Revised Final

Baseline Risk Assessment

Camp Allen Landfill
Norfolk Naval Base
Norfolk, Virginia

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Prepared For:

Department of the Navy
Atlantic Division
Naval Facilities
Engineering Command
Norfolk, Virginia

Under the

LANTDIV CLEAN Program

Comprehensive Long-Term
Environmental Action Navy

Reference:
Contract
N62470-89-D-4814

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February 1995

Baker

FOSTER WHEELER
FOSTER WHEELER ENVIRESPONSE, INC.

WESTON
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CAMP ALLEN LANDFILL, AREAS A AND B
NAVAL BASE, NORFOLK, VIRGINIA

CONTRACT TASK ORDER 0084

FEBRUARY 24, 1995

Prepared For:

DEPARTMENT OF THE NAVY
ATLANTIC DIVISION
NAVAL FACILITIES
ENGINEERING COMMAND
Norfolk, Virginia

Under:

LANTDIV CLEAN PROGRAM
Contract N62470-89-D-4814

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LIST OF ACRONYMS AND ABBREVIATIONS

ABS	Absorption Factor
AF	Adherence Factor
AT_c	Averaging time (carcinogenic)
AT_{nc}	Averaging time (noncarcinogenic)
ATSDR	Agency for Toxic Substances Disease Registry
AWQC	Ambient Water Quality Criteria
Baker	Baker Environmental, Inc.
BCF	Bioconcentration Factor
BW	Body Weight
CDI	Chronic Daily Intake
CF	Conversion Factor
CLEAN	Comprehensive Long-Term Environmental Action Navy
CLP	Contract Laboratory Program
cm	centimeter
COPC	Chemical of Potential Concern
CRAVE	Carcinogenic Risk Assessment Verification Endeavor
CSF	Carcinogenic Slope Factor
C_x	Concentration of Chemical x
CFR	Code of Federal Regulations
d	day
4,4'-DDT	Dichlorodiphenyl trichloroethane
4,4'-DDD	Dichlorodiphenyl dichloroethane
4,4'-DDE	Dichlorodiphenyl chloroethane
DoN	Department of the Navy
(D)	duplicate
ED	Exposure Duration
EF	Exposure Frequency
Eh	Oxidation-reduction potential
ft	feet
GW	groundwater
HA	Health Advisory
HEAST	Health Effects Assessment Summary Tables
HI	Hazard Index
HQ	Hazard Quotient
hr	hour

ICR	Incremental (Lifetime) Cancer Risk
ILCR	Incremental Lifetime Cancer Risk
IR	Ingestion/Inhalation Rate
IRIS	Integrated Risk Information System
K_{ow}	Octanol-water partitioning coefficient
K_{oc}	Organic carbon partitioning coefficient
LANTDIV	Atlantic Division, Naval Facilities Engineering Command
LOAEL	Lowest Observed Adverse Effect Level
m	meter
m-	meta
MCE/FMFLANT	Marine Corps Camp Elmore/Fleet Marine Force, Atlantic
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
mg/kg	milligrams per kilogram
mg/L	milligrams per liter
MI	Mobility Index
MSWLF	Municipal Solid Waste Landfill
ND	Not Detected
NOAA	National Oceanic and Atmospheric Administration
NOAEL	No Observable Adverse Effect Level
NOEL	No Observable Effect Level
NWI	National Wetlands Inventory
o-	ortho
p-	para
PAHs	polynuclear aromatic hydrocarbons
PC	Permeability Constant
PCBs	polychlorinated biphenyls
pH	pH
RA	Risk Assessment
RfC	Reference Concentration
RfD	Reference Dose
RI	Remediation Investigation
RMC	Reasonable Maximum Exposure
s	standard deviation
S	water solubility
SA	surface area
SW	surface water

t	student-t statistic
u	sample number
UCL	Upper Confidence Limit
UF	Uncertainty Factor
µg/L	micrograms per liter
µg/kg	micrograms per kilogram
USEPA	United States Environmental Protection Agency
$\bar{x}$	mean

SECTION 1

1.0 INTRODUCTION

As part of the Remedial Investigation (RI) for the Camp Allen Landfill, Naval Base Norfolk, the potential public health risks associated with potential current and future exposure to affected environmental media in Areas A and B were evaluated. This volume of the RI summarizes the findings with respect to the potential risks resulting from existing conditions at the sites, and has been developed using the analytical results presented in the "Remedial Investigation Report," Final, Camp Allen Landfill, Naval Base, Norfolk, Virginia, (Baker, July 1994).

The risks addressed in this baseline risk assessment (RA) represent the potential public health risks which might result from taking no remedial action at the Camp Allen Landfill site. In addition, an ecological assessment for Areas A and B has also been performed.

1.1 Overview, Areas A and B

Review of the data indicated that the contaminants of concern in various media at both Area A and Area B were primarily volatile organic compounds (VOCs), including vinyl chloride, and heavy metals. The objective of the baseline RA was to estimate the potential human health effects, if any, to humans and ecological receptors exposed to compounds contaminating the sites. More specifically, the baseline RA was performed for the no action scenario which reflects the present conditions without remedial action. Human exposure routes, dosages, associated human health effects and potential ecological effects associated with exposure from the contaminants of concern identified at the sites are estimated. At Area A, civilian employees at the Brig facility, Brig prisoners and local residents west of the Brig facility (both adults and children) were determined to be the populations of concern. At Area B and the area southeast of Area B, elementary school children, workers and local residents (both adults and children) were considered to be the potential receptors of concern.

Potential carcinogenic and noncarcinogenic health effects were estimated. Conservative exposure factor assumptions, the most recent toxicity information, and the most current regulatory criteria and standards were used throughout. Potential ecological effects were also evaluated by a comparison of available analytical data to appropriate surface water and sediment criteria and benthic macroinvertebrate study results .

1.2 Site Background

During the early 1940s, landfilling operations commenced in the Camp Allen area (Camp Allen Landfill). Disposal activities continued until about 1974 primarily in two areas, Area A and Area B (see Figure 1-1).

