of data from all three references.

Two separate oral RfDs for manganese have been established by EPA: one based on ingestion of manganese in water and the other based on the ingestion of manganese in food (USEPA 2001). The information used to determine the RfD for manganese in food was taken from many large populations consuming normal diets over an extended period of time with no adverse health effects (WHO 1973; NRC 1989; Schroeder et al. 1966). A NOAEL of 0.14 mg/kg-day (corresponding to 10 mg/day for a 70 kg adult) is based on a composite of data from all three references. The Food and Nutrition Board of the National Research Council (NRC 1989) determined an "adequate and safe" intake of manganese to be 2-5 mg/day for adults. This level was chosen because it includes an "extra margin of safety" from the level of 10 mg/day, which the NRC considered to be safe for an occasional intake. An RfD of $5x10^{-3}$ mg/kg-day for manganese in water is equivalent to a drinking water standard of 0.2 mg/L and is based on human chronic ingestion data (Kondakis et al. 1989). The RfD for ingestion of food is $1.4x10^{-1}$

mg/kg-day and is also based on a NOAEL derived from human chronic ingestion data (NRC 1989; WHO 1973; Freeland-Graves et al. 1987). Because these RfDs are based on NOAELs identified in chronic human studies, no uncertainty factors were applied in calculating them (USEPA 2001). EPA has assigned these chronic RfDs as surrogates for use with subchronic exposures without adjustment (USEPA 1994). The water and food RfDs have been combined to provide the RfD used in this assessment of 0.024 mg/kg/day.

The inhalation RfC for manganese is based on a LOAEL of 0.05 mg/m^3 as determined by Roels et al. (1992) (USEPA 2001). An uncertainty factor of 1,000 reflects factors of 10 to protect sensitive individuals, 10 for use of a LOAEL, and 10 for database limitations reflecting both the less than chronic periods of exposure and the lack of developmental data, as well as potential but unquantified differences in the toxicity of different forms of manganese. Thus, the RfC is $5x10^{-5}$ mg/m^3, which is equivalent to an inhalation RfD of $1.4x10^{-5}$ mg/kg-day. No cancer slope factors can be calculated for manganese at this time because of a lack of data.

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NAPHTHALENE AND 2-METHYLNAPHTHALENE

Naphthalene ($C_{10}H_8$, CAS Registry No. 91-20-3) is a white crystalline solid that is sometimes powdered or flaked. It has the odor of moth balls and is used as an ingredient of some moth repellants and toilet bowl cleaners. It is also a combustion product of crude oil, gasoline, diesel fuel (and thus a constituent of vehicle emissions) and tobacco. Melted naphthalene is corrosive to some forms of plastics, rubber, and coatings (HSDB 2000).

2-Methylnaphthalene ($C_{11}H_{10}$, CAS Registry No. 91-57-6) is also a solid, primarily used in vitamin K production and as a chemical intermediate, a dye carrier, and a pesticide adjuvant. Its presence in many products insures that it is also released to the environment via landfills, waste incinerators, and manufacturing effluents. As a combustion product of crude oil, 2-methyl-naphthalene is released to the environment during natural fires, petroleum refining, coal tar distillation, and in vehicle emissions. Greatest exposures to both substances are likely to be occupational, at workplaces where the they are used or produced from coal tar (HSDB 2000).

Cancer

Naphthalene has been tested in studies capable of detecting the ability to cause cancer; however, all but one study showed no such evidence. In the 2-year NTP study of chronic toxicity and carcinogenicity (NTP 1992), a statistically elevated incidence of lung alveolar/bronchiolar adenomas at the high dose level was observed in female mice at the highest dose tested. This study provides weak, possibly questionable, evidence of carcinogenicity, because (a) only one sex showed the tumorigenic effect; (b) only one tumor type (benign) was observed; (c) no dose-response existed, since the tumors were present only in the group receiving the highest dose; and (d) the strain of mice used in the study typically exhibits high spontaneous rates of lung adenomas.

EPA has reclassified the potential carcinogenicity of naphthalene to Group C, based on inadequate human carcinogenicity data and limited evidence of carcinogenicity in animals via the inhalation route (USEPA 2001).

No studies have been conducted on the carcinogenicity of 2-methylnaphthalene in humans. Its carcinogenicity potential has not been classified by EPA (USEPA 2001).

Systemic Toxicity

Several studies have been reported on the subchronic and chronic toxicity of naphthalene. At high inhalation and oral doses, naphthalene can produce injury. Cataracts were diagnosed in 8 of 29 chemical plant workers occupationally exposed to high doses of naphthalene for 5 years (Ghetto and Mariani 1956). In addition, skin contact with naphthalene can result in irritation or severe dermatitis in sensitized humans (Manahan 1989). The only reported effects of 2-methyl-naphthalene are skin irritation and skin photosensitization (HSDB 1999).

Naphthalene ingestion has resulted in abdominal pain, nausea, vomiting, diarrhea, urine darkening, bladder irritation, jaundice, anemia, and hyperthermia (Gerarde 1960). Daily oral administration of 1 g/kg to rabbits produced degenerative changes in the lens of the eye; within 2 weeks the whole lens became cataractous (Potts 1986). A review of the literature on cataract formation in rodents and rabbits resulting from naphthalene administration demonstrates that metabolic pathways of naphthalene differ among strains of rats, mice, and rabbits, and only some strains are susceptible to cataract formation from naphthalene ingested at high doses (Van Heyningen and Pirie 1976). Hemolytic anemia was observed in dogs given oral doses of 3-9 g of naphthalene (Zuelzer and Apt 1949), and the effect was reversible within 50 days after cessation of exposure.

The inhalation of high acute doses of naphthalene by humans has caused headache, confusion, eye irritation, nausea, profuse perspiration with vomiting, optic neuritis, hematuria, and edema (Gerarde 1960). In a 2-year study (NTP 1992), chronic exposure of mice to naphthalene at airborne concentrations of 10 or 30 ppm resulted in chronic inflammation of the lung and nose, metaplasia of the olfactory epithelium, and hyperplasia of the respiratory epithelium. In this study, a statistically significant increase in lung adenomas occurred in females of the highest dose group compared to controls.

Toxicity Values

The oral RfD for naphthalene is 2×10^{-2} mg/kg-day (USEPA 2001), based on an unpublished subchronic study (Battelle 1980), in which rats were administered naphthalene by gavage five days/week for 13 weeks. A NOAEL of 100 mg/kg-day was identified, based on decreased mean body weights in males at the two highest doses tested. There were no changes in either food or water consumption. The NOAEL was converted to an equivalent daily dose of 72 mg/kg-day, and the chronic RfD was calculated using an uncertainty factor of 1,000 to account for intra- and inter-species variability and for the use of a subchronic study. Oral chronic and subchronic RfDs were used for inhalation RfDs without further adjustment. No reference doses have been determined for 2-methylnaphthalene (USEPA 2001).

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2,3,7,8-TETRACHLORODIBENZO-*p*-DIOXIN (DIOXIN, OR TCDD)

2,3,7,8-tetrachlorodibenzo-*p*-dioxin ($C_{12}H_4Cl_{14}O_2$; CAS Registry No. 1746-01-6) is a white to colorless crystalline solid that is ubiquitous in the environment.

Cancer

TCDD, or dioxin, is a probable human carcinogen (Group B2), based on observations of increased soft tissue sarcoma and lymphomas in populations exposed primarily via contact with herbicides contaminated with 2,3,7,8-TCDD (EPA 1997). ATSDR (1989) summarized the epidemiological data as providing limited evidence that exposure to phenoxyacetic acid herbicides and/or chlorophenols is causally related to the risks of soft tissue sarcoma, but none of the data sufficed to implicate 2,3,7,8-TCDD alone. Animal carcinogenicity studies were related to dermal exposure, and indicated skin tumors resulted from application to female Swiss mice. Mixed results were obtained regarding the promoter-like potential of dioxin.

Mutagenicity

Genotoxicity of dioxin has been reported as mixed results in both humans and animals. Humans exposed to chemicals contaminated with dioxin (herbicide production workers) were reported to have increased chromosomal aberrations in peripheral lymphocytes, while soldiers exposed to Agent Orange had no such increases. Animal studies seem to indicate that while there is evidence of genotoxic and mutagenic effects, more definitive, confirmatory studies are required (ATSDR 1989).

Systemic Toxicity

Effects following dioxin exposure via the oral route have been well-studied in animals, and to a lesser extent in humans. With a variety of relevant experimental durations (i.e., subchronic or chronic; from >14 days to >1 year), adverse effects from dioxin were expressed predominantly as decreased longevity, as well as reproductive, immunological, and hepatic effects (ATSDR 1989). In general, for exposures of 15 days to 1 year, most of the animal NOAELs for these effects occur at ingestion of dioxins ranging from approximately 0.001 (longevity and liver effects) to 0.01 g/kg/day (immunotoxicity), with no human NOAELS. Animal LOAELs ranged from 0.001 g/kg/day (reproductive, liver effects) to 0.01 g/kg/day (immunotoxicity), with no human LOAELs. For exposures of 1 year or longer, only an animal NOAEL of 0.001 g/kg/day for liver toxicity was observed, with no human NOAELs or LOAELs.

One study evaluated the response of nude mouse skin to subchronic application. Application of approximately 5 g/kg/day resulted in chloracne (facial and upper body skin lesions). This effect has been observed mostly in humans, although quantitative dose levels have not been determined. A human minimum toxic effect dose of 0.1 g/kg was estimated (using non-human primate data and applying human data extrapolated from ingestion of PCB- and DBF-contaminated rice oil to dioxin dose data).

Toxicokinetics

The absorption of dioxin is most dependent on the vehicle of administration, such as in an oil base or in the diet, and factors determined from animal studies. After oral ingestion in an oil vehicle, absorption ranges from 50 to 80 percent, while in the diet, it is between 50 and 60 percent. Dermal absorption was to a much more limited extent, although it was also highly affected by the vehicle. Dermal absorption in rats has ranged from 40 percent (in methanol) to less than 2 percent (a soil paste or activated carbon). Data were not available for inhalation absorption. Once absorbed, dioxin appears to be distributed through the body via the lymphatic system (in rats), and concentrates in liver and fatty tissue. The average half life of dioxin in humans was estimated at approximately 7 years, although half lives as long as 27 years were also estimated (ATSDR 1989).

Metabolism

The primary metabolic processes following dioxin exposure involve the attachment of hydroxyl groups onto the parent compound or the substitution of a hydroxyl group for a chlorine atom. Although dioxin is not rapidly metabolized, once metabolites form, they are eliminated fairly rapidly as conjugates with glucuronide and sulfates. Metabolites are thought to be less toxic than dioxin itself, and it has been suggested that the rates of metabolism, as well as the types of metabolites may account for differential effects observed in several species (ATSDR 1989).

Toxicity Values

No oral reference dose has been calculated for 2,3,7,8-TCDD or its isomers (EPA 1997, 1999).

EPA (1997) has proposed an oral SF of 1.5 10^5 $(mg/kg\text{-}day)^{-1}$ for TCDD. An interim approach for extrapolating TCDD toxicity values for use with various isomers exhibiting fractional toxicities relative to 2,3,7,8-TCDD (i.e., use of toxicity equivalence factors) has been developed (EPA 1994). The critical study involved oral exposure of rats to dioxin in the diet for approximately 2 years, and yielded respiratory tumors and liver tumors (EPA 1997).

EPA (1997) has proposed an inhalation SF of 1.5 10^5 $(mg/kg\text{-}day)^{-1}$ for TCDD. The toxicity equivalence factor approach has also been proposed for use with inhalation exposures. The critical study was the same as that used for the oral SF.

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THALLIUM

Thallium (Tl, CAS Registry Number 7440-28-0) is a metallic element used as a catalyst, in alloys, optical lenses, electronics, jewelry, thermometers, pigments, and in scintillation counters; historically, the element was also used as a depilatory, a rodenticide, and an insecticide (Hammond and Beliles, 1980). In nature, thallium is a trace compound in many minerals, such as potassium, copper, gold, zinc, cadmium, and rubidium. Man-made thallium pollution sources are gaseous emissions from cement factories, coal-burning power plants, and metal sewers, and wastewater from ore-processing plants.

Carcinogenicity

No studies were located by ATSDR (1990) during the Agency's review of the toxicological literature regarding the carcinogenic effects of thallium in humans or animals via inhalation, oral, or dermal exposure routes.

Mutagenicity

No studies were located by ATSDR (1990) regarding the genotoxic effects of thallium in humans. However, thallium was genotoxic in mammalian cell and bacterial test systems: thallium induced dominant lethal mutations in male rats in vivo (Zasukhina et al., 1983), damaged rat embryo DNA in vitro (Zasukhina et al., 1981, 1983), enhanced viral-induced transformations of Syrian hamster embryo cells (Casto et al., 1979), and caused damage to bacterial DNA (Kanematsu et al., 1980).

Systemic Toxicity

From its historical uses as a depilatory or in pesticide formulations, a variety of cases of thallium intoxication have been recorded. Thallium is highly toxic, and its acute toxicity is characterized by gastrointestinal irritation, acute ascending paralysis, and psychic disturbances (Hammond and Beliles, 1980).

Thallium poisoning by the oral route causes a whole roster of acute effects (including nausea, vomiting, metallic taste, anorexia, mouth dryness, soreness of the gums, rhinorrhea, conjunctivitis, diarrhea, abdominal pain, peripheral neuropathy, etc.) that are reportedly duplicated under chronic exposure scenarios, along with changes in blood chemistry, hair loss, and damage to liver, kidney, intestines, and testes (Haley and Berndt, 1987). In several subchronic studies with laboratory animals where quantitative exposure information was known, oral exposures to thallium were associated with mortality and systemic effects in the respiratory, cardiovascular, gastrointestinal, hepatic, renal, dermal, ocular, neurological, and reproductive systems; NOAELS typically occurred at oral doses of 0.2 mg/kg bw per day of thallium (ATSDR, 1990).

Few data exist concerning the inhalation toxicity of thallium to humans or animals, and no data concerning dermal exposures were located by ATSDR (1990) during the

Agency's review of the toxicological literature. The inhalation data are insufficient either to attribute or to rule out effects of thallium exposure.

Toxicokinetics

Toxicokinetic studies of thallium are largely absent. The element may be absorbed via the gastrointestinal tract, and limited evidence suggests that the absorption may be complete (Barclay et al., 1953; Lie et al., 1960). Absorption through the skin may also occur (Hammond and Beliles, 1980). The element appears to distribute throughout the body, with limited evidence suggesting preferential accumulation in the kidney (Downs et al., 1960; Manzo et al., 1983), and to a lesser extent in the intestines, thyroid, testes, pancreas, skin, bone and spleen (Hammond and Beliles, 1980). The element has an estimated biological half-time of 3.3 days (Lie et al., 1960), although EPA (1980) estimates an excretion half-time of 21.7 days. Elimination was 32 percent in the feces and 21 percent in the urine by 8 days after gavage dosing of rats with 10 mg thallium (as $TlSO_4$)/kg bw (Lehman and Favari, 1985).

Toxicity Values

EPA has calculated an oral RfD for thallium carbonate at 0.00008 mg/kg-d, based on a subchronic oral study in rats that demonstrated increased levels of SGOT and LDH. An uncertainty factor of 3000 was used to adjust for extrapolation from subchronic to chronic and from animals to humans (10 each), for human variability (10), and for the lack of reproductive/developmental data (3).

EPA has calculated no inhalation RfC. No cancer slope factors have been derived. The lack of carcinogenicity information puts thallium carbonate into cancer classification Group D (unclassifiable as to carcinogenicity) (USEPA 2001).

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1,1,2-TRICHLOROETHANE

1,1,2-Trichloroethane (CAS Reg. No. 79-00-5), also known as vinyl trichloride, is a nonflammable liquid. 1,1,2-trichloroethane is used in the manufacture of 1,1-dichloroethene and as a solvent for fats, waxes, resins, and alkaloids; and in organic synthesis (Budavari et al. 1989, USEPA 1980).

Toxicokinetics

1,1,2-Trichloroethane is extensively absorbed after inhalation exposure. 1,1,2-Trichloroethane is also rapidly absorbed through the skin, probably because of the highly lipid soluble character of the compound (Kronevi et al. 1977). 1,1,2-Trichloroethane is rapidly absorbed and distributed in organs and tissues, and extensively metabolized. Major metabolites include chloroacetic acid, S-carboxymethylcysteine, and thiodiacetic acid. 1,1,2-Trichloroethane and/or its metabolites are primarily excreted through the lungs and urine (Morgan et al. 1970, 1972; Kronevi et al. 1977; Mitoma et al. 1985).

Systemic Toxicity

1,1,2-trichloroethane is a central nervous system depressant inducing narcosis; death results from respiratory arrest (ACGIH 1991). In mice, a concentration of 3750 ppm for 30 minutes produced central nervous system depression and significantly increased liver enzyme activity within 18 minutes and death in half the animals within 10 hours (Gehring 1968).

Carcinogenicity

1,1,2-Trichloroethane was assigned to weight-of-evidence group C, possible human carcinogen. Based on hepatocellular carcinomas and adrenal pheochromocytomas in one strain of mice. Carcinogenicity was not shown in rats. 1,1,2-Trichloroethane is structurally related to 1,2-dichloroethane, a probable human carcinogen (Group B2). Groups of $B6C3F_1$ mice or Osborne-Mendel rats (50 animals/sex/dose) were treated by gavage (NCI 1978). No effects on tumor development were noted in rats. Treated mice had significantly ($p < 0.01$) increased incidences of hepatocellular carcinomas.

Toxicity Values

EPA has established an oral RfD of 0.004 mg/kg-day (USEPA 2001). An uncertainty factor of 1,000 was incorporated in the calculations (10 for interspecies variation, 10 for intrahuman variability, and a factor of 10 for extrapolation to lifetime exposure from an intermediate exposure duration) (USEPA 2001).

EPA has established an oral cancer slope factor of 0.057 per mg/kg-day (USEPA 2001), based upon heptocellular carcinoma in mice (NCI 1978). An inhalation cancer slope factor of 0.056 per mg/kg-day based upon the oral cancer slope factor (USEPA 2001).

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TRICHLOROETHENE

Trichloroethene (trichloroethylene, C_2HCl_3, CAS Registry No. 79-01-6) is a colorless, nonflammable liquid with a sweetish odor resembling that of chloroform. It reacts violently with nitric acid and strong alkalies. All commercial grades of trichloroethene contain stabilizers to prevent autooxidation to phosgene or carbon monoxide gases. Most of the U.S. production of trichloroethene is used in vapor degreasing of fabricated metals, in the manufacture of polyvinyl chloride plastics, as a heat-transfer medium, in adhesives and paint strippers, and as a carrier solvent in textile dying and finishing (ACGIH 1991-1994).

The rate of pulmonary uptake doubles with exercise. Organs affected by excessive exposure to trichloroethene are the central nervous system, liver, kidney, heart, lung, and skin. In human volunteers, 50% of the dose is metabolized to trichloroethanol, 30% to minor metabolites primarily in the urine, and 8.4% appeared in feces, sweat, and saliva by the third day. Mice metabolize two and three times more trichloroethene than rats, depending on dose; and there is also considerable variability among individual humans in trichloroethylene metabolism (ACGIH 1991-1994).