1.2.1 Area A

Area A of the Camp Allen Landfill is a 45-acre site that was used for the disposal of wastes from the early 1940s until 1975. During this time, significant quantities of municipal, solid, and hazardous wastes were disposed including: general refuse; demolition debris; sludges from metal plating processes, parts cleaning and paint stripping operations; overage chemicals; various chlorinated organic solvents; acids; caustics; paints and paint thinners; pesticides; and asbestos. From approximated waste generation rates, it was estimated that about 40,000 pounds of metals plating sludge, 60,000 pounds of parts cleaning sludge, and 400,000 pounds of paint stripping residue were disposed at Area A.

In the mid-1940s, an incinerator was constructed in the southern portion of the Camp Allen area to burn combustible wastes. This incinerator operated until the mid-1960s. Materials too bulky for the incinerator were burned in Area A of the Camp Allen Landfill.

At present, the majority of Area A is capped with a grass cover to minimize surface erosion. Area A incorporates the Navy Brig facility and a heliport built over a portion of the landfill during the mid-1970s. The area is surrounded by drainage ditches which convey surface water runoff to Willoughby Bay. These drainage ditches drain to the remnants of Bousch Creek, the main channel of which was completely filled and replaced by a network of ditches and channels during the development of Naval Base Norfolk. Additionally, a residential area (Glenwood Park) is located to the west of the site.

1.2.2 Area B

Area B, the eastern portion of the Camp Allen Landfill, received wastes from a 1971 fire at the Camp Allen Salvage Yard. The Salvage Yard is located between Camp Allen Landfill Areas A and

B, and handled lubricating oil, organic solvents, paints and paint thinners, acids, caustics, and pesticides. The Salvage Yard fire occurred in the northern portion of the yard, with residue and debris resulting from this fire buried in the eastern portion of the landfill. At present, the area surrounding the Salvage Yard and Area B is fenced. A wider portion of the drainage ditch, located in the northeast portion of Area B, where water tends to pool, is referred to as the Pond Area. To the southeast of Area B is the Camp Allen Elementary School, as well as military housing.

1.3 Scope of Risk Assessment

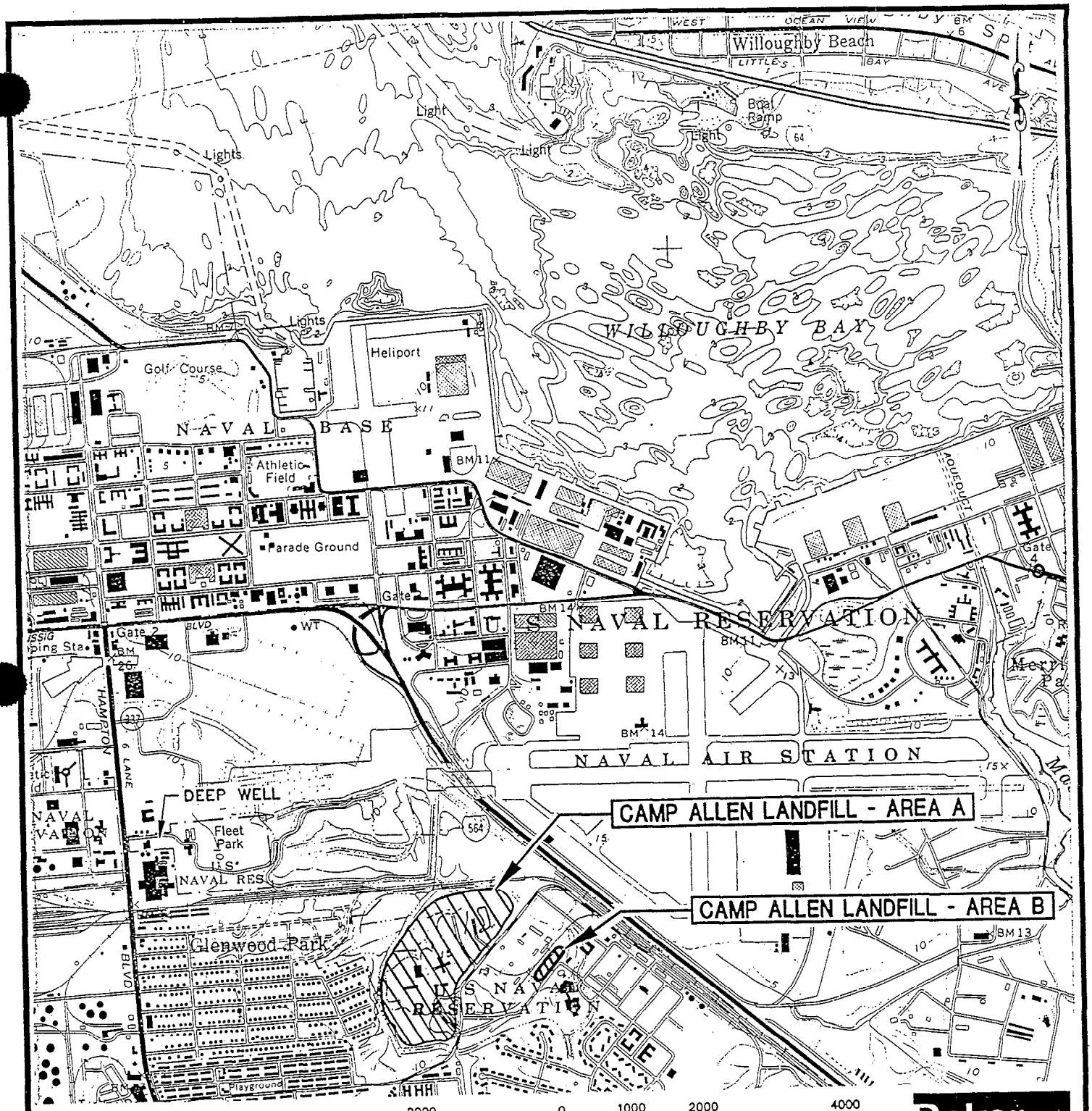
Soil, groundwater, surface water, sediment, and air samples were collected at each area. A comprehensive, quantitative human health evaluation and an ecological evaluation, including the assessment of chemical results from all the media sampled, have been performed for the Camp Allen Landfill Site.