Workers exposed to trichloroethene complain of headache, abnormal fatigue, gastric disturbance, irritability, anxiety, ocular and upper respiratory tract irritation, and alcohol intolerance; although no changes in performance tests occurred in one study, significant reductions in complex reaction time, memory, and perception were manifested in another. No children with congenital defects were born to women occupationally exposed over a 13-year period (Tola et al. 1980). In six epidemiologic studies, no evidence has surfaced to suggest that occupational trichloroethene exposure increases the incidence of cancer in humans (Axelson et al. 1978; Novotna et al. 1979; Spirtas et al. 1991; Stewart et al. 1991; Paddle 1983; Shindell and Ulrich 1985).

Noncarcinogenic toxicity values have been withdrawn (USEPA 2001) therefore these risks have not been quantified. EPA NCEA has developed a cancer slope factor of 0.006 per mg/kg/day, which has been used.

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VINYL CHLORIDE

Vinyl chloride, or chloroethylene (C_2H_3Cl, CAS Registry No. 75-01-4), is a colorless gas or liquid with a sweet odor. In the presence of moisture, it corrodes iron and steel. Formerly used as a refrigerant, aerosol propellant, drug and cosmetic ingredient, and extraction solvent, vinyl chloride is currently used in the manufacture of numerous products in building and construction and the automotive industry (in electrical wiring insulation and cables, piping, floor tiles, and industrial and household equipment); it is used in medical supplies, and is depended upon heavily by the rubber, paper, plastics, and glass industries.

No information is available on the potential human health effects associated with subchronic or chronic oral exposure to vinyl chloride. However, several studies have focused on the potential toxic effects in laboratory animals following subchronic or chronic oral exposure. These studies have identified hematotoxicity and hepatotoxicity as potential toxicity endpoints in animals following oral exposure. Two- to 3-year feeding studies in rats have identified NOAELs for hematotoxicity (hypercoagulation) in the range of 1.7-5.6 mg/kg/day and LOAELs for hepatotoxicity (cellular alteration) in the range of 0.018-1.8 mg/kg/day (Til et al. 1983, Feron et al. 1981).

No information is available on the potential human health effects associated with subchronic inhalation exposure to vinyl chloride. However, a large number of studies involving work-place exposures have focused on health effects associated with chronic inhalation exposure. These studies have identified a wide range of potential toxicity endpoints in humans resulting from inhalation exposure, including hepatotoxicity, respiratory toxicity, hematotoxicity, immunotoxicity, cardiovascular toxicity, dermal toxicity, musculoskeletal toxicity, neurotoxicity, and reproductive toxicity. However, these studies are all compromised by a failure to accurately report the vinyl chloride levels that workers were exposed to.

Many of the findings obtained from the human studies described above have been supported by animal studies. Three- to 12-month studies involving rats and mice have identified LOAELs for hepatotoxicity (hepatocellular hypertrophy with compression of sinusoids, fatty degeneration, increased liver weight, increased lipid droplets in and hyperplasia of hepatocytes) in the range of 10-3000 ppm (Wisniewska-Knypl et al. 1980, Torkelson et al. 1961, Bi et al. 1985, Schaffner 1978, Hehir et al. 1981). NOAELs for respiratory toxicity have been identified through 2 to 5 month rat and mouse studies as lying between 50 and 500 ppm (Hehir et al. 1981). NOAELs for hematotoxicity between 200 and 1000 ppm have been identified through rat and mouse studies of 2 to 6 months (Torkelson et al. 1961, Sharma and Gehring 1979). A LOAEL for immunotoxicity (increased spontaneous lymphocyte proliferation) of 10 ppm was identified in a 2 month mouse study (Sharma and Gehring 1979). Separate 3 and 6 month rat studies have identified 10 ppm as a NOAEL and LOAEL, respectively, for cardiovascular toxicity (increased heart weight) (Bi et al. 1985).

Three- to 12-month rat studies have identified NOAELs for male reproductive toxicity (testicular necrosis, decreased spermatogenesis, reduced male fertility, decreased testicular weight) of 10 and 50 ppm (Sokal et al. 1980, Short et al. 1977, Bi et al. 1985).

Because vinyl chloride has a relatively low boiling point (-13.4 deg. C), dermal exposure under the majority of environmental conditions is not likely to be significant since volatilization will occur before significant dermal uptake takes place. Therefore, no studies of toxicity in either humans or animals following subchronic or chronic dermal exposure to vinyl chloride have been completed.

Vinyl chloride is classified by EPA as a known human carcinogen (Group A) (USEPA 2001). No human studies investigating a link between oral exposure to vinyl chloride and an excess incidence of cancer have been conducted. However, rat studies have identified cancer effect levels (CELs), or the dosing level at which significant increases in cancer rates over controls occur, for hepatic tumors in rats following oral exposure to vinyl chloride in the range of 1.7-1.8 mg/kg/day (Feron et al. 1981, Til et al. 1983).

Human epidemiological studies focusing on workers have identified a wide range of possible cancers linked to chronic inhalation of vinyl chloride including cancers of the liver, brain, lung, and blood. However, these studies all lack detailed exposure data. Inhalation rat, mouse, and hamster studies have identified CELs for hepatic tumors between of 1 and 500 ppm (Hong et al. 1981, Drew et al. 1983, Lee et al. 1978, Maltoni et al. 1981, Bi et al. 1985). Drew et al. (1983) identified a CEL of 50 ppm for peritoneal cancer in a mouse study. CELs between 50 and 100 ppm were identified for lung cancer through mouse studies (Adkins et al. 1986, Suzuki 1982, Drew et al. 1983, Lee et al. 1978). CELs for mammary gland cancer of between 5 and 50 ppm were identified through rat and mouse studies (Hong et al. 1981, Maltoni et al. 1981).

Toxicity Values

EPA has established a oral slope factor for vinyl chloride of 1.5 mg/kg/day based on a rat feeding study (Feron, Hendriksen, Speek et al. 1981) that identified an increased incidence of lung and liver tumors (HEAST, 1994).

EPA has established an unverified inhalation slope factor for vinyl chloride of 3.2×10^{-2} mg/kg/day based on an rat inhalation study (Maltoni 1981) that identified an increased incidence of liver tumors (HEAST, 1994).

USEPA (2001) has provided an oral RfD of 0.003 mg/kg/day based on a rat hepatic endpoint, which has been used in this risk assessment.

Both in vivo and in vitro genotoxicity assays have been conducted using vinyl chloride. In vivo studies have identified chromosomal aberrations in rats (Anderson and Richardson 1981), chromosomal aberrations in human lymphocyte (Hansteen et al 1978, Kucerova et al. 1979, Purchase et al. 1978, Ducatman et al. 1975, Anderson et al. 1980, Fucic et al. 1990, Funes-Cravioto et al. 1975, Hrivnak et al. 1990) sister chromatid

exchange in human lymphocytes (Kucerova et al. 1979), and DNA alkylation in rats and mice (Laib et al. 1989, Gwinner et al. 1983, Singer et al. 1987, Bolt et al. 1986, Ciroussel et al. 1990, Eberle et al. 1989, Osterman-Golkar et al. 1977, Walles et al. 1988, Green and Hathway 1978). In vitro assays have identified reverse mutation in Salmonella typhimurium (Rannug et al. 1974, Bartsch et al. 1975, 1976, Andrews et al. 1976, Simmon et al. 1977, Elmore et al. 1976, Poncelet et al. 1980, de Meester et al. 1980, Victorin and Stahlberg 1988, McCann et al. 1975, Rannug et al. 1976), reverse mutation in Escherichia coli (Jacobsen et al. 1989), forward mutation in Schizo saccharomyces pombe (Loprieno et al. 1977) and Chinese hamster ovary cells (Huberman et al. 1975), RNA alkylation in rat liver microsomes (Laib and Bolt 1977), and inhibition of DNA synthesis in QT6 (avian cells) (Kandala et al. 1990).

Human studies have demonstrated that 42% of vinyl chloride is retained when administered through inhalation (Krajewski et al. 1980). Animal study data supports the contention that vinyl chloride is rapidly absorbed following inhalation, however, animal data is not complete enough to allow a quantitative estimate of the amount retained. Animal data has shown that vinyl chloride levels in animal blood fall rapidly once exposure ceases (Withey 1976). No study data is available characterizing the rate of uptake for vinyl chloride in humans following oral exposure, however animal studies have shown that vinyl chloride when administered through this route, is readily absorbed (Withey 1976). Human studies investigating the rate of absorption following dermal exposure are not available and animal studies have shown that dermal uptake of vinyl chloride vapor (the state that vinyl chloride is most likely to be encountered in environmentally) is insignificant relative to other exposure routes (Hefner et al. 1975).

Following inhalation exposure, animal studies have shown that vinyl chloride is rapidly distributed to a large number of organ and tissue types throughout the body. However, vinyl chloride is not stored in these organs/tissues for extended periods because of its rapid metabolism and excretion (Buchter et al. 1977). Vinyl chloride is distributed to the following tissues/organs in rats 72 hours after inhalation exposure (in order of decreasing concentration): liver, kidney, skin, lung, muscle, carcass, plasma, fat (Watanabe et al. 1976). Placental transfer of vinyl chloride to fetal blood and amniotic fluid is rapid in rats (Ungvary et al. 1978). 72 hours after oral administration to rats, the highest concentrations of vinyl chloride were found in the liver with lesser concentrations found in the skin, plasma, muscle, lung, fat, and carcass (Watanabe et al. 1976).

One study was identified that examined the metabolism of vinyl chloride in humans following inhalation exposure. In this study, analysis of liver specimens showed electrophilic vinyl chloride metabolites resulting from cytochrome P-450 activity to be mutagenic to Salmonella typhimurium (Sabadie et al. 1980). Extensive experimentation using laboratory animals has focused on the metabolism of vinyl chloride following inhalation. These studies have demonstrated that vinyl chloride metabolism following inhalation exposure is a dose-dependent saturable process (Hefner et al. 1975) that probably involves primarily a mixed-function oxidase pathway (Bolt et al. 1977). Animal studies suggest that the metabolism of vinyl chloride following oral exposure is similar to that identified for inhalation exposure (ATSDR 1990). No animal or human

studies were identified that examined the metabolic properties for vinyl chloride following oral dermal exposure.

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Attachment B

Risk Calculation Tables

Table B-1
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO EXPOSURES TO TOTAL SOIL INGESTION -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Incidental Ingestion of Total Soil**

Ingestion Rate = CR	50	mg/day
Exposure Frequency = EF	150	day/yr
Exposure Duration = ED	9	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	3,285	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	1.00E-06	kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	5.68E-05	1.67E-11	2.14E-12	NA	1.50E+05	--	3.22E-07
Inorganics							
ALUMINUM	5.57E+03	1.64E-03	2.10E-04	1.00E+00	NA	1.64E-03	--
ANTIMONY	9.61E-01	2.82E-07	3.63E-08	4.00E-04	NA	7.05E-04	--
ARSENIC	2.45E+00	7.19E-07	9.25E-08	3.00E-04	1.50E+00	2.40E-03	1.39E-07
BARIUM	7.81E+01	2.29E-05	2.95E-06	7.00E-02	NA	3.28E-04	--
COPPER	4.28E+01	1.26E-05	1.62E-06	4.00E-02	NA	3.14E-04	--
MANGANESE	1.66E+02	4.87E-05	6.27E-06	2.00E-02	NA	2.44E-03	--
THALLIUM	2.76E-01	8.10E-08	1.04E-08	7.00E-05	NA	1.16E-03	--
PAHs							
BENZ(A)ANTHRACENE	3.19E-01	9.36E-08	1.20E-08	NA	7.30E-01	--	8.79E-09
BENZO[A]PYRENE	2.89E-01	8.48E-08	1.09E-08	NA	7.30E+00	--	7.96E-08
BENZO(B)FLUORANTHENE	4.27E-01	1.25E-07	1.61E-08	NA	7.30E-01	--	1.18E-08
DIBENZ[A, H]ANTHRACENE	1.51E-01	4.43E-08	5.70E-09	NA	7.30E+00	--	4.16E-08
INDENO(1,2,3-CD)PYRENE	2.31E-01	6.78E-08	8.72E-09	NA	7.30E-01	--	6.36E-09
2-METHYLNAPHTHALENE	1.19E+00	3.49E-07	4.49E-08	2.00E-02	NA	1.75E-05	--
NAPHTHALENE	3.93E-01	1.15E-07	1.48E-08	2.00E-02	NA	5.77E-06	--
Cumulative Risk						9.00E-03	6.08E-07

Table B-2
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO EXPOSURES TO TOTAL SOIL INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Incidental Ingestion of Total Soil**

Ingestion Rate = CR	100 mg/day
Exposure Frequency = EF	150 day/yr
Exposure Duration = ED	24 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	8,760 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.91E-04	1.12E-10	3.84E-11	NA	1.50E+05	--	5.77E-06
Inorganics							
ALUMINUM	6.61E+03	3.88E-03	1.33E-03	1.00E+00	NA	3.88E-03	--
ANTIMONY	1.32E+00	7.75E-07	2.66E-07	4.00E-04	NA	1.94E-03	--
ARSENIC	8.64E+00	5.07E-06	1.74E-06	3.00E-04	1.50E+00	1.69E-02	2.61E-06
BARIUM	8.50E+01	4.99E-05	1.71E-05	7.00E-02	NA	7.13E-04	--
COPPER	5.56E+01	3.26E-05	1.12E-05	4.00E-02	NA	8.16E-04	--
MANGANESE	2.00E+02	1.17E-04	4.03E-05	2.00E-02	NA	5.87E-03	--
THALLIUM	3.36E-01	1.97E-07	6.76E-08	7.00E-05	NA	2.82E-03	--
PAHs							
BENZ(A)ANTHRACENE	4.27E-01	2.51E-07	8.59E-08	NA	7.30E-01	--	6.27E-08
BENZO[A]PYRENE	4.18E-01	2.45E-07	8.41E-08	NA	7.30E+00	--	6.14E-07
BENZO(B)FLUORANTHENE	5.17E-01	3.04E-07	1.04E-07	NA	7.30E-01	--	7.60E-08
DIBENZ[A, H]ANTHRACENE	3.11E-01	1.83E-07	6.26E-08	NA	7.30E+00	--	4.57E-07
INDENO(1,2,3-CD)PYRENE	3.51E-01	2.06E-07	7.07E-08	NA	7.30E-01	--	5.16E-08
2-METHYLNAPHTHALENE	7.70E-01	4.52E-07	1.55E-07	2.00E-02	NA	2.26E-05	--
NAPHTHALENE	4.91E-01	2.88E-07	9.88E-08	2.00E-02	NA	1.44E-05	--
Cumulative Risk						3.30E-02	9.64E-06

Table B-3
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO EXPOSURE TO DERMAL CONTACT WITH TOTAL SOIL -- CENTRAL TENDENCY SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Dermal Contact with Total Soil**

Surface Area for Contact = SA	6,200	cm^2/event
Adherence Factor = AF	0.01	mg/cm^2
Absorption Factor = ABS	chemical-specific	
Exposure Frequency = EF	150	event/yr
Exposure Duration = ED	9	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	3,285	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	1.00E-06	kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	5.68E-05	6.20E-13	7.97E-14	NA	1.50E+05	--	1.20E-08
Inorganics							
ALUMINUM	5.57E+03	0.00E+00	0.00E+00	1.00E+00	NA	0.00E+00	--
ANTIMONY	9.61E-01	0.00E+00	0.00E+00	6.00E-05	NA	0.00E+00	--
ARSENIC	2.45E+00	2.68E-08	3.44E-09	3.00E-04	1.50E+00	8.92E-05	5.16E-09
BARIUM	7.81E+01	0.00E+00	0.00E+00	4.90E-03	NA	0.00E+00	--
COPPER	4.28E+01	0.00E+00	0.00E+00	4.00E-02	NA	0.00E+00	--
MANGANESE	1.66E+02	0.00E+00	0.00E+00	8.00E-04	NA	0.00E+00	--
THALLIUM	2.76E-01	0.00E+00	0.00E+00	7.00E-05	NA	0.00E+00	--
PAHs							
BENZ(A)ANTHRACENE	3.19E-01	1.51E-08	1.94E-09	NA	7.30E-01	--	1.42E-09
BENZO[A]PYRENE	2.89E-01	1.37E-08	1.76E-09	NA	7.30E+00	--	1.28E-08
BENZO(B)FLUORANTHENE	4.27E-01	2.02E-08	2.60E-09	NA	7.30E-01	--	1.90E-09
DIBENZ[A, H]ANTHRACENE	1.51E-01	7.15E-09	9.19E-10	NA	7.30E+00	--	6.71E-09
INDENO(1,2,3-CD)PYRENE	2.31E-01	1.09E-08	1.41E-09	NA	7.30E-01	--	1.03E-09
2-METHYLNAPHTHALENE	1.19E+00	5.63E-08	7.24E-09	2.00E-02	NA	2.82E-06	--
NAPHTHALENE	3.93E-01	1.86E-08	2.39E-09	2.00E-02	NA	9.30E-07	--
Cumulative Risk						9.29E-05	4.10E-08

Table B-4
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO EXPOSURE TO DERMAL CONTACT WITH TOTAL SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: Site 16

* RME- Dermal Contact with Total Soil

Surface Area for Contact = SA	8,000 cm^2/event
Adherence Factor = AF	0.07 mg/cm^2
Absorption Factor = ABS	chemical-specific
Exposure Frequency = EF	150 event/yr
Exposure Duration = ED	24 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	8,760 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.91E-04	1.88E-11	6.46E-12	NA	1.50E+05	--	9.69E-07
Inorganics							
ALUMINUM	6.61E+03	0.00E+00	0.00E+00	1.00E+00	NA	0.00E+00	--
ANTIMONY	1.32E+00	0.00E+00	0.00E+00	6.00E-05	NA	0.00E+00	--
ARSENIC	8.64E+00	8.52E-07	2.92E-07	3.00E-04	1.50E+00	2.84E-03	4.38E-07
BARIUM	8.50E+01	0.00E+00	0.00E+00	4.90E-03	NA	0.00E+00	--
COPPER	5.56E+01	0.00E+00	0.00E+00	4.00E-02	NA	0.00E+00	--
MANGANESE	2.00E+02	0.00E+00	0.00E+00	8.00E-04	NA	0.00E+00	--
THALLIUM	3.36E-01	0.00E+00	0.00E+00	7.00E-05	NA	0.00E+00	--
PAHs							
BENZ(A)ANTHRACENE	4.27E-01	1.82E-07	6.26E-08	NA	7.30E-01	--	4.57E-08
BENZO[A]PYRENE	4.18E-01	1.79E-07	6.13E-08	NA	7.30E+00	--	4.47E-07
BENZO(B)FLUORANTHENE	5.17E-01	2.21E-07	7.58E-08	NA	7.30E-01	--	5.53E-08
DIBENZ[A, H]ANTHRACENE	3.11E-01	1.33E-07	4.56E-08	NA	7.30E+00	--	3.33E-07
INDENO(1,2,3-CD)PYRENE	3.51E-01	1.50E-07	5.14E-08	NA	7.30E-01	--	3.75E-08
2-METHYLNAPHTHALENE	7.70E-01	3.29E-07	1.13E-07	2.00E-02	NA	1.65E-05	--
NAPHTHALENE	4.91E-01	2.10E-07	7.19E-08	2.00E-02	NA	1.05E-05	--
Cumulative Risk						**2.87E-03**	**2.33E-06**

Table B-5
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO INHALATION OF PARTICULATES FROM TOTAL SOIL -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