The public health portion of the baseline RA assesses Areas A and B of the Camp Allen Landfill separately because the separate landfills result in two distinct areas of contamination. The risk assessment also evaluates current potential human exposure, as well as future potential human exposure in the event of a base closure and subsequent residential property development. However, base closure at Camp Allen is unlikely and the "Master Plan, Marine Corps Camp Elmore/Fleet Marine Force, Atlantic (MCE/FMFLANT), Norfolk, Virginia," October 1990, states that future development of the landfill will not occur. Furthermore, the Code of Federal Regulations, Volume 40 (40 CFR), Part 258 (EPA Criteria for Municipal Solid Waste Landfills (MSWLFs), Subpart F: "Closure and Post Closure Care") states that following the closure of all MSWLF units, the owner or operator must record a notation on the deed to the landfill facility property. The notation of the deed must in perpetuity notify any potential purchasers of the property that the property has been used as a landfill and that its future use is restricted so that the integrity of the final cover, liner(s) or other components of the containment system are not disturbed. Future potential risk values generated for future residential property development are, therefore, presented as conservative estimates of potential human health effects.

Ecological concerns were also evaluated throughout Areas A and B of the Camp Allen Landfill. Potential ecological effects were evaluated using available analytical data in conjunction with

terrestrial wildlife surveys and the results of a benthic macroinvertebrate study conducted in June of 1993.

SECTION 1.0 FIGURES



SOURCE: U.S.G.S. TOPOGRAPHIC MAP
NORFOLK NORTH QUADRANGLE
1965 (PHOTOREVISED 1986)

Baker
Baker Environmental, Inc.

FIGURE 1-1
LOCATION MAP
CAMP ALLEN LANDFILL

NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

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SECTION 2

2.0 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN

A discussion of laboratory analytical results and nature and extent of constituent contamination was presented in Sections 5 and 6 of the RI report. In the RI report, chemicals detected in environmental media were discussed with respect to applicable Federal and Commonwealth of Virginia criteria and/or standards. In these sections, a preliminary account of chemicals of potential concern (COPCs) was presented. Chemicals detected in environmental media sampled during the RI were reevaluated to select COPCs for evaluation in the baseline RA. Chemicals selected as COPCs were retained for quantitative evaluation. Chemicals not selected as COPCs are discussed in the uncertainties section of the baseline RA (Section 7.0).

COPC selection was based on the information provided in the USEPA Region III Technical Guidance on the Screening of Exposure Pathways and Selection of Contaminants of Concern, dated January 1993 (USEPA Region III, 1993) and USEPA's Risk Assessment Guidance for Superfund (RAGS), Volume I. Human Health Evaluation Manual (Part A), Interim Final, December 1989 (USEPA, 1989).

COPC selection was completed for each environmental medium and area of concern using analytical data obtained during Rounds 2 and 3 of the RI. The Camp Allen Landfill study area has been divided into three evaluation units as a result of past disposal practices. Area A encompasses the western portion of the Camp Allen Landfill. This area was used for the disposal of municipal, solid, and hazardous wastes from the early 1940s until 1975. The Glenwood Park neighborhood (residential area) is situated to the west of Area A. Numerous Glenwood Park residents are serviced by nonpotable residential wells for uses such as lawn watering and car washing. A residential well survey and analytical testing of these wells were conducted to evaluate the potential affects of the landfill on well water quality. The eastern portion of the Camp Allen Landfill (Area B) received wastes from a 1971 Salvage Yard fire. The Camp Allen Salvage Yard, which is currently active, separates the two landfill areas. Figure 2-1 presents Landfill Areas A, B, and the residential area which are of interest in the baseline RA.

Landfill Area B has been further subdivided. Subdivisions were determined based on the potential current activities within Area B and the potential for human exposure. Area B subunits include the Pond Area and the Camp Allen Elementary School Area.

2.1 COPC Selection Criteria

Both of the previously mentioned guidances provide a number of criteria by which chemical data can be evaluated. The primary criteria used in selecting a chemical as a COPC at the Camp Allen Landfill included comparison of maximum detected concentrations with USEPA Region III risk-based COPC screening concentrations, as derived in accordance with USEPA Region III Technical Guidance on the Screening of Exposure Pathways and Selection of Contaminants of Concern (USEPA Region III, January 1993), chemical prevalence, and site history.

Comparison of maximum detected sample concentrations with COPC screening concentrations was used as the primary selection criterion for chemicals detected in soils, groundwater , and air. Since no sediment COPC screening concentrations have been derived by USEPA Region III, residential soil COPC screening concentrations were applied as a secondary criterion for chemicals detected in sediments. Likewise, no surface water COPC screening concentrations are available for comparison with detected surface water concentrations. Therefore, the selection of surface water and sediment COPCs was conducted using other primary criteria and applied to detected maximum concentrations, as will be discussed in later paragraphs.

The prevalence of a chemical detected in a given environmental medium within a particular area of concern, as well as the history of site-related activities are other important criteria applied in selecting COPCs at the Camp Allen Landfill site. The prevalence of a chemical in an environmental medium can be described by the frequency and concentration with which it is detected. A detection frequency of 5 percent (1 positive detection in 20 samples) was the minimum detection frequency considered in the selection of COPCs in data sets comprised of 20 or more samples. Furthermore, chemicals reliably associated with past disposal activities at the Camp Allen Landfill were retained for further evaluation (i.e., trichloroethene, polychlorinated biphenyls, etc.). In conjunction with concentration comparisons with USEPA Region III risk-based COPC screening concentrations and evaluations of chemical prevalence and site history, a comparison to available state and Federal standards and criteria was conducted. Criteria used in selecting COPCs include available State and Federal Maximum Contaminant Levels and Maximum Contaminant Level Goals (MCLs and MCLGs, respectively).

Surface water criteria included USEPA Ambient Water Quality Criteria (AWQC) established for the protection of humans consuming water and aquatic organisms, and those established for the protection of humans consuming only aquatic organisms. The AWQC values recalculated from USEPA's Integrated Risk Information System (IRIS), which were derived based on a risk level of 1×10^{-6}, were used as the primary surface water criteria. If recalculated values were not available, then USEPA's published AWQC values were applied. If neither of these sets of criteria were available for the protection of humans consuming water and aquatic organisms, and for the protection of humans consuming only aquatic organisms, then acute or chronic freshwater or marine criteria were used.

Sediment criteria included screening values compiled by the National Oceanic and Atmospheric Administration (NOAA) for evaluating the potential for chemical constituents in sediments to cause adverse biological effects. Those NOAA concentrations stratified into the "effects range - median" (referred to as the ER-M concentrations) were used as the primary criteria for selecting sediment COPCs. A more detailed description of the NOAA sediment screening values will be provided in later paragraphs.

No site-specific background samples were collected for comparison with sample concentration. However, Table 2-1 presents background concentrations for metals in soils from various literature sources. The following paragraphs describe the criteria used for comparison to constituents detected at the Camp Allen Landfill site.

USEPA Region III COPC Screening Concentrations - Risk-based COPC screening concentrations were derived by USEPA, Region III in January of 1993 and provided in tabular format to support selection of COPCs and address two major limitations in the COPC selection process presented in RAGS. First, using COPC screening concentrations prioritizes chemical toxicity and focuses the risk assessment on those COPCs and potential exposure routes. Second, using the screening concentrations provides an absolute comparison of potential risks associated with the presence of a COPC in a given medium.

COPC screening concentrations are derived using conservative USEPA promulgated default values and the most recent toxicological criteria available. COPC screening concentrations for potentially carcinogenic and noncarcinogenic chemicals are individually derived based on a target incremental

lifetime cancer risk (ILCR) of 1×10^{-6} and a target hazard quotient of 0.1, respectively. For potential carcinogens, the toxicity criteria applicable to the derivation of COPC screening concentrations are oral and inhalation cancer slope factors; for noncarcinogens, they are chronic oral and inhalation reference doses. These toxicity criteria are subject to change as more updated information and results from the most recent toxicological/epidemiological studies become available. Therefore, the use of toxicity criteria in the derivation of COPC screening concentrations requires that the screening concentrations be updated periodically to reflect changes in the toxicity criteria.

Since only one set of COPC screening concentration tables have been issued by USEPA (January 1993), the values from these tables can be updated by incorporating information from another set of tables containing risk-based concentrations (RBCs) that are issued by USEPA Region III on a quarterly basis. The RBCs are derived using similar equations and USEPA promulgated default exposure assumptions that were used to derive the original set (January, 1993) COPC screening concentrations. The only difference in the derivation methodologies for the COPC screening concentrations and the RBCs is that RBCs for carcinogens have an age-adjusted ingestion rate and RBCs for noncarcinogens are derived based on a target hazard quotient of 1.0. An updated set of COPC screening concentrations can, therefore, be attained each quarter by using the carcinogenic RBCs issued quarterly by USEPA Region III and dividing the accompanying noncarcinogenic RBCs by a factor of 10. All COPC screening concentrations applied to the site are presented in the COPC Selection Summary Tables in Appendix A of this report (Tables A-1 through A-22).

Maximum Contaminant Levels (MCLs) - MCLs are potentially enforceable standards for public water supplies promulgated under the Safe Drinking Water Act and are designed for the protection of human health. MCLs have been adopted as enforceable standards for public drinking water systems, and apply to drinking water supplies consumed by a minimum of 25 persons. They have been developed for the prevention of human health effects associated with lifetime exposure (70 year lifetime) of an average adult (70 kg) consuming 2 liters of water per day. MCLs also consider the technical and economic feasibility of removing the constituent from a public water supply. State and Federal MCLs for constituents detected at the site are presented in Table 2-2.

Maximum Contaminant Level Goals (MCLGs) - MCLGs are usually nonenforceable guidelines based entirely on the potential for human health effects. The MCLs have been set as close to the MCLGs as is considered technically and economically feasible. MCLGs are specified as zero for

carcinogenic substances, based on the assumption of nonthreshold toxicity, and do not consider the technical or economic feasibility of achieving these goals. In addition, MCLGs for noncarcinogens are set based upon chronic toxicity or other data. Federal MCLGs are presented in Table 2-2.

Health Advisories (HAs) - HAs are guidelines developed by the USEPA Office of Drinking Water for non-regulated constituents in drinking water. These guidelines are designed to consider both acute and chronic toxic effects in children (assumed body weight of 10 kg) who consume 1 liter of water per day, and adults (assumed body weight of 70 kg) who consume 2 liters of water per day. HAs are generally available for acute (1 day), subchronic (10 days), and chronic (longer term) exposure scenarios. These guidelines are designed to consider only threshold effects and, as such, are not acceptable levels of potential human carcinogens. HAs are presented in Table 2-2.

Federal Ambient Water Quality Criteria (AWQC) AWQC are nonenforceable regulatory guidelines and are of primary utility in assessing acute and chronic toxic effects in aquatic organisms. They may also be used for identifying the potential for human health risks. AWQCs consider acute and chronic effects in both freshwater and saltwater aquatic life, and potential carcinogenic and noncarcinogenic health effects in humans from ingestion of both water (2 liters day) and aquatic organisms (6.5 grams day), or from ingestion of water alone (2 liters day). Published AWQCs available for the protection of human health from potential carcinogenic substances are based on an incremental cancer risk range of one additional case of cancer in an exposed population of 10,000,000 to 100,000 persons (i.e., the 10^{-7} to 10^{-5} range). Recalculated values derived from IRIS are based on an incremental lifetime cancer risk of one additional case of cancer in an exposed population of 1,000,000 persons (i.e., 1×10^{-6}). Federal AWQCs derived for the protection of human health (values recalculated from IRIS) are presented in Table 2-3. Federal acute and chronic AWQCs established for the protection of freshwater and marine organisms are presented in Table 2-4.