* CTE- Inhalation of Particles from Total Soil

Inhalation Rate = IR	0.55 m3/hr
Exposure Time = ET	10 hr/day
Exposure Frequency = EF	150 day/yr
Exposure Duration = ED	9 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	2,555 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * IR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/m3)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	4.30E-14	1.79E-15	1.79E-16	NA	1.50E+05	--	2.68E-11
Inorganics							
ALUMINUM	4.22E-06	1.75E-07	1.75E-08	1.00E-03	NA	1.75E-04	--
ANTIMONY	7.28E-10	3.02E-11	3.02E-12	NA	NA	--	--
ARSENIC	1.86E-09	7.71E-11	7.71E-12	NA	1.51E+01	--	1.16E-10
BARIUM	5.92E-08	2.46E-09	2.46E-10	1.40E-04	NA	1.75E-05	--
COPPER	3.24E-08	1.35E-09	1.35E-10	NA	NA	--	--
MANGANESE	1.26E-07	5.22E-09	5.22E-10	1.43E-05	NA	3.65E-04	--
THALLIUM	2.09E-10	8.68E-12	8.68E-13	NA	NA	--	--
PAHs							
BENZ(A)ANTHRACENE	2.42E-10	1.00E-11	1.00E-12	NA	3.10E-01	--	3.11E-13
BENZO[A]PYRENE	2.19E-10	9.09E-12	9.09E-13	NA	3.10E+00	--	2.82E-12
BENZO(B)FLUORANTHENE	3.23E-10	1.34E-11	1.34E-12	NA	3.10E-01	--	4.16E-13
DIBENZ[A, H]ANTHRACENE	1.14E-10	4.75E-12	4.75E-13	NA	NA	--	--
INDENO(1,2,3-CD)PYRENE	1.75E-10	7.27E-12	7.27E-13	NA	3.10E-01	--	2.25E-13
2-METHYLNAPHTHALENE	9.02E-10	3.74E-11	3.74E-12	NA	NA	--	--
NAPHTHALENE	2.98E-10	1.24E-11	1.24E-12	9.00E-04	NA	1.37E-08	--
Cumulative Risk						**5.58E-04**	**1.47E-10**

Table B-6
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO INHALATION OF PARTICULATES FROM TOTAL SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

* RME- Inhalation of Particles from Total Soil

Inhalation Rate = IR	0.625 m3/hr
Exposure Time = ET	24 hr/day
Exposure Frequency = EF	150 day/yr
Exposure Duration = ED	24 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	8,760 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * IR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/m3)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.45E-13	1.27E-14	4.37E-15	NA	1.50E+05	--	6.55E-10
Inorganics							
ALUMINUM	5.01E-06	4.41E-07	1.51E-07	1.00E-03	NA	4.41E-04	--
ANTIMONY	1.00E-09	8.81E-11	3.02E-11	NA	NA	--	--
ARSENIC	6.55E-09	5.76E-10	1.98E-10	NA	1.51E+01	--	2.98E-09
BARIUM	6.44E-08	5.67E-09	1.94E-09	1.40E-04	NA	4.05E-05	--
COPPER	4.21E-08	3.71E-09	1.27E-09	NA	NA	--	--
MANGANESE	1.52E-07	1.33E-08	4.57E-09	1.43E-05	NA	9.33E-04	--
THALLIUM	2.55E-10	2.24E-11	7.69E-12	NA	NA	--	--
PAHs							
BENZ(A)ANTHRACENE	3.23E-10	2.85E-11	9.77E-12	NA	3.10E-01	--	3.03E-12
BENZO[A]PYRENE	3.17E-10	2.79E-11	9.56E-12	NA	3.10E+00	--	2.96E-11
BENZO(B)FLUORANTHENE	3.92E-10	3.45E-11	1.18E-11	NA	3.10E-01	--	3.67E-12
DIBENZ[A, H]ANTHRACENE	2.36E-10	2.07E-11	7.11E-12	NA	NA	--	--
INDENO(1,2,3-CD)PYRENE	2.66E-10	2.34E-11	8.03E-12	NA	3.10E-01	--	2.49E-12
2-METHYLNAPHTHALENE	5.83E-10	5.14E-11	1.76E-11	NA	NA	--	--
NAPHTHALENE	3.72E-10	3.28E-11	1.12E-11	9.00E-04	NA	3.64E-08	--
Cumulative Risk						**1.41E-03**	**3.68E-09**

Table B-7
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO EXPOSURES TO TOTAL SOIL INGESTION -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: Site 16

*** CTE- Incidental Ingestion of Total Soil**

Ingestion Rate = CR	100 mg/day
Exposure Frequency = EF	150 day/yr
Exposure Duration = ED	2 yr
Body Weight = BW	15 kg
Averaging Time (Noncancer) = AT	730 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	5.68E-05	1.56E-10	4.45E-12	NA	1.50E+05	--	6.67E-07
Inorganics							
ALUMINUM	5.57E+03	1.53E-02	4.36E-04	1.00E+00	NA	1.53E-02	--
ANTIMONY	9.61E-01	2.63E-06	7.52E-08	4.00E-04	NA	6.58E-03	--
ARSENIC	2.45E+00	6.71E-06	1.92E-07	3.00E-04	1.50E+00	2.24E-02	2.88E-07
BARIUM	7.81E+01	2.14E-04	6.11E-06	7.00E-02	NA	3.06E-03	--
COPPER	4.28E+01	1.17E-04	3.35E-06	4.00E-02	NA	2.93E-03	--
MANGANESE	1.66E+02	4.55E-04	1.30E-05	2.00E-02	NA	2.27E-02	--
THALLIUM	2.76E-01	7.56E-07	2.16E-08	7.00E-05	NA	1.08E-02	--
PAHs							
BENZ(A)ANTHRACENE	3.19E-01	8.74E-07	2.50E-08	NA	7.30E-01	--	1.82E-08
BENZO[A]PYRENE	2.89E-01	7.92E-07	2.26E-08	NA	7.30E+00	--	1.65E-07
BENZO(B)FLUORANTHENE	4.27E-01	1.17E-06	3.34E-08	NA	7.30E-01	--	2.44E-08
DIBENZ[A, H]ANTHRACENE	1.51E-01	4.14E-07	1.18E-08	NA	7.30E+00	--	8.63E-08
INDENO(1,2,3-CD)PYRENE	2.31E-01	6.33E-07	1.81E-08	NA	7.30E-01	--	1.32E-08
2-METHYLNAPHTHALENE	1.19E+00	3.26E-06	9.32E-08	2.00E-02	NA	1.63E-04	--
NAPHTHALENE	3.93E-01	1.08E-06	3.08E-08	2.00E-02	NA	5.38E-05	--
Cumulative Risk						8.40E-02	1.26E-06

Table B-8
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO EXPOSURES TO TOTAL SOIL INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Incidental Ingestion of Total Soil**

Ingestion Rate = CR	200 mg/day
Exposure Frequency = EF	150 day/yr
Exposure Duration = ED	6 yr
Body Weight = BW	15 kg
Averaging Time (Noncancer) = AT	2,190 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.91E-04	1.05E-09	8.97E-11	NA	1.50E+05	--	1.35E-05
Inorganics							
ALUMINUM	6.61E+03	3.62E-02	3.10E-03	1.00E+00	NA	3.62E-02	--
ANTIMONY	1.32E+00	7.23E-06	6.20E-07	4.00E-04	NA	1.81E-02	--
ARSENIC	8.64E+00	4.73E-05	4.06E-06	3.00E-04	1.50E+00	1.58E-01	6.09E-06
BARIUM	8.50E+01	4.66E-04	3.99E-05	7.00E-02	NA	6.65E-03	--
COPPER	5.56E+01	3.05E-04	2.61E-05	4.00E-02	NA	7.62E-03	--
MANGANESE	2.00E+02	1.10E-03	9.39E-05	2.00E-02	NA	5.48E-02	--
THALLIUM	3.36E-01	1.84E-06	1.58E-07	7.00E-05	NA	2.63E-02	--
PAHs							
BENZ(A)ANTHRACENE	4.27E-01	2.34E-06	2.01E-07	NA	7.30E-01	--	1.46E-07
BENZO[A]PYRENE	4.18E-01	2.29E-06	1.96E-07	NA	7.30E+00	--	1.43E-06
BENZO(B)FLUORANTHENE	5.17E-01	2.83E-06	2.43E-07	NA	7.30E-01	--	1.77E-07
DIBENZ[A, H]ANTHRACENE	3.11E-01	1.70E-06	1.46E-07	NA	7.30E+00	--	1.07E-06
INDENO(1,2,3-CD)PYRENE	3.51E-01	1.92E-06	1.65E-07	NA	7.30E-01	--	1.20E-07
2-METHYLNAPHTHALENE	7.70E-01	4.22E-06	3.62E-07	2.00E-02	NA	2.11E-04	--
NAPHTHALENE	4.91E-01	2.69E-06	2.31E-07	2.00E-02	NA	1.35E-04	--
Cumulative Risk						**3.08E-01**	**2.25E-05**

Table B-9
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO EXPOSURE TO DERMAL CONTACT WITH TOTAL SOIL -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: Site 16

* CTE- Dermal Contact with Total Soil

Surface Area for Contact = SA	2,900 cm^2/event
Adherence Factor = AF	0.06 mg/cm^2
Absorption Factor = ABS	chemical specific
Exposure Frequency = EF	150 event/yr
Exposure Duration = ED	2 yr
Body Weight = BW	15 kg
Averaging Time (Noncancer) = AT	730 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	5.68E-05	8.12E-12	2.32E-13	NA	1.50E+05	--	3.48E-08
Inorganics							
ALUMINUM	5.57E+03	0.00E+00	0.00E+00	1.00E+00	NA	0.00E+00	--
ANTIMONY	9.61E-01	0.00E+00	0.00E+00	6.00E-05	NA	0.00E+00	--
ARSENIC	2.45E+00	3.50E-07	1.00E-08	3.00E-04	1.50E+00	1.17E-03	1.50E-08
BARIUM	7.81E+01	0.00E+00	0.00E+00	4.90E-03	NA	0.00E+00	--
COPPER	4.28E+01	0.00E+00	0.00E+00	4.00E-02	NA	0.00E+00	--
MANGANESE	1.66E+02	0.00E+00	0.00E+00	8.00E-04	NA	0.00E+00	--
THALLIUM	2.76E-01	0.00E+00	0.00E+00	7.00E-05	NA	0.00E+00	--
PAHs							
BENZ(A)ANTHRACENE	3.19E-01	1.98E-07	5.65E-09	NA	7.30E-01	--	4.12E-09
BENZO[A]PYRENE	2.89E-01	1.79E-07	5.12E-09	NA	7.30E+00	--	3.74E-08
BENZO(B)FLUORANTHENE	4.27E-01	2.65E-07	7.56E-09	NA	7.30E-01	--	5.52E-09
DIBENZ[A, H]ANTHRACENE	1.51E-01	9.36E-08	2.67E-09	NA	7.30E+00	--	1.95E-08
INDENO(1,2,3-CD)PYRENE	2.31E-01	1.43E-07	4.09E-09	NA	7.30E-01	--	2.99E-09
2-METHYLNAPHTHALENE	1.19E+00	7.37E-07	2.11E-08	2.00E-02	NA	3.69E-05	--
NAPHTHALENE	3.93E-01	2.44E-07	6.96E-09	2.00E-02	NA	1.22E-05	--
Cumulative Risk						1.22E-03	1.19E-07

Table B-10

TESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO EXPOSURE TO DERMAL CONTACT WITH TOTAL SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: Site 16

* RME- Dermal Contact with Total Soil

Surface Area for Contact = SA	3,600 cm^2/event
Adherence Factor = AF	0.2 mg/cm^2
Absorption Factor = ABS	chemical specific
Exposure Frequency = EF	150 event/yr
Exposure Duration = ED	6 yr
Body Weight = BW	15 kg
Averaging Time (Noncancer) = AT	2,190 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.91E-04	1.13E-10	9.69E-12	NA	1.50E+05	--	1.45E-06
Inorganics							
ALUMINUM	6.61E+03	0.00E+00	0.00E+00	1.00E+00	NA	0.00E+00	--
ANTIMONY	1.32E+00	0.00E+00	0.00E+00	6.00E-05	NA	0.00E+00	--
ARSENIC	8.64E+00	5.11E-06	4.38E-07	3.00E-04	1.50E+00	1.70E-02	6.57E-07
BARIUM	8.50E+01	0.00E+00	0.00E+00	4.90E-03	NA	0.00E+00	--
COPPER	5.56E+01	0.00E+00	0.00E+00	4.00E-02	NA	0.00E+00	--
MANGANESE	2.00E+02	0.00E+00	0.00E+00	8.00E-04	NA	0.00E+00	--
THALLIUM	3.36E-01	0.00E+00	0.00E+00	7.00E-05	NA	0.00E+00	--
PAHs							
BENZ(A)ANTHRACENE	4.27E-01	1.09E-06	9.39E-08	NA	7.30E-01	--	6.85E-08
BENZO[A]PYRENE	4.18E-01	1.07E-06	9.19E-08	NA	7.30E+00	--	6.71E-07
BENZO(B)FLUORANTHENE	5.17E-01	1.33E-06	1.14E-07	NA	7.30E-01	--	8.30E-08
DIBENZ[A, H]ANTHRACENE	3.11E-01	7.98E-07	6.84E-08	NA	7.30E+00	--	4.99E-07
INDENO(1,2,3-CD)PYRENE	3.51E-01	9.00E-07	7.72E-08	NA	7.30E-01	--	5.63E-08
2-METHYLNAPHTHALENE	7.70E-01	1.97E-06	1.69E-07	2.00E-02	NA	9.87E-05	--
NAPHTHALENE	4.91E-01	1.26E-06	1.08E-07	2.00E-02	NA	6.30E-05	--
Cumulative Risk						1.72E-02	3.49E-06

Table B-11
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO INHALATION OF PARTICULATES FROM TOTAL SOIL -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: Site 16

* CTE- Inhalation of Particles from Total Soil

Inhalation Rate = IR	0.416666667 m3/hr
Exposure Time = ET	10 hr/day
Exposure Frequency = EF	150 day/yr
Exposure Duration = ED	2 yr
Body Weight = BW	15 kg
Averaging Time (Noncancer) = AT	730 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * IR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/m3)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	4.30E-14	4.91E-15	1.40E-16	NA	1.50E+05	--	2.11E-11
Inorganics							
ALUMINUM	4.22E-06	4.82E-07	1.38E-08	1.00E-03	NA	4.82E-04	--
ANTIMONY	7.28E-10	8.31E-11	2.37E-12	NA	NA	--	--
ARSENIC	1.86E-09	2.12E-10	6.05E-12	NA	1.51E+01	--	9.14E-11
BARIUM	5.92E-08	6.75E-09	1.93E-10	1.40E-04	NA	4.82E-05	--
COPPER	3.24E-08	3.70E-09	1.06E-10	NA	NA	--	--
MANGANESE	1.26E-07	1.44E-08	4.10E-10	1.43E-05	NA	1.00E-03	--
THALLIUM	2.09E-10	2.39E-11	6.82E-13	NA	NA	--	--
PAHs							
BENZ(A)ANTHRACENE	2.42E-10	2.76E-11	7.88E-13	NA	3.10E-01	--	2.44E-13
BENZO[A]PYRENE	2.19E-10	2.50E-11	7.14E-13	NA	3.10E+00	--	2.21E-12
BENZO(B)FLUORANTHENE	3.23E-10	3.69E-11	1.06E-12	NA	3.10E-01	--	3.27E-13
DIBENZ[A, H]ANTHRACENE	1.14E-10	1.31E-11	3.73E-13	NA	NA	--	--
INDENO(1,2,3-CD)PYRENE	1.75E-10	2.00E-11	5.71E-13	NA	3.10E-01	--	1.77E-13
2-METHYLNAPHTHALENE	9.02E-10	1.03E-10	2.94E-12	NA	NA	--	--
NAPHTHALENE	2.98E-10	3.40E-11	9.71E-13	9.00E-04	NA	3.78E-08	--
Cumulative Risk						1.53E-03	1.15E-10

Table B-12
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO INHALATION OF PARTICULATES FROM TOTAL SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

* RME- Inhalation of Particles from Total Soil

Inhalation Rate = IR	0.416666667 m3/hr
Exposure Time = ET	24 hr/day
Exposure Frequency = EF	350 day/yr
Exposure Duration = ED	6 yr
Body Weight = BW	15 kg
Averaging Time (Noncancer) = AT	2,190 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * IR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/m3)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.45E-13	9.25E-14	7.93E-15	NA	1.50E+05	--	1.19E-09
Inorganics							
ALUMINUM	5.01E-06	3.20E-06	2.74E-07	1.00E-03	NA	3.20E-03	--
ANTIMONY	1.00E-09	6.39E-10	5.48E-11	NA	NA	--	--
ARSENIC	6.55E-09	4.18E-09	3.59E-10	NA	1.51E+01	--	5.42E-09
BARIUM	6.44E-08	4.12E-08	3.53E-09	1.40E-04	NA	2.94E-04	--
COPPER	4.21E-08	2.69E-08	2.31E-09	NA	NA	--	--
MANGANESE	1.52E-07	9.69E-08	8.30E-09	1.43E-05	NA	6.77E-03	--
THALLIUM	2.55E-10	1.63E-10	1.39E-11	NA	NA	--	--
PAHs							
BENZ(A)ANTHRACENE	3.23E-10	2.07E-10	1.77E-11	NA	3.10E-01	--	5.49E-12
BENZO[A]PYRENE	3.17E-10	2.02E-10	1.74E-11	NA	3.10E+00	--	5.38E-11
BENZO(B)FLUORANTHENE	3.92E-10	2.50E-10	2.15E-11	NA	3.10E-01	--	6.65E-12
DIBENZ[A, H]ANTHRACENE	2.36E-10	1.51E-10	1.29E-11	NA	NA	--	--
INDENO(1,2,3-CD)PYRENE	2.66E-10	1.70E-10	1.46E-11	NA	3.10E-01	--	4.52E-12
2-METHYLNAPHTHALENE	5.83E-10	3.73E-10	3.20E-11	NA	NA	--	--
NAPHTHALENE	3.72E-10	2.38E-10	2.04E-11	9.00E-04	NA	2.64E-07	--
Cumulative Risk						1.03E-02	6.68E-09

Table B-13
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RECREATIONAL USERS DUE TO EXPOSURES TO SURFACE SOIL INGESTION -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Incidental Ingestion of Surface Soil**

Ingestion Rate = CR	50 mg/day
Exposure Frequency = EF	48 day/yr
Exposure Duration = ED	9 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	3,285 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.13E-05	1.06E-12	1.36E-13	NA	1.50E+05	--	2.05E-08
Inorganics							
ALUMINUM	4.61E+03	4.33E-04	5.57E-05	1.00E+00	NA	4.33E-04	--
ARSENIC	1.87E+00	1.76E-07	2.26E-08	3.00E-04	1.50E+00	5.86E-04	3.39E-08
MANGANESE	1.45E+02	1.36E-05	1.75E-06	2.00E-02	NA	6.81E-04	--
THALLIUM	2.14E-01	2.01E-08	2.58E-09	7.00E-05	NA	2.87E-04	--
PAHs							
BENZ(A)ANTHRACENE	3.39E-01	3.18E-08	4.09E-09	NA	7.30E-01	--	2.99E-09
BENZO[A]PYRENE	2.59E-01	2.43E-08	3.13E-09	NA	7.30E+00	--	2.28E-08
BENZO(B)FLUORANTHENE	4.88E-01	4.58E-08	5.89E-09	NA	7.30E-01	--	4.30E-09
DIBENZ[A, H]ANTHRACENE	1.25E-01	1.17E-08	1.51E-09	NA	7.30E+00	--	1.10E-08
INDENO(1,2,3-CD)PYRENE	2.20E-01	2.07E-08	2.66E-09	NA	7.30E-01	--	1.94E-09
Cumulative Risk						**1.99E-03**	**9.74E-08**