State of Virginia Water Quality Standards (WQS) - The WQSs are State-enforceable standards used for identifying the potential for human health risks. WQSs protective of human health consider potential carcinogenic and noncarcinogenic health effects in humans from ingestion of both water (2 liters day) and aquatic organisms (6.5 grams day), or from ingestion of water alone (2 liter day). State WQSs available for the protection of human health from potential carcinogenic substances are derived based on an incremental lifetime cancer risk of one additional case of cancer in an exposed

population of 100,000 persons (i.e., 1×10^{-5}). State WQSs derived for the protection of human health are presented in Table 2-3. State acute and chronic WQSs established for the protection of freshwater and marine organisms are presented in Table 2-4.

2.2 Selection of COPCs

Five environmental media were investigated at the Camp Allen Landfill during the recent RI activities. These include soils; groundwaters; surface waters; sediments; and air. The selection of soil COPCs was stratified for surface and subsurface soils. Tables A-1 through A-22 in Appendix A present the selection of COPCs for each environmental medium and area of concern based on comparisons of Region III COPC screening concentrations and other criteria with maximum detected concentrations, and frequencies of detection. Information is presented in these tables only for those constituents detected at least once in the medium of interest. Other statistical information presented in the tables, i.e., arithmetic mean concentration, etc. were not used for selecting COPCs, but rather for determining exposure concentrations. Determination of exposure concentrations will be discussed in Section 3.0.

The following paragraphs discuss the selection of COPCs in both Areas A and B of the Camp Allen Landfill. Sample locations analytical results and corresponding figures are presented in the RI report.

2.2.1 Surface Soils

Surface soil samples collected from both Areas A and B were analyzed for selected volatile organic compounds (VOCs), semivolatile organic compounds (SVOCs), pesticides, PCBs, and inorganic compounds (metals). The COPC selection summaries for surface soils in Areas A and B are presented in Tables A-1 through A-3.

2.2.1.1 Area A Landfill

Surface soil samples were obtained from five locations within Area A. Table A-1 shows that no VOCs were detected in any of these samples. However, polycyclic aromatic hydrocarbons (PAHs), including benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene,

chrysene, fluoranthene, indeno(1,2,3-cd)pyrene, phenanthrene, and pyrene were detected at concentrations less than the corresponding Region III COPC screening concentrations. PAHs are a ubiquitous family of constituents which occur both naturally and anthropogenically by the incomplete combustion of fossil fuels, and are usually detected as a mixture of individual compounds. Since these PAHs were detected at concentrations less than the COPC screening concentrations by up to several orders-of-magnitude, they were not retained for further evaluation either individually, or as a family of compounds, in the baseline RA.

Trace levels of pesticides were also detected in surface soils collected within Area A. Since no pesticide was detected at a concentration exceeding the Region III COPC screening levels, pesticides were not retained as COPCs in Area A soils. However, aroclor-1260, a PCB, was detected in four of five samples at a maximum concentration of 420 μg/kg in Area A soils, which exceeds the corresponding Region III COPC screening concentration of 83 μg/kg. Because aroclor-1260 values exceed the corresponding Region III COPC screening concentration, aroclor-1260 was retained for further evaluation in the baseline RA.

Metals were detected in all surface soil samples collected. The maximum detected concentrations of aluminum (9,880 mg/kg), arsenic (70 mg/kg), barium (1,050 mg/kg), cadmium (88.9 mg/kg), chromium (121 mg/kg), copper (477 mg/kg), manganese (128 mg/kg), thallium (0.92 mg/kg), and vanadium (78.7 mg/kg) exceed the corresponding Region III COPC screening concentrations. In addition, the maximum detected concentration of lead (683 mg/kg) exceeds the USEPA residential soil screening level of 400 mg/kg. Therefore, aluminum, arsenic, barium, cadmium, chromium, copper, manganese, thallium and vanadium were retained as COPCs for quantitative evaluation in the baseline RA. Lead was retained for qualitative evaluation in the baseline RA since no toxicity criteria have been established for the quantitative risk evaluation of this metal. Concentrations of arsenic, cadmium and lead also exceed literature native background soil concentrations presented in Table 2-1.

In summary, the following constituents have been retained as COPCs for surface soils in the Area A landfill:

- Aroclor-1260
- Aluminum

- Arsenic
- Barium
- Cadmium
- Chromium
- Copper
- Lead
- Manganese
- Thallium
- Vanadium

2.2.1.2 Area B Landfill

A total of eight surface soil locations were sampled in Area B. Samples collected from four locations in the landfill and pond area (inside of the fenceline) were analyzed for VOCs, SVOCs, pesticides, PCBs, and inorganic compounds. Three samples collected from the school area soil samples were analyzed for only inorganics, while one sample (collected from location SSB-09 outside of the landfill fenceline, across the road from the elementary school) was analyzed for VOCs, SVOCs, pesticides, PCBs, and inorganic compounds.

Landfill and Pond Area

Table A-2 summarizes the selection of COPCs for the landfill and pond area. VOCs, namely methylene chloride and 2-butanone, were each detected with a frequency of 25 percent, but were also detected in corresponding laboratory and sampling blanks (i.e., field, rinsate, and/or trip). Furthermore, maximum detected concentrations of these chemicals are less than the respective Region III COPC screening concentrations. Therefore, these compounds were not retained as COPCs.

PAHs and pesticides were detected at approximately the same concentrations detected in Area A samples, supporting the assumption that these classes of compounds are either present from sources other than the Camp Allen Landfill or are ubiquitous to the area. All maximum detected concentrations were less than the corresponding Region III COPC screening concentrations; therefore, these chemicals were not retained as surface soil COPCs in the landfill and pond area.

Aroclor-1260 was detected in all four surface soil samples collected from the landfill and pond area with a maximum concentration (780 µg/kg) exceeding the corresponding Region III COPC

screening concentration. Therefore, this compound has been retained as a COPC for further evaluation.

Arsenic, cadmium, chromium, and manganese, were detected in all four surface soil samples collected from the landfill and pond area. The maximum detected concentrations of these metals (11.6 mg/kg, 20.5 mg/kg, 44.3 mg/kg, and 102 mg/kg, respectively) exceed the corresponding Region III COPC screening concentrations. As a result, these metals were retained as COPCs for further evaluation in the baseline RA.