Table B-14
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RECREATIONAL USERS DUE TO EXPOSURES TO SURFACE SOIL INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Incidental Ingestion of Surface Soil**

Ingestion Rate = CR	100 mg/day
Exposure Frequency = EF	96 day/yr
Exposure Duration = ED	24 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	8,760 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	3.79E-05	1.42E-11	4.88E-12	NA	1.50E+05	--	7.32E-07
Inorganics							
ALUMINUM	5.97E+03	2.24E-03	7.69E-04	1.00E+00	NA	2.24E-03	--
ARSENIC	2.67E+00	1.00E-06	3.44E-07	3.00E-04	1.50E+00	3.34E-03	5.16E-07
MANGANESE	1.85E+02	6.95E-05	2.38E-05	2.00E-02	NA	3.48E-03	--
THALLIUM	3.07E-01	1.15E-07	3.95E-08	7.00E-05	NA	1.65E-03	--
PAHs							
BENZ(A)ANTHRACENE	5.61E-01	2.11E-07	7.23E-08	NA	7.30E-01	--	5.28E-08
BENZO[A]PYRENE	4.68E-01	1.76E-07	6.03E-08	NA	7.30E+00	--	4.40E-07
BENZO(B)FLUORANTHENE	6.78E-01	2.55E-07	8.73E-08	NA	7.30E-01	--	6.38E-08
DIBENZ[A, H]ANTHRACENE	3.81E-01	1.43E-07	4.91E-08	NA	7.30E+00	--	3.58E-07
INDENO(1,2,3-CD)PYRENE	4.17E-01	1.57E-07	5.37E-08	NA	7.30E-01	--	3.92E-08
Cumulative Risk						**1.07E-02**	**2.20E-06**

Table B-15

ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RECREATIONAL USERS DUE TO EXPOSURE TO DERMAL CONTACT WITH SURFACE SOIL -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: Site 16

*** CTE- Dermal Contact with Surface Soil**

Surface Area for Contact = SA	6,200	cm^2/event
Adherence Factor = AF	0.16	mg/cm^2
Absorption Factor = ABS	chemical-specific	
Exposure Frequency = EF	48	event/yr
Exposure Duration = ED	1	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	365	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	1.00E-06	kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.13E-05	6.32E-13	9.03E-15	NA	1.50E+05	--	1.35E-09
Inorganics							
ALUMINUM	4.61E+03	0.00E+00	0.00E+00	1.00E+00	NA	0.00E+00	--
ARSENIC	1.87E+00	1.05E-07	1.49E-09	3.00E-04	1.50E+00	3.49E-04	2.24E-09
MANGANESE	1.45E+02	0.00E+00	0.00E+00	8.00E-04	NA	0.00E+00	--
THALLIUM	2.14E-01	0.00E+00	0.00E+00	7.00E-05	NA	0.00E+00	--
PAHs							
BENZ(A)ANTHRACENE	3.39E-01	8.21E-08	1.17E-09	NA	7.30E-01	--	8.57E-10
BENZO[A]PYRENE	2.59E-01	6.27E-08	8.96E-10	NA	7.30E+00	--	6.54E-09
BENZO(B)FLUORANTHENE	4.88E-01	1.18E-07	1.69E-09	NA	7.30E-01	--	1.23E-09
DIBENZ[A, H]ANTHRACENE	1.25E-01	3.03E-08	4.33E-10	NA	7.30E+00	--	3.16E-09
INDENO(1,2,3-CD)PYRENE	2.20E-01	5.33E-08	7.61E-10	NA	7.30E-01	--	5.56E-10
Cumulative Risk						**3.49E-04**	**1.59E-08**

Table B-16

ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RECREATIONAL USERS DUE TO EXPOSURE TO DERMAL CONTACT WITH SURFACE SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Dermal Contact with Surface Soil**

Surface Area for Contact = SA	8,000	cm^2/event
Adherence Factor = AF	0.32	mg/cm^2
Absorption Factor = ABS	chemical-specific	
Exposure Frequency = EF	96	event/yr
Exposure Duration = ED	24	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	8,760	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	1.00E-06	kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	3.79E-05	1.09E-11	3.75E-12	NA	1.50E+05	--	5.62E-07
Inorganics							
ALUMINUM	5.97E+03	0.00E+00	0.00E+00	1.00E+00	NA	0.00E+00	--
ARSENIC	2.67E+00	7.70E-07	2.64E-07	3.00E-04	1.50E+00	2.57E-03	3.96E-07
MANGANESE	1.85E+02	0.00E+00	0.00E+00	8.00E-04	NA	0.00E+00	--
THALLIUM	3.07E-01	0.00E+00	0.00E+00	7.00E-05	NA	0.00E+00	--
PAHs							
BENZ(A)ANTHRACENE	5.61E-01	7.01E-07	2.41E-07	NA	7.30E-01	--	1.76E-07
BENZO[A]PYRENE	4.68E-01	5.85E-07	2.01E-07	NA	7.30E+00	--	1.46E-06
BENZO(B)FLUORANTHENE	6.78E-01	8.48E-07	2.91E-07	NA	7.30E-01	--	2.12E-07
DIBENZ[A, H]ANTHRACENE	3.81E-01	4.76E-07	1.63E-07	NA	7.30E+00	--	1.19E-06
INDENO(1,2,3-CD)PYRENE	4.17E-01	5.21E-07	1.79E-07	NA	7.30E-01	--	1.31E-07
Cumulative Risk						**2.57E-03**	**4.13E-06**

Table B-17
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RECREATIONAL USERS DUE TO INHALATION OF PARTICULATES FROM SURFACE SOIL -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Inhalation of Particles from Surface Soil**

Inhalation Rate = IR	0.833	m3/hr
Exposure Time = ET	4	hr/day
Exposure Frequency = EF	48	day/yr
Exposure Duration = ED	1	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	3,650	days
Averaging Time (Cancer) = AT	25,550	days

Intake (mg/kg-day) = Conc * IR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/m3)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	8.56E-15	5.36E-18	7.66E-19	NA	1.50E+05	--	1.15E-13
Inorganics							
ALUMINUM	3.49E-06	2.19E-09	3.12E-10	1.00E-03	NA	2.19E-06	--
ARSENIC	1.42E-09	8.87E-13	1.27E-13	NA	1.51E+01	--	1.91E-12
MANGANESE	1.10E-07	6.88E-11	9.82E-12	1.43E-05	NA	4.81E-06	--
THALLIUM	1.62E-10	1.01E-13	1.45E-14	NA	NA	--	--
PAHs							
BENZ(A)ANTHRACENE	2.57E-10	1.61E-13	2.30E-14	NA	3.10E-01	--	7.12E-15
BENZO[A]PYRENE	1.96E-10	1.23E-13	1.75E-14	NA	3.10E+00	--	5.44E-14
BENZO(B)FLUORANTHENE	3.70E-10	2.31E-13	3.31E-14	NA	3.10E-01	--	1.02E-14
DIBENZ[A, H]ANTHRACENE	9.47E-11	5.93E-14	8.47E-15	NA	NA	--	--
INDENO(1,2,3-CD)PYRENE	1.67E-10	1.04E-13	1.49E-14	NA	3.10E-01	--	4.62E-15
Cumulative Risk						**6.99E-06**	**2.10E-12**

Table B-18
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RECREATIONAL USERS DUE TO INHALATION OF PARTICULATES FROM SURFACE SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: Site 16

*** RME- Inhalation of Particles from Surface Soil**

Inhalation Rate = IR	0.833 m3/hr
Exposure Time = ET	8 hr/day
Exposure Frequency = EF	96 day/yr
Exposure Duration = ED	24 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	3,650 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * IR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/m3)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	2.87E-14	1.73E-15	2.46E-16	NA	1.50E+05	--	3.70E-11
Inorganics							
ALUMINUM	4.52E-06	2.72E-07	3.88E-08	1.00E-03	NA	2.72E-04	--
ARSENIC	2.02E-09	1.22E-10	1.74E-11	NA	1.51E+01	--	2.62E-10
MANGANESE	1.40E-07	8.42E-09	1.20E-09	1.43E-05	NA	5.89E-04	--
THALLIUM	2.33E-10	1.40E-11	2.00E-12	NA	NA	--	--
PAHs							
BENZ(A)ANTHRACENE	4.25E-10	2.55E-11	3.65E-12	NA	3.10E-01	--	1.13E-12
BENZO[A]PYRENE	3.55E-10	2.13E-11	3.04E-12	NA	3.10E+00	--	9.44E-12
BENZO(B)FLUORANTHENE	5.14E-10	3.09E-11	4.41E-12	NA	3.10E-01	--	1.37E-12
DIBENZ[A, H]ANTHRACENE	2.89E-10	1.73E-11	2.48E-12	NA	NA	--	--
INDENO(1,2,3-CD)PYRENE	3.16E-10	1.90E-11	2.71E-12	NA	3.10E-01	--	8.41E-13
Cumulative Risk						**8.61E-04**	**3.12E-10**

Table B-19
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RECREATIONAL USERS DUE TO EXPOSURES TO SURFACE SOIL INGESTION -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: Site 16

*** CTE- Incidental Ingestion of Surface Soil**

Ingestion Rate = CR	100 mg/day
Exposure Frequency = EF	48 day/yr
Exposure Duration = ED	2 yr
Body Weight = BW	15 kg
Averaging Time (Noncancer) = AT	730 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.13E-05	9.91E-12	2.83E-13	NA	1.50E+05	--	4.25E-08
Inorganics							
ALUMINUM	4.61E+03	4.04E-03	1.15E-04	1.00E+00	NA	4.04E-03	--
ARSENIC	1.87E+00	1.64E-06	4.68E-08	3.00E-04	1.50E+00	5.46E-03	7.03E-08
MANGANESE	1.45E+02	1.27E-04	3.63E-06	2.00E-02	NA	6.36E-03	--
THALLIUM	2.14E-01	1.88E-07	5.36E-09	7.00E-05	NA	2.68E-03	--
PAHs							
BENZ(A)ANTHRACENE	3.39E-01	2.97E-07	8.49E-09	NA	7.30E-01	--	6.20E-09
BENZO[A]PYRENE	2.59E-01	2.27E-07	6.49E-09	NA	7.30E+00	--	4.74E-08
BENZO(B)FLUORANTHENE	4.88E-01	4.28E-07	1.22E-08	NA	7.30E-01	--	8.92E-09
DIBENZ[A, H]ANTHRACENE	1.25E-01	1.10E-07	3.13E-09	NA	7.30E+00	--	2.29E-08
INDENO(1,2,3-CD)PYRENE	2.20E-01	1.93E-07	5.51E-09	NA	7.30E-01	--	4.02E-09
Cumulative Risk						**1.85E-02**	**2.02E-07**

Table B-20
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RECREATIONAL USERS DUE TO EXPOSURES TO SURFACE SOIL INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Incidental Ingestion of Surface Soil**

Ingestion Rate = CR	200 mg/day
Exposure Frequency = EF	96 day/yr
Exposure Duration = ED	6 yr
Body Weight = BW	15 kg
Averaging Time (Noncancer) = AT	2,190 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	3.79E-05	1.33E-10	1.14E-11	NA	1.50E+05	--	1.71E-06
Inorganics							
ALUMINUM	5.97E+03	2.09E-02	1.79E-03	1.00E+00	NA	2.09E-02	--
ARSENIC	2.67E+00	9.36E-06	8.03E-07	3.00E-04	1.50E+00	3.12E-02	1.20E-06
MANGANESE	1.85E+02	6.49E-04	5.56E-05	2.00E-02	NA	3.24E-02	--
THALLIUM	3.07E-01	1.08E-06	9.23E-08	7.00E-05	NA	1.54E-02	--
PAHs							
BENZ(A)ANTHRACENE	5.61E-01	1.97E-06	1.69E-07	NA	7.30E-01	--	1.23E-07
BENZO[A]PYRENE	4.68E-01	1.64E-06	1.41E-07	NA	7.30E+00	--	1.03E-06
BENZO(B)FLUORANTHENE	6.78E-01	2.38E-06	2.04E-07	NA	7.30E-01	--	1.49E-07
DIBENZ[A, H]ANTHRACENE	3.81E-01	1.34E-06	1.15E-07	NA	7.30E+00	--	8.36E-07
INDENO(1,2,3-CD)PYRENE	4.17E-01	1.46E-06	1.25E-07	NA	7.30E-01	--	9.15E-08
Cumulative Risk						**1.00E-01**	**5.14E-06**

Table B-21

ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RECREATIONAL USERS DUE TO EXPOSURE TO DERMAL CONTACT WITH SURFACE SOIL -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Dermal Contact with Surface Soil**

Surface Area for Contact = SA	2,900 cm^2/event
Adherence Factor = AF	0.16 mg/cm^2
Absorption Factor = ABS	chemical-specific
Exposure Frequency = EF	48 event/yr
Exposure Duration = ED	2 yr
Body Weight = BW	15 kg
Averaging Time (Noncancer) = AT	730 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.13E-05	1.38E-12	3.94E-14	NA	1.50E+05	--	5.91E-09
Inorganics							
ALUMINUM	4.61E+03	0.00E+00	0.00E+00	1.00E+00	NA	0.00E+00	--
ARSENIC	1.87E+00	2.28E-07	6.52E-09	3.00E-04	1.50E+00	7.61E-04	9.78E-09
MANGANESE	1.45E+02	0.00E+00	0.00E+00	8.00E-04	NA	0.00E+00	--
THALLIUM	2.14E-01	0.00E+00	0.00E+00	7.00E-05	NA	0.00E+00	--
PAHs							
BENZ(A)ANTHRACENE	3.39E-01	1.79E-07	5.12E-09	NA	7.30E-01	--	3.74E-09
BENZO[A]PYRENE	2.59E-01	1.37E-07	3.91E-09	NA	7.30E+00	--	2.86E-08
BENZO(B)FLUORANTHENE	4.88E-01	2.58E-07	7.37E-09	NA	7.30E-01	--	5.38E-09
DIBENZ[A, H]ANTHRACENE	1.25E-01	6.61E-08	1.89E-09	NA	7.30E+00	--	1.38E-08
INDENO(1,2,3-CD)PYRENE	2.20E-01	1.16E-07	3.32E-09	NA	7.30E-01	--	2.43E-09
Cumulative Risk						**7.61E-04**	**6.96E-08**

Table B-22
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RECREATIONAL USERS DUE TO EXPOSURE TO DERMAL CONTACT WITH SURFACE SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Dermal Contact with Surface Soil**

Surface Area for Contact = SA	3,600	cm^2/event
Adherence Factor = AF	0.32	mg/cm^2
Absorption Factor = ABS	chemical-specific	
Exposure Frequency = EF	96	event/yr
Exposure Duration = ED	6	yr
Body Weight = BW	15	kg
Averaging Time (Noncancer) = AT	2,190	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	1.00E-06	kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	3.79E-05	2.30E-11	1.97E-12	NA	1.50E+05	--	2.95E-07
Inorganics							
ALUMINUM	5.97E+03	0.00E+00	0.00E+00	1.00E+00	NA	0.00E+00	--
ARSENIC	2.67E+00	1.62E-06	1.39E-07	3.00E-04	1.50E+00	5.39E-03	2.08E-07
MANGANESE	1.85E+02	0.00E+00	0.00E+00	8.00E-04	NA	0.00E+00	--
THALLIUM	3.07E-01	0.00E+00	0.00E+00	7.00E-05	NA	0.00E+00	--
PAHs							
BENZ(A)ANTHRACENE	5.61E-01	1.47E-06	1.26E-07	NA	7.30E-01	--	9.22E-08
BENZO[A]PYRENE	4.68E-01	1.23E-06	1.05E-07	NA	7.30E+00	--	7.69E-07
BENZO(B)FLUORANTHENE	6.78E-01	1.78E-06	1.53E-07	NA	7.30E-01	--	1.11E-07
DIBENZ[A, H]ANTHRACENE	3.81E-01	1.00E-06	8.58E-08	NA	7.30E+00	--	6.26E-07
INDENO(1,2,3-CD)PYRENE	4.17E-01	1.10E-06	9.39E-08	NA	7.30E-01	--	6.85E-08
Cumulative Risk						**5.39E-03**	**2.17E-06**

Table B-23

ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RECREATIONAL USERS DUE TO INHALATION OF PARTICULATES FROM SURFACE SOIL -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Inhalation of Particles from Surface Soil**

Inhalation Rate = IR	0.833	m3/hr
Exposure Time = ET	4	hr/day
Exposure Frequency = EF	48	day/yr
Exposure Duration = ED	2	yr
Body Weight = BW	15	kg
Averaging Time (Noncancer) = AT	730	days
Averaging Time (Cancer) = AT	25,550	days

Intake (mg/kg-day) = Conc * IR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/m3)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	8.56E-15	2.50E-16	7.14E-18	NA	1.50E+05	--	1.07E-12
Inorganics							
ALUMINUM	3.49E-06	1.02E-07	2.91E-09	1.00E-03	NA	1.02E-04	--
ARSENIC	1.42E-09	4.14E-11	1.18E-12	NA	1.51E+01	--	1.79E-11
MANGANESE	1.10E-07	3.21E-09	9.17E-11	1.43E-05	NA	2.24E-04	--
THALLIUM	1.62E-10	4.74E-12	1.35E-13	NA	NA	--	--
PAHs							
BENZ(A)ANTHRACENE	2.57E-10	7.50E-12	2.14E-13	NA	3.10E-01	--	6.64E-14
BENZO[A]PYRENE	1.96E-10	5.73E-12	1.64E-13	NA	3.10E+00	--	5.08E-13
BENZO(B)FLUORANTHENE	3.70E-10	1.08E-11	3.09E-13	NA	3.10E-01	--	9.57E-14
DIBENZ[A, H]ANTHRACENE	9.47E-11	2.77E-12	7.90E-14	NA	NA	--	--
INDENO(1,2,3-CD)PYRENE	1.67E-10	4.87E-12	1.39E-13	NA	3.10E-01	--	4.31E-14
Cumulative Risk						**3.26E-04**	**1.96E-11**

Table B-24

ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RECREATIONAL USERS DUE TO INHALATION OF PARTICULATES FROM SURFACE SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Inhalation of Particles from Surface Soil**