In summary, the following constituents have been retained as COPCs for surface soils in the Area B landfill and pond:

- Aroclor-1260
- Arsenic
- Cadmium
- Chromium
- Manganese

Elementary School Area

Table A-3 shows that only pesticides and inorganics were detected in surface soil samples collected from within the elementary school area. The maximum detected concentrations of the pesticides were less than corresponding Region III COPC screening concentrations, and consequently, were not retained as COPCs. However, the maximum detected concentrations of arsenic (25.1 mg/kg), chromium (869 mg/kg), manganese (61.2 mg/kg), and vanadium (128 mg/kg) exceeded the corresponding Region III COPC screening concentrations. As such, these elements were retained as surface soil COPCs in this area.

In summary, the following constituents have been retained as COPCs for surface soils in the elementary school area:

- Arsenic
- Chromium
- Manganese
- Vanadium

2.2.2 Subsurface Soils

Subsurface soil samples collected from both Areas A and B were analyzed for VOCs, SVOCs, pesticides, PCBs, and inorganics (metals - Area B only). The COPC selection summaries for subsurface soils in Areas A and B are presented in Tables A-4 and A-5, respectively.

2.2.2.1 Area A Landfill

Subsurface soil samples were collected from ten locations within Area A. Table A-4 shows that eleven VOCs were detected in these samples. Only the maximum concentration for toluene (3,000,000 µg/kg) exceeds the corresponding Region III COPC screening concentration (1,600,000 µg/kg). Toluene was therefore retained as a subsurface soil COPC in Area A.

SVOCs, including PAHs were detected at frequencies of detection greater than 5 percent. Benzo(a)pyrene was detected at 72J µg/kg which approaches the Region III COPC screening value of 88 mg/kg. Other potentially carcinogenic PAH concentrations were approximately one order-of-magnitude lower than their corresponding screening values. Therefore, the potentially carcinogenic PAHs were not retained for further evaluation. Noncarcinogenic PAHs including naphthalene, 2-methylnaphthalene, and acenaphthene were also detected at concentrations well below their corresponding COPC soil screening values. Because maximum detected concentrations were less than the corresponding Region III COPC screening concentrations, PAHs were not retained as subsurface soil COPCs.

Other SVOCs including methylphenols and phthalates were detected but at concentrations below corresponding Region III COPC screening values. These chemicals were not retained as COPCs.

Dieldrin, Aroclor-1254 and Aroclor-1260 were detected at frequencies greater than 5 percent, and at maximum concentrations of 89 µg/kg, 1,600 µg/kg and 1,800 µg/kg, respectively. These values exceed the corresponding Region III COPC screening concentrations. Therefore, these compounds were retained as COPCs for quantitative evaluation of risks associated with subsurface soils.

In summary, the following constituents have been retained as COPCs for subsurface soils in the Area A landfill:

- Toluene
- Aroclor-1254
- Aroclor-1260
- Dieldrin

2.2.2.2 Area B Landfill

Subsurface soil samples were collected from a total of ten locations throughout the area designated Area B, which consists of both the landfill and pond area and elementary school area. Unlike the selection of surface soil COPCs, which were selected for the landfill and pond area and elementary school area separately, the selection of subsurface soil COPCs was done so as to evaluate both of these areas as one large area, since under a future exposure scenario, it is assumed that the probability of exposure will be uniform over all areas of concern in Area B. The subsurface soil exposure scenarios will be discussed in Section 3.0 of this report.

Table A-5 shows that, with the exception of carbon disulfide, the same VOCs were detected in the subsurface soils of Area B. Additional VOCs detected in Area B include: 1,1-dichloroethane, 1,2-dichloroethane, trichloroethene, and vinyl chloride. All detected VOCs occurred at a frequency greater than 5 percent. The maximum detected concentrations of trichloroethene (3,100 µg/kg) and vinyl chloride (16 µg/kg) exceeded corresponding Region III COPC screening concentrations; therefore, these two compounds were retained as subsurface soil COPCs for Area B.

SVOCs, including phthalates, noncarcinogenic PAHs, cresols, and 1,2-dichlorobenzenes were detected in subsurface soils at frequencies exceeding 5 percent. However, none of the maximum detected concentrations for these compounds exceeded Region III COPC screening concentrations; therefore no SVOCs were retained as subsurface soil COPCs in Area B.

4,4'-DDD, 4,4'-DDE, Aroclor-1254, dieldrin, endosulfans I and II, and endrin aldehyde were detected in subsurface soils at frequencies exceeding 5 percent. Of these pesticides and PCBs, 4,4'-DDD, Aroclor-1254 and dieldrin were detected at maximum concentrations (3,800 µg/kg, 9,500 µg/kg and 1,500 µg/kg, respectively) exceeding Region III COPC screening concentrations. These chemicals were retained as subsurface soil COPCs in Area B.

Inorganics detected in subsurface soils occurred at frequencies equal to, or greater than 60 percent. The maximum detected concentrations of aluminum (15,500 mg/kg), antimony (8 mg/kg), arsenic (60.5 mg/kg), barium (1,480 mg/kg), beryllium (5.6 mg/kg), manganese (63.5 mg/kg), thallium (2 mg/kg), and vanadium (149 mg/kg) exceed corresponding Region III COPC screening concentrations. These metals were consequently retained as COPCs for further quantitative evaluation.

In summary, the following constituents have been retained as COPCs for subsurface soils in the Area B landfill:

- Trichloroethene
- Vinyl Chloride
- 4,4'-DDD
- Aroclor-1254
- Dieldrin
- Aluminum
- Antimony
- Arsenic
- Barium
- Beryllium
- Manganese
- Thallium
- Vanadium

2.2.3 Columbia (Shallow) Aquifer Groundwater

Tables A-6 and A-7 summarize the COPC selections performed for constituents detected in the shallow aquifer. All samples were analyzed for VOCs, SVOCs, pesticides, PCBs, and unfiltered and filtered inorganics.

2.2.3.1 Area A

Table A-6 indicates that twelve VOCs were detected in groundwater samples collected from Area A. All detected VOCs occurred with a frequency of greater than 5 percent. Of the twelve VOCs, 1,2-dichloroethane (3 µg/L), 1,2-dichloroethene (6,100 µg/L), 2-butanone (4,300 µg/L), 4-methyl-2-pentanone (16,000 µg/L), benzene (310 µg/L), methylene chloride (57 µg/L), tetrachloroethene (620 µg/L), toluene (5,400 µg/L), trichloroethene (1,800 µg/L), and vinyl chloride (3,300 µg/L) were

detected at concentrations exceeding their Region III COPC screening concentrations. These VOCs were, therefore, retained as COPCs in shallow groundwater.

SVOCs, including phthalates, PAHs, cresols, and 1,2-dichlorobenzenes were detected in shallow groundwater samples at frequencies exceeding 5 percent. Maximum detected concentrations of 2-methylphenol (1,800 µg/L), 4-methylphenol (21,000 µg/L), 2,4-dimethylphenol (1,400 µg/L), and bis(2-ethylhexyl)phthalate (13 µg/L) exceeded corresponding Region III COPC screening concentrations and were retained as COPCs.

4,4'-DDD, aldrin, gamma-chlordane, and heptachlor epoxide were also detected in shallow groundwater samples at frequencies greater than 5 percent. Aldrin and heptachlor epoxide were retained as COPCs since maximum detected concentrations (0.026 µg/L and 0.14 µg/L, respectively) exceeded Region III COPC screening concentrations.

Of the unfiltered metals detected in the shallow aquifer, antimony, arsenic, barium, beryllium, cadmium, chromium, copper, manganese, nickel, and vanadium were retained because of exceedances of corresponding Region III COPC screening concentrations. Although no Region III COPC screening concentrations, State, or Federal MCLs exist for aluminum, this inorganic was retained for qualitative evaluation in the risk assessment because of prevalence (100 percent frequency of detection) and the magnitude of the maximum detected aluminum concentration (132,000 µg/L). Unfiltered lead was also retained for qualitative evaluation since the maximum detected concentration (381 µg/L) exceeds the State MCL and Federal action level of 50 µg/L and 15 µg/L, respectively.

Arsenic, barium and manganese were retained as COPCs in filtered groundwater samples for quantitative evaluation. Lead was also retained for qualitative evaluation in the baseline RA, because filtered maximum detected concentrations exceed Region III COPC screening concentrations, State, and Federal MCLs. The maximum concentration detected for dissolved lead exceeded the State MCL and the Federal action level.

In summary, the following constituents have been retained as COPCs for shallow groundwater underlying the Area A landfill:

- 1,2-Dichloroethane
- 1,2-Dichloroethene
- 2-Butanone
- 4-Methyl-2-Pentanone
- Benzene
- Methylene Chloride
- Tetrachloroethene
- Toluene
- Trichloroethene
- Vinyl Chloride
- 2-Methylphenol
- 2,4-Dimethylphenol
- 4-Methylphenol
- Bis(2-ethylhexyl)phthalate
- Aldrin
- Heptachlor Epoxide
- Aluminum
- Antimony
- Arsenic
- Barium
- Beryllium
- Cadmium
- Chromium
- Copper
- Lead
- Manganese
- Nickel
- Vanadium
- Dissolved Arsenic
- Dissolved Barium
- Dissolved Lead
- Dissolved Manganese

2.2.3.2 Residential Wells

A total of 57 wells were sampled and analyzed for volatile organic compounds in the Glenwood Park community, which is located to the west of the Camp Allen Landfill. Residential wells were not sampled for less environmentally mobile semivolatile organics, pesticides, PCBs, or inorganic analyses. Three of the 57 well locations (well numbers 22, 39, and 56) contained detectable levels of volatile organics. The nearest residential well is approximately 500 feet from the estimated western boundary of the Area A landfill. These residential wells are reportedly screened in the shallow zone.

Well location 39 displayed the presence of 2-butanone (76 μg/L) which could be attributable to sampling or analytical activities, and therefore, may not be associate with past site activities. 2-Butanone is a contaminant of some reagent grade methanols, thus making the presence of 2-butanone in environmental samples questionable. Furthermore, the detected concentration of 2-butanone is less than the corresponding Region III COPC screening concentration for tap water (2,200 μg/L) and was not retained as a COPC for further evaluation in the baseline RA.

Tetrachloroethene was detected in a groundwater sample, collected from well location 22, at a concentration of 10 μg/L. The presence of tetrachloroethene at this location is suspicious since analytical data for wells situated between Area A and well location 22 (well locations 23, 37 and 43) did not indicate detectable levels of the VOC. In addition, the detected concentration is equivalent to the detection limit reported for water samples. However, VOC concentrations may vary greatly between wells, and because the detected concentration exceeds the corresponding Region III COPC screening concentration for tap water (1.1 μg/L), tetrachloroethene was retained as a COPC for quantitative evaluation in the baseline RA.

Well location 56 displayed the presence of 1,2-dichloroethane (38 μg/L) and 2-butanone (10 μg/L). The detected concentration of 2-butanone is less than the corresponding Region III COPC screening concentration for tap water (2,200 μg/L). Therefore, 2-butanone was not retained as a COPC for further evaluation in the baseline RA. The detected concentration of 1,2-dichloroethane exceeded the corresponding Region III COPC screening concentration for tapwater (0.12 μg/L), and consequently, was retained as a COPC in the baseline RA.

In conclusion, the following VOCs were retained as COPCs in the shallow aquifer underlying the Glenwood Park residential area:

- Tetrachloroethene
- 1,2-Dichloroethane

2.2.3.3 Area B

Table A-7 indicates that thirteen VOCs were detected in shallow groundwater samples collected from Area B. All detected VOCs occurred at a frequency greater than 5 percent. Of the thirteen

VOCs, 1,1-dichloroethene (51 μg/L), 1,2-dichloroethane (180 μg/L), 1,2-dichloroethene (1,600 μg/L), benzene (410 μg/L), chlorobenzene (48 μg/L), tetrachloroethene (10 μg/L), trichloroethene (520 μg/L), and vinyl chloride (940 μg/L) were detected at concentrations exceeding their respective Region III COPC screening concentrations. These VOCs were therefore retained as COPCs in Area B shallow groundwater.