Inhalation Rate = IR	0.833 m3/hr
Exposure Time = ET	10 hr/day
Exposure Frequency = EF	96 day/yr
Exposure Duration = ED	6 yr
Body Weight = BW	15 kg
Averaging Time (Noncancer) = AT	2,190 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * IR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/m3)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	2.87E-14	4.19E-15	3.59E-16	NA	1.50E+05	--	5.39E-11
Inorganics							
ALUMINUM	4.52E-06	6.61E-07	5.66E-08	1.00E-03	NA	6.61E-04	--
ARSENIC	2.02E-09	2.95E-10	2.53E-11	NA	1.51E+01	--	3.82E-10
MANGANESE	1.40E-07	2.05E-08	1.75E-09	1.43E-05	NA	1.43E-03	--
THALLIUM	2.33E-10	3.40E-11	2.91E-12	NA	NA	--	--
PAHs							
BENZ(A)ANTHRACENE	4.25E-10	6.21E-11	5.32E-12	NA	3.10E-01	--	1.65E-12
BENZO[A]PYRENE	3.55E-10	5.18E-11	4.44E-12	NA	3.10E+00	--	1.38E-11
BENZO(B)FLUORANTHENE	5.14E-10	7.50E-11	6.43E-12	NA	3.10E-01	--	1.99E-12
DIBENZ[A, H]ANTHRACENE	2.89E-10	4.22E-11	3.61E-12	NA	NA	--	--
INDENO(1,2,3-CD)PYRENE	3.16E-10	4.61E-11	3.96E-12	NA	3.10E-01	--	1.23E-12
Cumulative Risk						**2.09E-03**	**4.55E-10**

Table B-25
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CONSTRUCTION WORKERS DUE TO EXPOSURES TO TOTAL SOIL INGESTION -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: Site 16

*** CTE- Incidental Ingestion of Total Soil**

Ingestion Rate = CR	100 mg/day
Exposure Frequency = EF	80 day/yr
Exposure Duration = ED	1 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	365 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	5.68E-05	1.78E-11	2.54E-13	NA	1.50E+05	--	3.81E-08
Inorganics							
ARSENIC	2.45E+00	7.67E-07	1.10E-08	3.00E-04	1.50E+00	2.56E-03	1.64E-08
PAHs							
BENZ(A)ANTHRACENE	3.19E-01	9.99E-08	1.43E-09	NA	7.30E-01	--	1.04E-09
BENZO[A]PYRENE	2.89E-01	9.05E-08	1.29E-09	NA	7.30E+00	--	9.44E-09
BENZO(B)FLUORANTHENE	4.27E-01	1.34E-07	1.91E-09	NA	7.30E-01	--	1.39E-09
DIBENZ[A, H]ANTHRACENE	1.51E-01	4.73E-08	6.75E-10	NA	7.30E+00	--	4.93E-09
2-METHYLNAPHTHALENE	1.19E+00	3.73E-07	5.32E-09	2.00E-02	NA	1.86E-05	--
Cumulative Risk						**2.58E-03**	**7.14E-08**

Table B-26
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CONSTRUCTION WORKERS DUE TO EXPOSURES TO TOTAL SOIL INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: Site 16

*** RME- Incidental Ingestion of Total Soil**

Ingestion Rate = CR	480	mg/day
Exposure Frequency = EF	160	day/yr
Exposure Duration = ED	1	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	365	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	1.00E-06	kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.91E-04	5.74E-10	8.20E-12	NA	1.50E+05	--	1.23E-06
Inorganics							
ARSENIC	8.64E+00	2.60E-05	3.71E-07	3.00E-04	1.50E+00	8.66E-02	5.57E-07
PAHs							
BENZ(A)ANTHRACENE	4.27E-01	1.28E-06	1.83E-08	NA	7.30E-01	--	1.34E-08
BENZO[A]PYRENE	4.18E-01	1.26E-06	1.79E-08	NA	7.30E+00	--	1.31E-07
BENZO(B)FLUORANTHENE	5.17E-01	1.55E-06	2.22E-08	NA	7.30E-01	--	1.62E-08
DIBENZ[A, H]ANTHRACENE	3.11E-01	9.35E-07	1.34E-08	NA	7.30E+00	--	9.75E-08
2-METHYLNAPHTHALENE	7.70E-01	2.31E-06	3.31E-08	2.00E-02	NA	1.16E-04	--
Cumulative Risk						**8.68E-02**	**2.04E-06**

Table B-27
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CONSTRUCTION WORKERS DUE TO EXPOSURE TO DERMAL CONTACT WITH TOTAL SOIL -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Dermal Contact with Total Soil**

Surface Area for Contact = SA	3,140 cm^2/event
Adherence Factor = AF	0.1 mg/cm^2
Absorption Factor = ABS	chemical-specific
Exposure Frequency = EF	80 event/yr
Exposure Duration = ED	1 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	365 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	5.68E-05	1.68E-12	2.39E-14	NA	1.50E+05	--	3.59E-09
Inorganics							
ARSENIC	2.45E+00	7.23E-08	1.03E-09	3.00E-04	1.50E+00	2.41E-04	1.55E-09
PAHs							
BENZ(A)ANTHRACENE	3.19E-01	4.08E-08	5.82E-10	NA	7.30E-01	--	4.25E-10
BENZO[A]PYRENE	2.89E-01	3.69E-08	5.28E-10	NA	7.30E+00	--	3.85E-09
BENZO(B)FLUORANTHENE	4.27E-01	5.46E-08	7.80E-10	NA	7.30E-01	--	5.69E-10
DIBENZ[A, H]ANTHRACENE	1.51E-01	1.93E-08	2.76E-10	NA	7.30E+00	--	2.01E-09
2-METHYLNAPHTHALENE	1.19E+00	1.52E-07	2.17E-09	2.00E-02	NA	7.60E-06	--
Cumulative Risk						**2.48E-04**	**1.20E-08**

Table B-28
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CONSTRUCTION WORKER DUE TO EXPOSURE TO DERMAL CONTACT WITH TOTAL SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Dermal Contact with Total Soil**

Surface Area for Contact = SA	3,800 cm^2/event
Adherence Factor = AF	0.3 mg/cm^2
Absorption Factor = ABS	chemical-specific
Exposure Frequency = EF	160 event/yr
Exposure Duration = ED	1 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	365 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.91E-04	4.09E-11	5.84E-13	NA	1.50E+05	--	8.77E-08
Inorganics							
ARSENIC	8.64E+00	1.85E-06	2.64E-08	3.00E-04	1.50E+00	6.17E-03	3.97E-08
PAHs							
BENZ(A)ANTHRACENE	4.27E-01	3.96E-07	5.66E-09	NA	7.30E-01	--	4.13E-09
BENZO[A]PYRENE	4.18E-01	3.88E-07	5.54E-09	NA	7.30E+00	--	4.05E-08
BENZO(B)FLUORANTHENE	5.17E-01	4.80E-07	6.85E-09	NA	7.30E-01	--	5.00E-09
DIBENZ[A, H]ANTHRACENE	3.11E-01	2.89E-07	4.12E-09	NA	7.30E+00	--	3.01E-08
2-METHYLNAPHTHALENE	7.70E-01	7.15E-07	1.02E-08	2.00E-02	NA	3.57E-05	--
Cumulative Risk						**6.20E-03**	**2.07E-07**

Table B-29
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CONSTRUCTION WORKER DUE TO INHALATION OF PARTICULATES FROM TOTAL SOIL -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Inhalation of Particles from Total Soil**

Inhalation Rate = IR	1.5	m3/hr
Exposure Time = ET	8	hr/day
Exposure Frequency = EF	80	day/yr
Exposure Duration = ED	1	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	365	days
Averaging Time (Cancer) = AT	25,550	days

Intake (mg/kg-day) = Conc * IR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/m3)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	4.30E-14	1.62E-15	2.31E-17	NA	1.50E+05	--	3.46E-12
Inorganics							
ARSENIC	1.86E-09	6.97E-11	9.96E-13	NA	1.51E+01	--	1.50E-11
PAHs							
BENZ(A)ANTHRACENE	2.42E-10	9.08E-12	1.30E-13	NA	3.10E-01	--	4.02E-14
BENZO[A]PYRENE	2.19E-10	8.23E-12	1.18E-13	NA	3.10E+00	--	3.64E-13
BENZO(B)FLUORANTHENE	3.23E-10	1.22E-11	1.74E-13	NA	3.10E-01	--	5.38E-14
DIBENZ[A, H]ANTHRACENE	1.14E-10	4.30E-12	6.14E-14	NA	NA	--	--
2-METHYLNAPHTHALENE	9.02E-10	3.39E-11	4.84E-13	NA	NA	--	--
Cumulative Risk						**0.00E+00**	**1.90E-11**

Table B-30
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CONSTRUCTION WORKERS DUE TO INHALATION OF PARTICULATES FROM TOTAL SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Inhalation of Particles from Total Soil**

Inhalation Rate = IR	2.5 m3/hr
Exposure Time = ET	8 hr/day
Exposure Frequency = EF	160 day/yr
Exposure Duration = ED	1 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	365 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * IR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/m3)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.45E-13	1.81E-14	2.59E-16	NA	1.50E+05	--	3.88E-11
Inorganics							
ARSENIC	6.55E-09	8.20E-10	1.17E-11	NA	1.51E+01	--	1.77E-10
PAHs							
BENZ(A)ANTHRACENE	3.23E-10	4.05E-11	5.79E-13	NA	3.10E-01	--	1.79E-13
BENZO[A]PYRENE	3.17E-10	3.97E-11	5.67E-13	NA	3.10E+00	--	1.76E-12
BENZO(B)FLUORANTHENE	3.92E-10	4.91E-11	7.01E-13	NA	3.10E-01	--	2.17E-13
DIBENZ[A, H]ANTHRACENE	2.36E-10	2.95E-11	4.22E-13	NA	NA	--	--
2-METHYLNAPHTHALENE	5.83E-10	7.31E-11	1.04E-12	NA	NA	--	--
Cumulative Risk						**0.00E+00**	**2.18E-10**

Table B-31
ESTIMATES OF CANCER AND NONCANCER RISKS FOR COMMERCIAL WORKERS DUE TO EXPOSURES TO SURFACE SOIL INGESTION -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Incidental Ingestion of Surface Soil**

Ingestion Rate = CR	50 mg/day
Exposure Frequency = EF	150 day/yr
Exposure Duration = ED	12 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	4,380 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.13E-05	3.32E-12	5.69E-13	NA	1.50E+05	--	8.53E-08
Inorganics							
ARSENIC	1.87E+00	5.49E-07	9.41E-08	3.00E-04	1.50E+00	1.83E-03	1.41E-07
PAHs							
BENZ(A)ANTHRACENE	3.39E-01	9.95E-08	1.71E-08	NA	7.30E-01	--	1.25E-08
BENZO[A]PYRENE	2.59E-01	7.60E-08	1.30E-08	NA	7.30E+00	--	9.51E-08
BENZO(B)FLUORANTHENE	4.88E-01	1.43E-07	2.46E-08	NA	7.30E-01	--	1.79E-08
DIBENZ[A, H]ANTHRACENE	1.25E-01	3.67E-08	6.29E-09	NA	7.30E+00	--	4.59E-08
Cumulative Risk						**1.83E-03**	**3.98E-07**

Table B-32
ESTIMATES OF CANCER AND NONCANCER RISKS FOR COMMERCIAL WORKERS DUE TO EXPOSURES TO SURFACE SOIL INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Incidental Ingestion of Surface Soil**

Ingestion Rate = CR	50 mg/day
Exposure Frequency = EF	250 day/yr
Exposure Duration = ED	25 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	9,125 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * CR * EF * ED *CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	3.79E-05	1.85E-11	6.62E-12	NA	1.50E+05	--	9.93E-07
Inorganics							
ARSENIC	2.67E+00	1.31E-06	4.67E-07	3.00E-04	1.50E+00	4.35E-03	7.00E-07
PAHs							
BENZ(A)ANTHRACENE	5.61E-01	2.74E-07	9.80E-08	NA	7.30E-01	--	7.16E-08
BENZO[A]PYRENE	4.68E-01	2.29E-07	8.18E-08	NA	7.30E+00	--	5.97E-07
BENZO(B)FLUORANTHENE	6.78E-01	3.32E-07	1.18E-07	NA	7.30E-01	--	8.65E-08
DIBENZ[A, H]ANTHRACENE	3.81E-01	1.86E-07	6.66E-08	NA	7.30E+00	--	4.86E-07
Cumulative Risk						**4.35E-03**	**2.93E-06**

Table B-33
ESTIMATES OF CANCER AND NONCANCER RISKS FOR COMMERCIAL WORKERS DUE TO EXPOSURE TO DERMAL CONTACT WITH SURFACE SOIL -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Dermal Contact with Surface Soil**

Surface Area for Contact = SA	3,100 cm^2/event
Adherence Factor = AF	0.02 mg/cm^2
Absorption Factor = ABS	chemical-specific
Exposure Frequency = EF	150 event/yr
Exposure Duration = ED	12 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	4,380 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	1.13E-05	1.23E-13	2.12E-14	NA	1.50E+05	--	3.17E-09
Inorganics							
ARSENIC	1.87E+00	2.04E-08	3.50E-09	3.00E-04	1.50E+00	6.81E-05	5.25E-09
PAHs							
BENZ(A)ANTHRACENE	3.39E-01	1.60E-08	2.75E-09	NA	7.30E-01	--	2.01E-09
BENZO[A]PYRENE	2.59E-01	1.23E-08	2.10E-09	NA	7.30E+00	--	1.53E-08
BENZO(B)FLUORANTHENE	4.88E-01	2.31E-08	3.96E-09	NA	7.30E-01	--	2.89E-09
DIBENZ[A, H]ANTHRACENE	1.25E-01	5.91E-09	1.01E-09	NA	7.30E+00	--	7.40E-09
Cumulative Risk						**6.81E-05**	**3.61E-08**

Table B-34
ESTIMATES OF CANCER AND NONCANCER RISKS FOR COMMERCIAL WORKERS DUE TO EXPOSURE TO DERMAL CONTACT WITH SURFACE SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: Site 16

*** RME- Dermal Contact with Surface Soil**

Surface Area for Contact = SA	3,800 cm^2/event
Adherence Factor = AF	0.2 mg/cm^2
Absorption Factor = ABS	chemical-specific
Exposure Frequency = EF	250 event/yr
Exposure Duration = ED	25 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	9,125 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	1.00E-06 kg/mg

Intake (mg/kg-day) = Conc * SA * AF * ABS * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	3.79E-05	8.46E-12	3.02E-12	NA	1.50E+05	--	4.53E-07
Inorganics							
ARSENIC	2.67E+00	5.96E-07	2.13E-07	3.00E-04	1.50E+00	1.99E-03	3.19E-07
PAHs							
BENZ(A)ANTHRACENE	5.61E-01	5.42E-07	1.94E-07	NA	7.30E-01	--	1.41E-07
BENZO[A]PYRENE	4.68E-01	4.52E-07	1.62E-07	NA	7.30E+00	--	1.18E-06
BENZO(B)FLUORANTHENE	6.78E-01	6.55E-07	2.34E-07	NA	7.30E-01	--	1.71E-07
DIBENZ[A, H]ANTHRACENE	3.81E-01	3.68E-07	1.32E-07	NA	7.30E+00	--	9.60E-07
Cumulative Risk						**1.99E-03**	**3.22E-06**

Table B-35
ESTIMATES OF CANCER AND NONCANCER RISKS FOR COMMERCIAL WORKERS DUE TO INHALATION OF PARTICULATES FROM SURFACE SOIL -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Inhalation of Particles from Surface Soil**

Inhalation Rate = IR	0.833 m3/hr
Exposure Time = ET	4 hr/day
Exposure Frequency = EF	150 day/yr
Exposure Duration = ED	12 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	4,380 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * IR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/m3)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	8.56E-15	1.67E-16	2.87E-17	NA	1.50E+05	--	4.31E-12
Inorganics							
ARSENIC	1.42E-09	2.77E-11	4.75E-12	NA	1.51E+01	--	7.17E-11
PAHs							
BENZ(A)ANTHRACENE	2.57E-10	5.02E-12	8.61E-13	NA	3.10E-01	--	2.67E-13
BENZO[A]PYRENE	1.96E-10	3.84E-12	6.58E-13	NA	3.10E+00	--	2.04E-12
BENZO(B)FLUORANTHENE	3.70E-10	7.23E-12	1.24E-12	NA	3.10E-01	--	3.84E-13
DIBENZ[A, H]ANTHRACENE	9.47E-11	1.85E-12	3.18E-13	NA	NA	--	--
Cumulative Risk						**0.00E+00**	**7.87E-11**

Table B-36
ESTIMATES OF CANCER AND NONCANCER RISKS FOR COMMERCIAL WORKERS DUE TO INHALATION OF PARTICULATES FROM SURFACE SOIL -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Inhalation of Particles from Surface Soil**

Inhalation Rate = IR	0.833	m3/hr
Exposure Time = ET	8	hr/day
Exposure Frequency = EF	250	day/yr
Exposure Duration = ED	25	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	9,125	days
Averaging Time (Cancer) = AT	25,550	days

Intake (mg/kg-day) = Conc * IR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/m3)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Dioxin/Furans							
TCDD-TEQ	2.87E-14	1.87E-15	6.69E-16	NA	1.50E+05	--	1.00E-10
Inorganics							
ARSENIC	2.02E-09	1.32E-10	4.71E-11	NA	1.51E+01	--	7.11E-10
PAHs							
BENZ(A)ANTHRACENE	4.25E-10	2.77E-11	9.90E-12	NA	3.10E-01	--	3.07E-12
BENZO[A]PYRENE	3.55E-10	2.31E-11	8.26E-12	NA	3.10E+00	--	2.56E-11
BENZO(B)FLUORANTHENE	5.14E-10	3.35E-11	1.20E-11	NA	3.10E-01	--	3.71E-12
DIBENZ[A, H]ANTHRACENE	2.89E-10	1.88E-11	6.72E-12	NA	NA	--	--
Cumulative Risk						**0.00E+00**	**8.44E-10**

Table B-37
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO EXPOSURES TO GROUNDWATER INGESTION -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: Site 16

*** CTE- Ingestion of Groundwater**

Ingestion Rate = CR	1.4 L/day
Exposure Frequency = EF	250 day/yr
Exposure Duration = ED	9 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	3,285 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * CR * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
BARIUM	4.36E-02	5.97E-04	7.68E-05	7.00E-02	NA	8.53E-03	--
MANGANESE	5.89E-01	8.07E-03	1.04E-03	2.00E-02	NA	4.03E-01	--
Semivolatiles							
BIS(2-ETHYLHEXYL)PHTHALATE	5.34E-03	7.32E-05	9.41E-06	2.20E-02	1.40E-02	3.33E-03	1.32E-07
1,4-DICHLOROBENZENE	4.94E-03	6.77E-05	8.70E-06	3.00E-02	2.40E-02	2.26E-03	2.09E-07
Volatiles							
1,1,2-TRICHLOROETHANE	1.10E-03	1.51E-05	1.94E-06	4.00E-03	5.70E-02	3.77E-03	1.10E-07
1,1-DICHLOROETHENE	1.19E-03	1.63E-05	2.10E-06	1.00E-01	NA	1.63E-04	--
1,2-DICHLOROETHANE	1.08E-03	1.48E-05	1.90E-06	3.00E-02	9.10E-02	4.93E-04	1.73E-07
ACETONE	9.87E-03	1.35E-04	1.74E-05	1.00E-01	NA	1.35E-03	--
BENZENE	1.11E-03	1.52E-05	1.95E-06	3.00E-03	5.50E-02	5.07E-03	1.08E-07
CHLOROFORM	1.10E-03	1.51E-05	1.94E-06	1.00E-02	6.10E-03	1.51E-03	1.18E-08
CIS-1,2-DICHLOROETHENE	6.08E-03	8.33E-05	1.07E-05	1.00E-02	NA	8.33E-03	--
TOTAL 1,2-DICHLOROETHENE	6.04E-03	8.27E-05	1.06E-05	2.00E-02	NA	4.14E-03	--
TRICHLOROETHENE	8.91E-01	1.22E-02	1.57E-03	6.00E-03	1.10E-02	2.03E+00	1.73E-05
VINYL CHLORIDE	2.22E-03	3.04E-05	3.91E-06	3.00E-03	1.50E+00	1.01E-02	5.86E-06
Cumulative Risk						**2.49E+00**	**2.39E-05**

Table B-38
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO EXPOSURES TO GROUNDWATER INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Ingestion of Groundwater**

Ingestion Rate = CR	2.3	L/day
Exposure Frequency = EF	350	day/yr
Exposure Duration = ED	24	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	8,760	days
Averaging Time (Cancer) = AT	25,550	days

Intake (mg/kg-day) = Conc * CR * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
BARIUM	3.78E-02	1.19E-03	4.08E-04	7.00E-02	NA	1.70E-02	--
MANGANESE	1.17E+00	3.69E-02	1.26E-02	2.00E-02	NA	1.84E+00	--
Semivolatiles							
BIS(2-ETHYLHEXYL)PHTHALATE	5.58E-03	1.76E-04	6.03E-05	2.20E-02	1.40E-02	7.99E-03	8.44E-07
1,4-DICHLOROBENZENE	5.15E-03	1.62E-04	5.56E-05	3.00E-02	2.40E-02	5.41E-03	1.34E-06
Volatiles							
1,1,2-TRICHLOROETHANE	8.14E-04	2.56E-05	8.79E-06	4.00E-03	5.70E-02	6.41E-03	5.01E-07
1,1-DICHLOROETHENE	9.53E-04	3.00E-05	1.03E-05	1.00E-01	NA	3.00E-04	--
1,2-DICHLOROETHANE	7.86E-04	2.48E-05	8.49E-06	3.00E-02	9.10E-02	8.25E-04	7.73E-07
ACETONE	8.09E-03	2.55E-04	8.74E-05	1.00E-01	NA	2.55E-03	--
BENZENE	8.22E-04	2.59E-05	8.88E-06	3.00E-03	5.50E-02	8.63E-03	4.88E-07
CHLOROFORM	8.01E-04	2.52E-05	8.65E-06	1.00E-02	6.10E-03	2.52E-03	5.28E-08
CIS-1,2-DICHLOROETHENE	5.59E-03	1.76E-04	6.04E-05	1.00E-02	NA	1.76E-02	--
TOTAL 1,2-DICHLOROETHENE	6.87E-03	2.16E-04	7.42E-05	2.00E-02	NA	1.08E-02	--
TRICHLOROETHENE	6.30E+00	1.98E-01	6.81E-02	6.00E-03	1.10E-02	3.31E+01	7.49E-04
VINYL CHLORIDE	1.64E-03	5.17E-05	1.77E-05	3.00E-03	1.50E+00	1.72E-02	2.66E-05
Cumulative Risk						**3.50E+01**	**7.79E-04**

Table B-39
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO EXPOSURE TO DERMAL CONTACT WITH GROUNDWATER -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: Site 16

*** CTE- Dermal Contact with Groundwater**

Surface Area Available for Contact = SA	18,200	cm2
Event Time = ET	0.166666667	hr/day
Permeability Constant = PC	chemical-specific	cm/hr
Exposure Frequency = EF	250	day/yr
Exposure Duration = ED	9	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	3,285	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	0.001	L/cm3

Intake (mg/kg-day) = Conc * SA * PC * ET * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
BARIUM	4.36E-02	1.29E-06	1.66E-07	4.90E-03	NA	2.64E-04	--
MANGANESE	5.89E-01	1.75E-05	2.25E-06	8.00E-04	NA	2.19E-02	--
Semivolatiles							
BIS(2-ETHYLHEXYL)PHTHALATE	5.34E-03	3.65E-06	4.69E-07	2.20E-02	1.40E-02	1.66E-04	6.56E-09
1,4-DICHLOROBENZENE	4.94E-03	9.09E-06	1.17E-06	3.00E-02	2.40E-02	3.03E-04	2.81E-08
Volatiles							
1,1,2-TRICHLOROETHANE	1.10E-03	2.74E-07	3.53E-08	4.00E-03	5.70E-02	6.86E-05	2.01E-09
1,1-DICHLOROETHENE	1.19E-03	3.14E-07	4.04E-08	1.00E-01	NA	3.14E-06	--
1,2-DICHLOROETHANE	1.08E-03	1.70E-07	2.18E-08	3.00E-02	9.10E-02	5.66E-06	1.99E-09
ACETONE	9.87E-03	1.67E-07	2.15E-08	1.00E-01	NA	1.67E-06	--
BENZENE	1.11E-03	6.92E-07	8.90E-08	3.00E-03	5.50E-02	2.31E-04	4.89E-09
CHLOROFORM	1.10E-03	2.91E-07	3.74E-08	1.00E-02	6.10E-03	2.91E-05	2.28E-10
CIS-1,2-DICHLOROETHENE	6.08E-03	1.80E-06	2.32E-07	1.00E-02	NA	1.80E-04	--
TOTAL 1,2-DICHLOROETHENE	6.04E-03	1.97E-07	2.54E-08	2.00E-02	NA	9.86E-06	--
TRICHLOROETHENE	8.91E-01	4.23E-04	5.44E-05	6.00E-03	1.10E-02	7.05E-02	5.98E-07
VINYL CHLORIDE	2.22E-03	4.81E-07	6.18E-08	3.00E-03	1.50E+00	1.60E-04	9.28E-08
Cumulative Risk						**9.38E-02**	**7.35E-07**

Table B-40
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO EXPOSURE TO DERMAL CONTACT WITH GROUNDWATER -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: Site 16

*** RME- Dermal Contact with Groundwater**

Surface Area Available for Contact = SA	22,000	cm2
Event Time = ET	0.583333333	hr/day
Permeability Constant = PC	chemical-specific	cm/hr
Exposure Frequency = EF	350	day/yr
Exposure Duration = ED	24	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	8,760	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	0.001	L/cm3

Intake (mg/kg-day) = Conc * SA * PC * ET * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
BARIUM	3.78E-02	6.65E-06	2.28E-06	4.90E-03	NA	1.36E-03	--
MANGANESE	1.17E+00	2.06E-04	7.05E-05	8.00E-04	NA	2.57E-01	--
Semivolatiles							
BIS(2-ETHYLHEXYL)PHTHALATE	5.58E-03	2.26E-05	7.74E-06	2.20E-02	1.40E-02	1.03E-03	1.08E-07
1,4-DICHLOROBENZENE	5.15E-03	5.61E-05	1.92E-05	3.00E-02	2.40E-02	1.87E-03	4.62E-07
Volatiles							
1,1,2-TRICHLOROETHANE	8.14E-04	1.20E-06	4.12E-07	4.00E-03	5.70E-02	3.01E-04	2.35E-08
1,1-DICHLOROETHENE	9.53E-04	1.49E-06	5.11E-07	1.00E-01	NA	1.49E-05	--
1,2-DICHLOROETHANE	7.86E-04	7.32E-07	2.51E-07	3.00E-02	9.10E-02	2.44E-05	2.28E-08
ACETONE	8.09E-03	8.11E-07	2.78E-07	1.00E-01	NA	8.11E-06	--
BENZENE	8.22E-04	3.03E-06	1.04E-06	3.00E-03	5.50E-02	1.01E-03	5.72E-08
CHLOROFORM	8.01E-04	1.25E-06	4.30E-07	1.00E-02	6.10E-03	1.25E-04	2.62E-09
CIS-1,2-DICHLOROETHENE	5.59E-03	9.83E-06	3.37E-06	1.00E-02	NA	9.83E-04	--
TOTAL 1,2-DICHLOROETHENE	6.87E-03	1.33E-06	4.55E-07	2.00E-02	NA	6.64E-05	--
TRICHLOROETHENE	6.30E+00	1.77E-02	6.08E-03	6.00E-03	1.10E-02	2.95E+00	6.68E-05
VINYL CHLORIDE	1.64E-03	2.10E-06	7.22E-07	3.00E-03	1.50E+00	7.02E-04	1.08E-06
Cumulative Risk						**3.22E+00**	**6.86E-05**

Table B-41
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO INHALATION OF VOLATILES FROM GROUNDWATER -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Inhalation of Volatiles from Groundwater**

Showers per Day = SD	1	event/day
Exposure Frequency = EF	175	day/yr
Exposure Duration = ED	9	yr
Averaging Time (Noncancer) = AT	3,285	days
Averaging Time (Cancer) = AT	25,550	days

Intake (mg/kg-day) = Conc * EF * ED * SD / AT

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg/shower)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Volatiles							
1,1,2-TRICHLOROETHANE	3.68E-05	1.76E-05	1.09E-06	NA	5.60E-02	--	6.09E-08
1,1-DICHLOROETHENE	5.33E-05	2.56E-05	1.58E-06	NA	NA	--	--
1,2-DICHLOROETHANE	4.08E-05	1.95E-05	1.20E-06	1.40E-03	9.10E-02	1.40E-02	1.10E-07
ACETONE	9.83E-05	4.72E-05	2.91E-06	NA	NA	--	--
BENZENE	5.25E-05	2.52E-05	1.55E-06	1.71E-03	2.90E-02	1.47E-02	4.50E-08
CHLOROFORM	4.38E-05	2.10E-05	1.29E-06	8.60E-05	8.05E-02	2.44E-01	1.04E-07
CIS-1,2-DICHLOROETHENE	2.63E-04	1.26E-04	7.77E-06	NA	NA	--	--
TOTAL 1,2-DICHLOROETHENE	2.67E-04	1.28E-04	7.90E-06	NA	NA	--	--
TRICHLOROETHENE	3.54E-02	1.70E-02	1.05E-03	NA	6.00E-03	--	6.28E-06
VINYL CHLORIDE	9.50E-05	4.56E-05	2.81E-06	2.90E-02	3.10E-02	1.57E-03	8.71E-08
Cumulative Risk						**2.74E-01**	**6.68E-06**

Table B-42
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO INHALATION OF VOLATILES FROM GROUNDWATER -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Inhalation of Volatiles from Groundwater**

Showers per Day = SD	1 event/day
Exposure Frequency = EF	350 day/yr
Exposure Duration = ED	24 yr
Averaging Time (Non-Cancer) = AT	3,285 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * EF * ED * SD / AT

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/kg/shower)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Volatiles							
1,1,2-TRICHLOROETHANE	2.72E-05	6.96E-05	8.95E-06	NA	5.60E-02	--	5.01E-07
1,1-DICHLOROETHENE	4.27E-05	1.09E-04	1.40E-05	NA	NA	--	--
1,2-DICHLOROETHANE	2.97E-05	7.58E-05	9.75E-06	1.40E-03	9.10E-02	5.42E-02	8.87E-07
ACETONE	8.06E-05	2.06E-04	2.65E-05	NA	NA	--	--
BENZENE	3.89E-05	9.94E-05	1.28E-05	1.71E-03	2.90E-02	5.81E-02	3.71E-07
CHLOROFORM	3.19E-05	8.16E-05	1.05E-05	8.60E-05	8.05E-02	9.49E-01	8.44E-07
CIS-1,2-DICHLOROETHENE	2.42E-04	6.18E-04	7.94E-05	NA	NA	--	--
TOTAL 1,2-DICHLOROETHENE	3.04E-04	7.78E-04	1.00E-04	NA	NA	--	--
TRICHLOROETHENE	2.50E-01	6.40E-01	8.23E-02	NA	6.00E-03	--	4.94E-04
VINYL CHLORIDE	7.02E-05	1.80E-04	2.31E-05	2.90E-02	3.10E-02	6.19E-03	7.15E-07
Cumulative Risk						**1.07E+00**	**4.97E-04**

Table B-43
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO EXPOSURES TO GROUNDWATER INGESTION -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: Site 16

* CTE- Ingestion of Groundwater

Ingestion Rate = CR	0.74 L/day
Exposure Frequency = EF	250 day/yr
Exposure Duration = ED	2 yr
Body Weight = BW	15 kg
Averaging Time (Noncancer) = AT	730 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * CR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
BARIUM	4.36E-02	1.47E-03	4.21E-05	7.00E-02	NA	2.10E-02	--
MANGANESE	5.89E-01	1.99E-02	5.69E-04	2.00E-02	NA	9.95E-01	--
Semivolatiles							
BIS(2-ETHYLHEXYL)PHTHALATE	5.34E-03	1.80E-04	5.16E-06	2.20E-02	1.40E-02	8.20E-03	7.22E-08
1,4-DICHLOROBENZENE	4.94E-03	1.67E-04	4.77E-06	3.00E-02	2.40E-02	5.56E-03	1.14E-07
Volatiles							
1,1,2-TRICHLOROETHANE	1.10E-03	3.72E-05	1.06E-06	4.00E-03	5.70E-02	9.29E-03	6.05E-08
1,1-DICHLOROETHENE	1.19E-03	4.02E-05	1.15E-06	1.00E-01	NA	4.02E-04	--
1,2-DICHLOROETHANE	1.08E-03	3.65E-05	1.04E-06	3.00E-02	9.10E-02	1.22E-03	9.49E-08
ACETONE	9.87E-03	3.34E-04	9.53E-06	1.00E-01	NA	3.34E-03	--
BENZENE	1.11E-03	3.75E-05	1.07E-06	3.00E-03	5.50E-02	1.25E-02	5.89E-08
CHLOROFORM	1.10E-03	3.72E-05	1.06E-06	1.00E-02	6.10E-03	3.72E-03	6.48E-09
CIS-1,2-DICHLOROETHENE	6.08E-03	2.05E-04	5.87E-06	1.00E-02	NA	2.05E-02	--
TOTAL 1,2-DICHLOROETHENE	6.04E-03	2.04E-04	5.83E-06	2.00E-02	NA	1.02E-02	--
TRICHLOROETHENE	8.91E-01	3.01E-02	8.60E-04	6.00E-03	1.10E-02	5.02E+00	9.46E-06
VINYL CHLORIDE	2.22E-03	7.50E-05	2.14E-06	3.00E-03	1.50E+00	2.50E-02	3.21E-06
Cumulative Risk						**6.13E+00**	**1.31E-05**

Table B-44
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO EXPOSURES TO GROUNDWATER INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: Site 16

*** RME- Ingestion of Groundwater**

Ingestion Rate = CR	1.5	L/day
Exposure Frequency = EF	350	day/yr
Exposure Duration = ED	6	yr
Body Weight = BW	15	kg
Averaging Time (Noncancer) = AT	2,190	days
Averaging Time (Cancer) = AT	25,550	days

Intake (mg/kg-day) = Conc * CR * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
BARIUM	3.78E-02	3.62E-03	3.11E-04	7.00E-02	NA	5.18E-02	--
MANGANESE	1.17E+00	1.12E-01	9.62E-03	2.00E-02	NA	5.61E+00	--
Semivolatiles							
BIS(2-ETHYLHEXYL)PHTHALATE	5.58E-03	5.35E-04	4.59E-05	2.20E-02	1.40E-02	2.43E-02	6.42E-07
1,4-DICHLOROBENZENE	5.15E-03	4.94E-04	4.23E-05	3.00E-02	2.40E-02	1.65E-02	1.02E-06
Volatiles							
1,1,2-TRICHLOROETHANE	8.14E-04	7.81E-05	6.69E-06	4.00E-03	5.70E-02	1.95E-02	3.81E-07
1,1-DICHLOROETHENE	9.53E-04	9.14E-05	7.83E-06	1.00E-01	NA	9.14E-04	--
1,2-DICHLOROETHANE	7.86E-04	7.54E-05	6.46E-06	3.00E-02	9.10E-02	2.51E-03	5.88E-07
ACETONE	8.09E-03	7.76E-04	6.65E-05	1.00E-01	NA	7.76E-03	--
BENZENE	8.22E-04	7.88E-05	6.76E-06	3.00E-03	5.50E-02	2.63E-02	3.72E-07
CHLOROFORM	8.01E-04	7.68E-05	6.58E-06	1.00E-02	6.10E-03	7.68E-03	4.02E-08
CIS-1,2-DICHLOROETHENE	5.59E-03	5.36E-04	4.59E-05	1.00E-02	NA	5.36E-02	--
TOTAL 1,2-DICHLOROETHENE	6.87E-03	6.59E-04	5.65E-05	2.00E-02	NA	3.29E-02	--
TRICHLOROETHENE	6.30E+00	6.04E-01	5.18E-02	6.00E-03	1.10E-02	1.01E+02	5.70E-04
VINYL CHLORIDE	1.64E-03	1.57E-04	1.35E-05	3.00E-03	1.50E+00	5.24E-02	2.02E-05
Cumulative Risk						**1.07E+02**	**5.93E-04**

Table B-45
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO EXPOSURE TO DERMAL CONTACT WITH GROUNDWATER -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: Site 16

*** CTE- Dermal Contact with Groundwater**

Surface Area Available for Contact = SA	9,000 cm2
Event Time = ET	0.33 hr/day
Permeability Constant = PC	chemical-specific cm/hr
Exposure Frequency = EF	250 day/yr
Exposure Duration = ED	2 yr
Body Weight = BW	15 kg
Averaging Time (Noncancer) = AT	730 days
Averaging Time (Cancer) = AT	25,550 days
Conversion Factor = CF	0.001 L/cm3

Intake (mg/kg-day) = Conc * SA * PC * ET * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
BARIUM	4.36E-02	5.91E-06	1.69E-07	4.90E-03	NA	1.21E-03	--
MANGANESE	5.89E-01	7.99E-05	2.28E-06	8.00E-04	NA	9.98E-02	--
Semivolatiles							
BIS(2-ETHYLHEXYL)PHTHALATE	5.34E-03	1.67E-05	4.76E-07	2.20E-02	1.40E-02	7.57E-04	6.66E-09
1,4-DICHLOROBENZENE	4.94E-03	4.15E-05	1.19E-06	3.00E-02	2.40E-02	1.38E-03	2.85E-08
Volatiles							
1,1,2-TRICHLOROETHANE	1.10E-03	1.25E-06	3.58E-08	4.00E-03	5.70E-02	3.13E-04	2.04E-09
1,1-DICHLOROETHENE	1.19E-03	1.44E-06	4.10E-08	1.00E-01	NA	1.44E-05	--
1,2-DICHLOROETHANE	1.08E-03	7.76E-07	2.22E-08	3.00E-02	9.10E-02	2.59E-05	2.02E-09
ACETONE	9.87E-03	7.63E-07	2.18E-08	1.00E-01	NA	7.63E-06	--
BENZENE	1.11E-03	3.16E-06	9.03E-08	3.00E-03	5.50E-02	1.05E-03	4.97E-09
CHLOROFORM	1.10E-03	1.33E-06	3.79E-08	1.00E-02	6.10E-03	1.33E-04	2.31E-10
CIS-1,2-DICHLOROETHENE	6.08E-03	8.25E-06	2.36E-07	1.00E-02	NA	8.25E-04	--
TOTAL 1,2-DICHLOROETHENE	6.04E-03	9.01E-07	2.57E-08	2.00E-02	NA	4.51E-05	--
TRICHLOROETHENE	8.91E-01	1.93E-03	5.52E-05	6.00E-03	1.10E-02	3.22E-01	6.08E-07
VINYL CHLORIDE	2.22E-03	2.20E-06	6.28E-08	3.00E-03	1.50E+00	7.33E-04	9.42E-08
Cumulative Risk						**4.29E-01**	**7.46E-07**

Table B-46
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RESIDENTS DUE TO EXPOSURE TO DERMAL CONTACT WITH GROUNDWATER -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Dermal Contact with Groundwater**

Surface Area Available for Contact = SA	10,500	cm2
Event Time = ET	1	hr/day
Permeability Constant = PC	chemical-specific	cm/hr
Exposure Frequency = EF	350	day/yr
Exposure Duration = ED	6	yr
Body Weight = BW	15	kg
Averaging Time (Noncancer) = AT	2,190	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	0.001	L/cm3

Intake (mg/kg-day) = Conc * SA * PC * ET * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
BARIUM	3.78E-02	2.54E-05	2.17E-06	4.90E-03	NA	5.18E-03	--
MANGANESE	1.17E+00	7.85E-04	6.73E-05	8.00E-04	NA	9.82E-01	--
Semivolatiles							
BIS(2-ETHYLHEXYL)PHTHALATE	5.58E-03	8.61E-05	7.38E-06	2.20E-02	1.40E-02	3.92E-03	1.03E-07
1,4-DICHLOROBENZENE	5.15E-03	2.14E-04	1.84E-05	3.00E-02	2.40E-02	7.14E-03	4.41E-07
Volatiles							
1,1,2-TRICHLOROETHANE	8.14E-04	4.59E-06	3.93E-07	4.00E-03	5.70E-02	1.15E-03	2.24E-08
1,1-DICHLOROETHENE	9.53E-04	5.69E-06	4.88E-07	1.00E-01	NA	5.69E-05	--
1,2-DICHLOROETHANE	7.86E-04	2.80E-06	2.40E-07	3.00E-02	9.10E-02	9.32E-05	2.18E-08
ACETONE	8.09E-03	3.10E-06	2.65E-07	1.00E-01	NA	3.10E-05	--
BENZENE	8.22E-04	1.16E-05	9.93E-07	3.00E-03	5.50E-02	3.86E-03	5.46E-08
CHLOROFORM	8.01E-04	4.79E-06	4.10E-07	1.00E-02	6.10E-03	4.79E-04	2.50E-09
CIS-1,2-DICHLOROETHENE	5.59E-03	3.75E-05	3.22E-06	1.00E-02	NA	3.75E-03	--
TOTAL 1,2-DICHLOROETHENE	6.87E-03	5.07E-06	4.35E-07	2.00E-02	NA	2.54E-04	--
TRICHLOROETHENE	6.30E+00	6.77E-02	5.80E-03	6.00E-03	1.10E-02	1.13E+01	6.38E-05
VINYL CHLORIDE	1.64E-03	8.04E-06	6.89E-07	3.00E-03	1.50E+00	2.68E-03	1.03E-06
Cumulative Risk						**1.23E+01**	**6.55E-05**

Table B-47
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CONSTRUCTION WORKERS DUE TO EXPOSURES TO GROUNDWATER INGESTION -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: Site 16

*** CTE- Ingestion of Groundwater**

Ingestion Rate = CR	0.025 L/day
Exposure Frequency = EF	80 day/yr
Exposure Duration = ED	1 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	365 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * CR * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
BARIUM	4.36E-02	3.41E-06	4.88E-08	7.00E-02	NA	4.88E-05	--
MANGANESE	5.89E-01	4.61E-05	6.59E-07	2.00E-02	NA	2.31E-03	--
Semivolatiles							
BIS(2-ETHYLHEXYL)PHTHALATE	5.34E-03	4.18E-07	5.97E-09	2.20E-02	1.40E-02	1.90E-05	8.36E-11
1,4-DICHLOROBENZENE	4.94E-03	3.87E-07	5.52E-09	3.00E-02	2.40E-02	1.29E-05	1.33E-10
Volatiles							
1,1,2-TRICHLOROETHANE	1.10E-03	8.61E-08	1.23E-09	4.00E-03	5.70E-02	2.15E-05	7.01E-11
1,1-DICHLOROETHENE	1.19E-03	9.32E-08	1.33E-09	1.00E-01	NA	9.32E-07	--
1,2-DICHLOROETHANE	1.08E-03	8.45E-08	1.21E-09	3.00E-02	9.10E-02	2.82E-06	1.10E-10
ACETONE	9.87E-03	7.73E-07	1.10E-08	1.00E-01	NA	7.73E-06	--
BENZENE	1.11E-03	8.69E-08	1.24E-09	3.00E-03	5.50E-02	2.90E-05	6.83E-11
CHLOROFORM	1.10E-03	8.61E-08	1.23E-09	1.00E-02	6.10E-03	8.61E-06	7.50E-12
CIS-1,2-DICHLOROETHENE	6.08E-03	4.76E-07	6.80E-09	1.00E-02	NA	4.76E-05	--
TOTAL 1,2-DICHLOROETHENE	6.04E-03	4.73E-07	6.75E-09	2.00E-02	NA	2.36E-05	--
TRICHLOROETHENE	8.91E-01	6.97E-05	9.96E-07	6.00E-03	1.10E-02	1.16E-02	1.10E-08
VINYL CHLORIDE	2.22E-03	1.74E-07	2.48E-09	3.00E-03	1.50E+00	5.79E-05	3.72E-09
Cumulative Risk						**1.42E-02**	**1.52E-08**

Table B-48
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RESIDENTS DUE TO EXPOSURES TO GROUNDWATER INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: Site 16

*** RME- Incidental Ingestion of Groundwater**

Ingestion Rate = CR	0.05 L/day
Exposure Frequency = EF	160 day/yr
Exposure Duration = ED	1 yr
Body Weight = BW	70 kg
Averaging Time (Noncancer) = AT	365 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * CR * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
BARIUM	3.78E-02	1.18E-05	1.69E-07	7.00E-02	NA	1.69E-04	--
MANGANESE	1.17E+00	3.66E-04	5.23E-06	2.00E-02	NA	1.83E-02	--
Semivolatiles							
BIS(2-ETHYLHEXYL)PHTHALATE	5.58E-03	1.75E-06	2.50E-08	2.20E-02	1.40E-02	7.94E-05	3.49E-10
1,4-DICHLOROBENZENE	5.15E-03	1.61E-06	2.30E-08	3.00E-02	2.40E-02	5.38E-05	5.53E-10
Volatiles							
1,1,2-TRICHLOROETHANE	8.14E-04	2.55E-07	3.64E-09	4.00E-03	5.70E-02	6.37E-05	2.08E-10
1,1-DICHLOROETHENE	9.53E-04	2.98E-07	4.26E-09	1.00E-01	NA	2.98E-06	--
1,2-DICHLOROETHANE	7.86E-04	2.46E-07	3.52E-09	3.00E-02	9.10E-02	8.20E-06	3.20E-10
ACETONE	8.09E-03	2.53E-06	3.62E-08	1.00E-01	NA	2.53E-05	--
BENZENE	8.22E-04	2.57E-07	3.68E-09	3.00E-03	5.50E-02	8.58E-05	2.02E-10
CHLOROFORM	8.01E-04	2.51E-07	3.58E-09	1.00E-02	6.10E-03	2.51E-05	2.19E-11
CIS-1,2-DICHLOROETHENE	5.59E-03	1.75E-06	2.50E-08	1.00E-02	NA	1.75E-04	--
TOTAL 1,2-DICHLOROETHENE	6.87E-03	2.15E-06	3.07E-08	2.00E-02	NA	1.08E-04	--
TRICHLOROETHENE	6.30E+00	1.97E-03	2.82E-05	6.00E-03	1.10E-02	3.29E-01	3.10E-07
VINYL CHLORIDE	1.64E-03	5.14E-07	7.34E-09	3.00E-03	1.50E+00	1.71E-04	1.10E-08
Cumulative Risk						**3.48E-01**	**3.23E-07**

Table B-49

ESTIMATES OF CANCER AND NONCANCER RISKS FOR CONSTRUCTION WORKERS DUE TO EXPOSURE TO DERMAL CONTACT WITH GROUNDWATER -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Dermal Contact with Groundwater**

Surface Area Available for Contact = SA	2,300	cm2
Event Time = ET	8	hr/day
Permeability Constant = PC	chemical-specific	cm/hr
Exposure Frequency = EF	80	day/yr
Exposure Duration = ED	1	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	365	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	0.001	L/cm3

Intake (mg/kg-day) = Conc * SA * PC * ET * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
BARIUM	4.36E-02	2.51E-06	3.59E-08	4.90E-03	NA	5.13E-04	--
MANGANESE	5.89E-01	3.39E-05	4.85E-07	8.00E-04	NA	4.24E-02	--
Semivolatiles							
BIS(2-ETHYLHEXYL)PHTHALATE	5.34E-03	7.08E-06	1.01E-07	2.20E-02	1.40E-02	3.22E-04	1.42E-09
1,4-DICHLOROBENZENE	4.94E-03	1.76E-05	2.52E-07	3.00E-02	2.40E-02	5.88E-04	6.05E-09
Volatiles							
1,1,2-TRICHLOROETHANE	1.10E-03	5.32E-07	7.60E-09	4.00E-03	5.70E-02	1.33E-04	4.33E-10
1,1-DICHLOROETHENE	1.19E-03	6.10E-07	8.72E-09	1.00E-01	NA	6.10E-06	--
1,2-DICHLOROETHANE	1.08E-03	3.30E-07	4.71E-09	3.00E-02	9.10E-02	1.10E-05	4.29E-10
ACETONE	9.87E-03	3.24E-07	4.63E-09	1.00E-01	NA	3.24E-06	--
BENZENE	1.11E-03	1.34E-06	1.92E-08	3.00E-03	5.50E-02	4.48E-04	1.06E-09
CHLOROFORM	1.10E-03	5.64E-07	8.06E-09	1.00E-02	6.10E-03	5.64E-05	4.92E-11
CIS-1,2-DICHLOROETHENE	6.08E-03	3.50E-06	5.00E-08	1.00E-02	NA	3.50E-04	--
TOTAL 1,2-DICHLOROETHENE	6.04E-03	3.83E-07	5.47E-09	2.00E-02	NA	1.91E-05	--
TRICHLOROETHENE	8.91E-01	8.21E-04	1.17E-05	6.00E-03	1.10E-02	1.37E-01	1.29E-07
VINYL CHLORIDE	2.22E-03	9.34E-07	1.33E-08	3.00E-03	1.50E+00	3.11E-04	2.00E-08
Cumulative Risk						**1.82E-01**	**1.59E-07**

Table B-50
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CONSTRUCTION WORKERS DUE TO EXPOSURE TO DERMAL CONTACT WITH GROUNDWATER -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: Site 16

*** RME- Dermal Contact with Groundwater**

Surface Area Available for Contact = SA	5,700	cm2
Event Time = ET	8	hr/day
Permeability Constant = PC	chemical-specific	cm/hr
Exposure Frequency = EF	160	day/yr
Exposure Duration = ED	1	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	365	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	0.001	L/cm3

Intake (mg/kg-day) = Conc * SA * PC * ET * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
BARIUM	3.78E-02	1.08E-05	1.54E-07	4.90E-03	NA	2.20E-03	--
MANGANESE	1.17E+00	3.34E-04	4.77E-06	8.00E-04	NA	4.18E-01	--
Semivolatiles							
BIS(2-ETHYLHEXYL)PHTHALATE	5.58E-03	3.66E-05	5.24E-07	2.20E-02	1.40E-02	1.67E-03	7.33E-09
1,4-DICHLOROBENZENE	5.15E-03	9.12E-05	1.30E-06	3.00E-02	2.40E-02	3.04E-03	3.13E-08
Volatiles							
1,1,2-TRICHLOROETHANE	8.14E-04	1.95E-06	2.79E-08	4.00E-03	5.70E-02	4.88E-04	1.59E-09
1,1-DICHLOROETHENE	9.53E-04	2.42E-06	3.46E-08	1.00E-01	NA	2.42E-05	--
1,2-DICHLOROETHANE	7.86E-04	1.19E-06	1.70E-08	3.00E-02	9.10E-02	3.97E-05	1.55E-09
ACETONE	8.09E-03	1.32E-06	1.88E-08	1.00E-01	NA	1.32E-05	--
BENZENE	8.22E-04	4.93E-06	7.04E-08	3.00E-03	5.50E-02	1.64E-03	3.87E-09
CHLOROFORM	8.01E-04	2.04E-06	2.91E-08	1.00E-02	6.10E-03	2.04E-04	1.77E-10
CIS-1,2-DICHLOROETHENE	5.59E-03	1.60E-05	2.28E-07	1.00E-02	NA	1.60E-03	--
TOTAL 1,2-DICHLOROETHENE	6.87E-03	2.16E-06	3.08E-08	2.00E-02	NA	1.08E-04	--
TRICHLOROETHENE	6.30E+00	2.88E-02	4.11E-04	6.00E-03	1.10E-02	4.80E+00	4.52E-06
VINYL CHLORIDE	1.64E-03	3.42E-06	4.88E-08	3.00E-03	1.50E+00	1.14E-03	7.33E-08
Cumulative Risk						**5.23E+00**	**4.64E-06**

Table B-51
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RECREATIONAL USERS DUE TO EXPOSURES TO SEEP WATER INGESTION -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: Site 16

*** CTE- Incidental Ingestion of Seep Water**

Ingestion Rate = CR	0.025	L/hr
Exposure Time=ET	1	hr/day
Exposure Frequency = EF	4	day/yr
Exposure Duration = ED	1	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	3,285	days
Averaging Time (Cancer) = AT	25,550	days

Intake (mg/kg-day) = Conc * CR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ARSENIC	3.30E-03	7.18E-10	9.23E-11	3.00E-04	1.50E+00	2.39E-06	1.38E-10
BARIUM	2.23E-01	4.85E-08	6.23E-09	7.00E-02	NA	6.93E-07	--
MANGANESE	8.72E-01	1.90E-07	2.44E-08	2.00E-02	NA	9.48E-06	--
PAHs							
2-METHYLNAPHTHALENE	5.17E-04	1.12E-10	1.45E-11	2.00E-02	NA	5.62E-09	--
NAPHTHALENE	2.37E-03	5.15E-10	6.63E-11	2.00E-02	NA	2.58E-08	--
Pesticides/PCBs							
ALPHA-BHC	1.93E-05	4.20E-12	5.40E-13	NA	6.30E+00	--	3.40E-12
DIELDRIN	4.00E-05	8.70E-12	1.12E-12	5.00E-05	1.60E+01	1.74E-07	1.79E-11
HEPTACHLOR EPOXIDE	2.33E-05	5.07E-12	6.51E-13	1.30E-05	9.10E+00	3.90E-07	5.93E-12
Semivolatiles							
DIBENZOFURAN	9.75E-03	2.12E-09	2.73E-10	4.00E-03	NA	5.30E-07	--
Volatiles							
VINYL CHLORIDE	6.50E-04	1.41E-10	1.82E-11	3.00E-03	1.50E+00	4.71E-08	2.73E-11
Cumulative Risk						**1.37E-05**	**1.93E-10**

Table B-52
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RECREATIONAL USERS DUE TO EXPOSURES TO SEEP WATER INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Incidental Ingestion of Seep Water**

Ingestion Rate = CR	0.05	L/hr
Exposure Time=ET	1	hr/day
Exposure Frequency = EF	7	day/yr
Exposure Duration = ED	24	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	8,760	days
Averaging Time (Cancer) = AT	25,550	days

Intake (mg/kg-day) = Conc * CR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ARSENIC	3.42E-03	4.68E-08	1.61E-08	3.00E-04	1.50E+00	1.56E-04	2.41E-08
BARIUM	3.70E-01	5.07E-06	1.74E-06	7.00E-02	NA	7.24E-05	--
MANGANESE	2.17E+00	2.97E-05	1.02E-05	2.00E-02	NA	1.49E-03	--
PAHs							
2-METHYLNAPHTHALENE	9.50E-04	1.30E-08	4.46E-09	2.00E-02	NA	6.51E-07	--
NAPHTHALENE	4.00E-03	5.48E-08	1.88E-08	2.00E-02	NA	2.74E-06	--
Pesticides/PCBs							
ALPHA-BHC	3.25E-05	4.45E-10	1.53E-10	NA	6.30E+00	--	9.62E-10
DIELDRIN	4.24E-05	5.81E-10	1.99E-10	5.00E-05	1.60E+01	1.16E-05	3.19E-09
HEPTACHLOR EPOXIDE	2.00E-05	2.74E-10	9.39E-11	1.30E-05	9.10E+00	2.11E-05	8.55E-10
Semivolatiles							
DIBENZOFURAN	1.15E-02	1.58E-07	5.40E-08	4.00E-03	NA	3.94E-05	--
Volatiles							
VINYL CHLORIDE	6.68E-04	9.15E-09	3.14E-09	3.00E-03	1.50E+00	3.05E-06	4.71E-09
Cumulative Risk						**1.79E-03**	**3.38E-08**

Table B-53
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RECREATIONAL USERS DUE TO EXPOSURE TO DERMAL CONTACT WITH SEEP WATER -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Dermal Contact with Seep Water**

Surface Area for Contact=SA	5,000	cm2
Event Time = ET	1	hr/day
Permeability Constant = PC	chemical-specific	cm/hr
Exposure Frequency = EF	4	day/yr
Exposure Duration = ED	1	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	3,285	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	0.001	L/cm3

Intake (mg/kg-day) = Conc * SA * PC * ET * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ARSENIC	3.30E-03	1.44E-10	1.85E-11	3.00E-04	1.50E+00	4.78E-07	2.77E-11
BARIUM	2.23E-01	9.70E-09	1.25E-09	4.90E-03	NA	1.98E-06	--
MANGANESE	8.72E-01	3.79E-08	4.88E-09	8.00E-04	NA	4.74E-05	--
PAHs							
2-METHYLNAPHTHALENE	5.17E-04	1.55E-09	1.99E-10	2.00E-02	NA	7.76E-08	--
NAPHTHALENE	2.37E-03	7.11E-09	9.14E-10	2.00E-02	NA	3.56E-07	--
Pesticides/PCBs							
ALPHA-BHC	1.93E-05	1.59E-11	2.05E-12	NA	6.30E+00	--	1.29E-11
DIELDRIN	4.00E-05	2.78E-12	3.58E-13	5.00E-05	1.60E+01	5.57E-08	5.73E-12
HEPTACHLOR EPOXIDE	2.33E-05	1.11E-11	1.43E-12	1.30E-05	9.10E+00	8.57E-07	1.30E-11
Semivolatiles							
DIBENZOFURAN	9.75E-03	2.59E-08	3.33E-09	4.00E-03	NA	6.47E-06	--
Volatiles							
VINYL CHLORIDE	6.50E-04	2.06E-10	2.65E-11	3.00E-03	1.50E+00	6.88E-08	3.98E-11
Cumulative Risk						**5.77E-05**	**9.92E-11**

Table B-54
ESTIMATES OF CANCER AND NONCANCER RISKS FOR ADULT RECREATIONAL USERS DUE TO EXPOSURE TO DERMAL CONTACT WITH SEEP WATER -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Dermal Contact with Seep Water**

Surface Area Available for Contact = SA	6,700	cm2
Event Time = ET	1	hr/day
Permeability Constant = PC	:hemical-specific	cm/hr
Exposure Frequency = EF	7	day/yr
Exposure Duration = ED	24	yr
Body Weight = BW	70	kg
Averaging Time (Noncancer) = AT	8,760	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	0.001	L/cm3

Intake (mg/kg-day) = Conc * SA * PC * ET * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ARSENIC	3.42E-03	6.28E-09	2.15E-09	3.00E-04	1.50E+00	2.09E-05	3.23E-09
BARIUM	3.70E-01	6.79E-07	2.33E-07	4.90E-03	NA	1.39E-04	--
MANGANESE	2.17E+00	3.98E-06	1.37E-06	8.00E-04	NA	4.98E-03	--
PAHs							
2-METHYLNAPHTHALENE	9.50E-04	1.20E-07	4.13E-08	2.00E-02	NA	6.02E-06	--
NAPHTHALENE	4.00E-03	5.07E-07	1.74E-07	2.00E-02	NA	2.53E-05	--
Pesticides/PCBs							
ALPHA-BHC	3.25E-05	1.13E-09	3.89E-10	NA	6.30E+00	--	2.45E-09
DIELDRIN	4.24E-05	1.25E-10	4.27E-11	5.00E-05	1.60E+01	2.49E-06	6.83E-10
HEPTACHLOR EPOXIDE	2.00E-05	4.04E-10	1.38E-10	1.30E-05	9.10E+00	3.11E-05	1.26E-09
Semivolatiles							
DIBENZOFURAN	1.15E-02	1.29E-06	4.41E-07	4.00E-03	NA	3.22E-04	--
Volatiles							
VINYL CHLORIDE	6.68E-04	8.95E-09	3.07E-09	3.00E-03	1.50E+00	2.98E-06	4.60E-09
Cumulative Risk						**5.53E-03**	**1.22E-08**

Table B-55
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RECREATIONAL USERS DUE TO EXPOSURES TO SEEP WATER INGESTION -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Incidental Ingestion of Seep Water**

Ingestion Rate = CR	0.025	L/hr
Exposure Time=ET	1	hr/day
Exposure Frequency = EF	4	day/yr
Exposure Duration = ED	2	yr
Body Weight = BW	15	kg
Averaging Time (Noncancer) = AT	730	days
Averaging Time (Cancer) = AT	25,550	days

Intake (mg/kg-day) = Conc * CR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ARSENIC	3.30E-03	3.01E-08	8.61E-10	3.00E-04	1.50E+00	1.00E-04	1.29E-09
BARIUM	2.23E-01	2.04E-06	5.82E-08	7.00E-02	NA	2.91E-05	--
MANGANESE	8.72E-01	7.96E-06	2.28E-07	2.00E-02	NA	3.98E-04	--
PAHs							
2-METHYLNAPHTHALENE	5.17E-04	4.72E-09	1.35E-10	2.00E-02	NA	2.36E-07	--
NAPHTHALENE	2.37E-03	2.16E-08	6.18E-10	2.00E-02	NA	1.08E-06	--
Pesticides/PCBs							
ALPHA-BHC	1.93E-05	1.76E-10	5.04E-12	NA	6.30E+00	--	3.17E-11
DIELDRIN	4.00E-05	3.65E-10	1.04E-11	5.00E-05	1.60E+01	7.31E-06	1.67E-10
HEPTACHLOR EPOXIDE	2.33E-05	2.13E-10	6.08E-12	1.30E-05	9.10E+00	1.64E-05	5.53E-11
Semivolatiles							
DIBENZOFURAN	9.75E-03	8.90E-08	2.54E-09	4.00E-03	NA	2.23E-05	--
Volatiles							
VINYL CHLORIDE	6.50E-04	5.94E-09	1.70E-10	3.00E-03	1.50E+00	1.98E-06	2.54E-10
Cumulative Risk						**5.77E-04**	**1.80E-09**

Table B-56
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RECREATIONAL USERS DUE TO EXPOSURES TO SEEP WATER INGESTION -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: **Site 16**

*** RME- Incidental Ingestion of Seep Water**

Ingestion Rate = CR	0.05 L/hr
Exposure Time=ET	1 hr/day
Exposure Frequency = EF	7 day/yr
Exposure Duration = ED	6 yr
Body Weight = BW	15 kg
Averaging Time (Noncancer) = AT	2,190 days
Averaging Time (Cancer) = AT	25,550 days

Intake (mg/kg-day) = Conc * CR * ET * EF * ED / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ARSENIC	3.42E-03	2.19E-07	1.87E-08	3.00E-04	1.50E+00	7.29E-04	2.81E-08
BARIUM	3.70E-01	2.37E-05	2.03E-06	7.00E-02	NA	3.38E-04	--
MANGANESE	2.17E+00	1.39E-04	1.19E-05	2.00E-02	NA	6.94E-03	--
PAHs							
2-METHYLNAPHTHALENE	9.50E-04	6.07E-08	5.21E-09	2.00E-02	NA	3.04E-06	--
NAPHTHALENE	4.00E-03	2.56E-07	2.19E-08	2.00E-02	NA	1.28E-05	--
Pesticides/PCBs							
ALPHA-BHC	3.25E-05	2.08E-09	1.78E-10	NA	6.30E+00	--	1.12E-09
DIELDRIN	4.24E-05	2.71E-09	2.32E-10	5.00E-05	1.60E+01	5.42E-05	3.72E-09
HEPTACHLOR EPOXIDE	2.00E-05	1.28E-09	1.10E-10	1.30E-05	9.10E+00	9.83E-05	9.97E-10
Semivolatiles							
DIBENZOFURAN	1.15E-02	7.35E-07	6.30E-08	4.00E-03	NA	1.84E-04	--
Volatiles							
VINYL CHLORIDE	6.68E-04	4.27E-08	3.66E-09	3.00E-03	1.50E+00	1.42E-05	5.49E-09
Cumulative Risk						**8.37E-03**	**3.94E-08**

Table B-57
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RECREATIONAL USERS DUE TO EXPOSURE TO DERMAL CONTACT WITH SEEP WATER -- CENTRAL TENDENCY EXPOSURE SCENARIO (CTE)

Site Number: **Site 16**

*** CTE- Dermal Contact with Seep Water**

Surface Area Available for Contact = SA	2,700	cm2
Event Time = ET	1	hr/day
Permeability Constant = PC	chemical-specific	cm/hr
Exposure Frequency = EF	4	day/yr
Exposure Duration = ED	2	yr
Body Weight = BW	15	kg
Averaging Time (Noncancer) = AT	730	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	0.001	L/cm3

Intake (mg/kg-day) = Conc * SA * PC * ET * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ARSENIC	3.30E-03	3.25E-09	9.30E-11	3.00E-04	1.50E+00	1.08E-05	1.39E-10
BARIUM	2.23E-01	2.20E-07	6.28E-09	4.90E-03	NA	4.49E-05	--
MANGANESE	8.72E-01	8.60E-07	2.46E-08	8.00E-04	NA	1.08E-03	--
PAHs							
2-METHYLNAPHTHALENE	5.17E-04	3.52E-08	1.01E-09	2.00E-02	NA	1.76E-06	--
NAPHTHALENE	2.37E-03	1.61E-07	4.61E-09	2.00E-02	NA	8.06E-06	--
Pesticides/PCBs							
ALPHA-BHC	1.93E-05	3.62E-10	1.03E-11	NA	6.30E+00	--	6.51E-11
DIELDRIN	4.00E-05	6.31E-11	1.80E-12	5.00E-05	1.60E+01	1.26E-06	2.89E-11
HEPTACHLOR EPOXIDE	2.33E-05	2.53E-10	7.22E-12	1.30E-05	9.10E+00	1.94E-05	6.57E-11
Semivolatiles							
DIBENZOFURAN	9.75E-03	5.87E-07	1.68E-08	4.00E-03	NA	1.47E-04	--
Volatiles							
VINYL CHLORIDE	6.50E-04	4.68E-09	1.34E-10	3.00E-03	1.50E+00	1.56E-06	2.01E-10
Cumulative Risk						**1.31E-03**	**5.00E-10**

Table B-58
ESTIMATES OF CANCER AND NONCANCER RISKS FOR CHILD RECREATIONAL USERS DUE TO EXPOSURE TO DERMAL CONTACT WITH SEEP WATER -- REASONABLE MAXIMUM SCENARIO (RME)

Site Number: Site 16

*** RME- Dermal Contact with Seep Water**

Surface Area Available for Contact = SA	3,300	cm2
Event Time = ET	1	hr/day
Permeability Constant = PC	chemical-specific	cm/hr
Exposure Frequency = EF	7	day/yr
Exposure Duration = ED	6	yr
Body Weight = BW	15	kg
Averaging Time (Noncancer) = AT	2,190	days
Averaging Time (Cancer) = AT	25,550	days
Conversion Factor = CF	0.001	L/cm3

Intake (mg/kg-day) = Conc * SA * PC * ET * EF * ED * CF / (BW * AT)

NCADI = Daily intake - Noncarcinogens
CADI = Daily Intake - Carcinogens
HQ = Hazard Quotient - Noncarcinogens = NCADI / RfD
Risk = Cancer Risk = CADI * SF

Chemical of Concern	EPC (mg/L)	NCADI (mg/kg-day)	CADI (mg/kg-day)	Chronic RfD (mg/kg-day)	SF per (mg/kg-day)	HQ	Risk
Inorganics							
ARSENIC	3.42E-03	1.44E-08	1.24E-09	3.00E-04	1.50E+00	4.81E-05	1.86E-09
BARIUM	3.70E-01	1.56E-06	1.34E-07	4.90E-03	NA	3.19E-04	--
MANGANESE	2.17E+00	9.16E-06	7.85E-07	8.00E-04	NA	1.14E-02	--
PAHs							
2-METHYLNAPHTHALENE	9.50E-04	2.77E-07	2.37E-08	2.00E-02	NA	1.38E-05	--
NAPHTHALENE	4.00E-03	1.16E-06	9.98E-08	2.00E-02	NA	5.82E-05	--
Pesticides/PCBs							
ALPHA-BHC	3.25E-05	2.61E-09	2.23E-10	NA	6.30E+00	--	1.41E-09
DIELDRIN	4.24E-05	2.86E-10	2.45E-11	5.00E-05	1.60E+01	5.72E-06	3.93E-10
HEPTACHLOR EPOXIDE	2.00E-05	9.28E-10	7.96E-11	1.30E-05	9.10E+00	7.14E-05	7.24E-10
Semivolatiles							
DIBENZOFURAN	1.15E-02	2.96E-06	2.54E-07	4.00E-03	NA	7.40E-04	--
Volatiles							
VINYL CHLORIDE	6.68E-04	2.06E-08	1.76E-09	3.00E-03	1.50E+00	6.86E-06	2.65E-09
Cumulative Risk						**1.27E-02**	**7.02E-09**

Table B-59
CALCULATIONS OF AIR CONCENTRATIONS DUE TO DUST ENTRAINMENT FROM SURFACE SOIL AND TOTAL SOIL

Site Number: Site 16

Model Equations:

Particulate Emmision Factor	PEF = Q/C x [(3,600 s/h)/(.36 x (1- V) x (Um/Ut)^3 x F(x))] =	1.32E+09 m3/kg
Air Concentration	Cair = Csoil/PEF	

Model Constants:

Q/C	90.8	g/m2-s per kg/m3	Inverse Mean Concentration at Center of 0.05 square, U.S. EPA 1996
V	0.5	unitless	Default, U.S. EPA 1996
Um	4.69	m/s	Mean annual wind speed, EPA, 1996
Ut	11.32	m/s	Equivalent thresghold value of windspeed at 7 m, EPA, 1996
F(x)	0.194	unitless	Default, U.S. EPA 1996

Reference for the model: USEPA Soil Screening Guidance: Technical Background Document. Office of Emergency and Remedial Response. U.S. EPA, 1996.

Chemical	Csoil, Surface Soil		Csoil, Total Soil		Cair, Surface Soil Particulate		Cair, Total Soil Particulate	
	CTE EPC	RME EPC	CTE EPC	RME EPC	CTE EPC	RME EPC	CTE EPC	RME EPC
	mg/kg	mg/kg	mg/kg	mg/kg	mg/m3	mg/m3	mg/m3	mg/m3
Dioxin/Furans								
TCDD-TEQ	1.13E-05	3.79E-05	5.68E-05	1.91E-04	8.56E-15	2.87E-14	4.30E-14	1.45E-13
Inorganics								
ALUMINUM	4.61E+03	5.97E+03	5.57E+03	6.61E+03	3.49E-06	4.52E-06	4.22E-06	5.01E-06
ANTIMONY	NA	NA	9.61E-01	1.32E+00	NA	NA	7.28E-10	1.00E-09
ARSENIC	1.87E+00	2.67E+00	2.45E+00	8.64E+00	1.42E-09	2.02E-09	1.86E-09	6.55E-09
BARIUM	NA	NA	7.81E+01	8.50E+01	NA	NA	5.92E-08	6.44E-08
COPPER	NA	NA	4.28E+01	5.56E+01	NA	NA	3.24E-08	4.21E-08
MANGANESE	1.45E+02	1.85E+02	1.66E+02	2.00E+02	1.10E-07	1.40E-07	1.26E-07	1.52E-07
THALLIUM	2.14E-01	3.07E-01	2.76E-01	3.36E-01	1.62E-10	2.33E-10	2.09E-10	2.55E-10
PAHs								
BENZ(A)ANTHRACENE	3.39E-01	5.61E-01	3.19E-01	4.27E-01	2.57E-10	4.25E-10	2.42E-10	3.23E-10
BENZO[A]PYRENE	2.59E-01	4.68E-01	2.89E-01	4.18E-01	1.96E-10	3.55E-10	2.19E-10	3.17E-10
BENZO(B)FLUORANTHENE	4.88E-01	6.78E-01	4.27E-01	5.17E-01	3.70E-10	5.14E-10	3.23E-10	3.92E-10
DIBENZ[A, H]ANTHRACENE	1.25E-01	3.81E-01	1.51E-01	3.11E-01	9.47E-11	2.89E-10	1.14E-10	2.36E-10
INDENO(1,2,3-CD)PYRENE	2.20E-01	4.17E-01	2.31E-01	3.51E-01	1.67E-10	3.16E-10	1.75E-10	2.66E-10
2-METHYLNAPHTHALENE	NA	NA	1.19E+00	7.70E-01	NA	NA	9.02E-10	5.83E-10
NAPHTHALENE	NA	NA	3.93E-01	4.91E-01	NA	NA	2.98E-10	3.72E-10

NA = Not applicable because the chemical is not a COPC in this media.

Table B-60
INPUTS FOR ADULT RESIDENT SHOWER MODEL
DAVISVILLE SITE 16

Parameters in the Model	Units	Value
Liquid Phase t.c. CO2	cm/h	20
Gas Phase t.c. H2O	cm/h	3000
Water Viscosity at 20C	cp	1.002
Water Viscosity at 45C	cp	0.596
Shower Temp	K	318
Droplet Diameter	mm	1
Drop Time	s	2
Shower Flow Rate	L/min	20
Shower Stall Volume	m^3	6.00E+00
Shower Duration	min	1.20E+01
Air Exchange Rate	min^{-1}	0.0166667
Body Weight	kg	70
RT	atm-m^3/mol	2.40E-02
Duration in Shower Room	min	2.00E+01
Ventillation Rate	L/min	1.40E+01

Table B-61
CHEMICAL PHYSICAL PARAMETERS FOR ADULT RESIDENT SHOWER MODEL
DAVISVILLE SITE 16

Chemical	Henry's Law atm-m3/mol-K	Molecular Weight g/mol	Gas Phase Transfer Coefficient cm/hr	Liquid Phase Transfer Coefficient cm/hr	Overall Mass Transfer Coefficient cm/hr	Adjusted Overall Mass Transfer Coefficient cm/hr
1,1,2-Trichloroethane	9.10E-04	1.30E+02	1.12E+03	1.16E+01	9.13E+00	1.23E+01
1,1-Dichloroethene	2.60E-02	9.70E+01	1.29E+03	1.35E+01	1.33E+01	1.80E+01
1,2-Dichloroethane	9.80E-04	9.90E+01	1.28E+03	1.33E+01	1.06E+01	1.43E+01
Acetone	3.90E-05	5.80E+01	1.67E+03	1.74E+01	2.35E+00	3.17E+00
Benzene	5.56E-03	7.81E+01	1.44E+03	1.50E+01	1.44E+01	1.94E+01
Chloroform	3.70E-03	1.19E+02	1.17E+03	1.22E+01	1.14E+01	1.54E+01
cis 1,2-Dichloroethene	4.10E-03	9.70E+01	1.29E+03	1.35E+01	1.27E+01	1.71E+01
total 1,2-Dichloroethene	9.40E-03	9.70E+01	1.29E+03	1.35E+01	1.31E+01	1.77E+01
Trichloroethene	1.00E-02	1.30E+02	1.12E+03	1.16E+01	1.14E+01	1.53E+01
Vinyl chloride	2.70E-02	1.10E+02	1.21E+03	1.26E+01	1.25E+01	1.69E+01

Henry's Law and Molecular Weight information from www.epa.gov/region09/waste/sfund/prg/s5_02.htm

Table B-62
ESTIMATES OF EXPOSURE POINT CONCENTRATIONS FOR ADULT RESIDENT SHOWER MODEL
DAVISVILLE SITE 16

Chemical	RME Groundwater Concentration ug/L	Concentration Leaving Shower Droplet ug/L	Indoor VOC Generation Rate ug/m3-min	Inhalation Exposure Per Shower mg/kg/shower
1,1,2-Trichloroethane	8.14E-01	2.74E-01	9.14E-01	2.72E-05
1,1-Dichloroethene	9.53E-01	4.30E-01	1.43E+00	4.27E-05
1,2-Dichloroethane	7.86E-01	2.99E-01	9.96E-01	2.97E-05
Acetone	8.09E+00	8.12E-01	2.71E+00	8.06E-05
Benzene	8.22E-01	3.92E-01	1.31E+00	3.89E-05
Chloroform	8.01E-01	3.21E-01	1.07E+00	3.19E-05
cis 1,2-Dichloroethene	5.59E+00	2.43E+00	8.11E+00	2.42E-04
total 1,2-Dichloroethene	6.87E+00	3.06E+00	1.02E+01	3.04E-04
Trichloroethene	6.30E+03	2.52E+03	8.40E+03	2.50E-01
Vinyl chloride	1.64E+00	7.07E-01	2.36E+00	7.02E-05

Chemical	CTE Groundwater Concentration ug/L	Concentration Leaving Shower Droplet ug/L	Indoor VOC Generation Rate ug/m3-min	Inhalation Exposure Per Shower mg/kg/shower
1,1,2-Trichloroethane	1.10E+00	3.71E-01	1.24E+00	3.68E-05
1,1-Dichloroethene	1.19E+00	5.37E-01	1.79E+00	5.33E-05
1,2-Dichloroethane	1.08E+00	4.11E-01	1.37E+00	4.08E-05
Acetone	9.87E+00	9.91E-01	3.30E+00	9.83E-05
Benzene	1.11E+00	5.29E-01	1.76E+00	5.25E-05
Chloroform	1.10E+00	4.41E-01	1.47E+00	4.38E-05
cis 1,2-Dichloroethene	6.08E+00	2.65E+00	8.82E+00	2.63E-04
total 1,2-Dichloroethene	6.04E+00	2.69E+00	8.98E+00	2.67E-04
Trichloroethene	8.91E+02	3.57E+02	1.19E+03	3.54E-02
Vinyl chloride	2.22E+00	9.57E-01	3.19E+00	9.50E-05