Of seventeen SVOCs detected in the shallow groundwater underlying Area B, the following were detected with maximum concentrations exceeding corresponding Region III COPC screening concentrations: 1,4-dichlorobenzene (3 μg/L), bis(2-chloroethyl)ether (8 μg/), bis(2-ethylhexyl)phthalate (5 μg/L). These compounds were consequently retained as COPCs.

Dieldrin, gamma-BHC and heptachlor epoxide were detected in shallow groundwater samples at frequencies greater than 5 percent. The maximum detected concentrations of these pesticides were 0.043 μg/L, 0.15 μg/L and 0.006 μg/L, respectively, exceeded Region III COPC screening concentrations. Dieldrin, gamma-BHC and heptachlor epoxide were, therefore, retained as COPCs in the shallow groundwater underlying Area B.

Of the unfiltered metals detected in the shallow aquifer, aluminum, antimony, arsenic, barium, beryllium, cadmium, chromium, copper, manganese, mercury, nickel, vanadium, and zinc were retained as COPCs due to prevalence and exceedances of corresponding Region III COPC screening concentrations by maximum detected concentrations. Unfiltered lead was also retained for qualitative evaluation since the maximum detected concentration (1,020 μg/L) exceeds the State MCL and Federal action level (50 μg/L and 15 μg/L, respectively).

Antimony, arsenic, chromium, manganese, and vanadium were retained as dissolved COPCs for quantitative evaluation. Lead was retained for qualitative evaluation in the baseline RA, since filtered maximum detected concentrations exceed Region III COPC screening concentrations, State, and Federal MCLs.

In summary, the following constituents have been retained as COPCs for shallow groundwater underlying the Area B landfill:

- 1,1-Dichloroethene

- 1,2-Dichloroethane
- 1,2-Dichloroethene
- Benzene
- Chlorobenzene
- Tetrachloroethene
- Trichloroethene
- Vinyl Chloride
- 1,4-Dichlorobenzene
- Bis(2-chloroethyl)ether
- Bis(2-ethylhexyl)phthalate
- Dieldrin
- Gamma-BHC
- Heptachlor Epoxide
- Aluminum
- Antimony
- Arsenic
- Barium
- Beryllium
- Cadmium
- Chromium
- Copper
- Lead
- Manganese
- Mercury
- Nickel
- Vanadium
- Zinc
- Dissolved Antimony
- Dissolved Arsenic
- Dissolved Chromium
- Dissolved Manganese
- Dissolved Vanadium

2.2.4 Yorktown (Deep) Aquifer Groundwater

Tables A-8 and A-9 summarize the COPC selections for constituents detected in the deep aquifer. All samples were analyzed for VOCs, SVOCs, pesticides, PCBs, and unfiltered and filtered inorganics.

2.2.4.1 Area A

Table A-8 shows that ten VOCs were detected in deep groundwater samples collected from the Yorktown Aquifer underlying Area A. All detected VOCs occurred at frequencies greater than 5

percent. Of the detected VOCs, 1,2-dichloroethane (38 μg/L), 1,2-dichloroethene (540 μg/L), benzene (3 μg/L), chloroform (8 μg/L), tetrachloroethene (4 μg/L), trichloroethene (100 μg/L), and vinyl chloride (100 μg/L) were detected at concentrations exceeding their respective Region III COPC screening values. These VOCs were therefore retained as COPCs in the deep groundwater underlying Area A.

Eight SVOCs were detected in deep groundwater samples at frequencies exceeding 5 percent. Although bis(2-chloroethyl)ether was detected in only one sample out of seventeen samples analyzed, it was retained as a COPC in deep groundwater since the maximum detected concentration (2 μg/L), exceeded the corresponding Region III COPC screening concentration. Bis(2-chloroethyl)ether was not detected in any other environmental media at the Camp Allen Landfill site.

4,4'-DDT and heptachlor epoxide were the only pesticides detected in deep groundwater samples collected from Area A. Both were detected at frequencies greater than 5 percent. However, only heptachlor epoxide was retained as a COPC since the maximum detected concentration (0.0065 μg/L) exceeded the corresponding Region III COPC screening concentration (0.0012 μg/L).

Of the unfiltered metals detected in the deep aquifer, aluminum, arsenic, cadmium, chromium, manganese, thallium, and vanadium were retained as unfiltered COPCs due to prevalence and exceedances of corresponding Region III COPC screening concentrations by maximum detected concentrations. Unfiltered lead was also retained as a COPC since the maximum detected concentration (44.2 μg/L) exceeds the established Federal action level (15 μg/L). Due to the lack of available toxicity criteria, unfiltered lead will be qualitatively evaluated in the baseline RA.

Dissolved arsenic and manganese were retained as COPCs for quantitative evaluation in the baseline RA, since their filtered maximum detected concentrations exceed corresponding Region III COPC screening concentrations.

In summary, the following constituents have been retained as COPCs for deep groundwater underlying the Area A landfill:

- 1,2-Dichloroethane
- 1,2-Dichloroethene

- Benzene
- Chloroform
- Tetrachloroethene
- Trichloroethene
- Vinyl Chloride
- Bis(2-chloroethyl)ether
- Heptachlor Epoxide
- Aluminum
- Arsenic
- Cadmium
- Chromium
- Lead
- Manganese
- Thallium
- Vanadium
- Dissolved Arsenic
- Dissolved Manganese

2.2.4.2 Area B

Table A-9 shows that eight VOCs were detected in deep groundwater samples collected from Area B. All detected VOCs occurred at a frequency greater than 5 percent. Of the eight VOCs, 1,2-dichloroethane (450 μg/L), 1,2-dichloroethene (16 μg/L), benzene (12 μg/L), chloroform (1 μg/L), trichloroethene (35 μg/L), and vinyl chloride (3 μg/L) were detected at concentrations exceeding their respective Region III COPC screening concentrations. These VOCs were therefore retained as COPCs in Area B deep groundwater.

Only two SVOCs were detected in the deep groundwater underlying Area B, diethylphthalate and phenol. Neither of these were retained as COPCs since their maximum detected concentrations were less than corresponding Region III COPC screening concentrations.

4,4'-DDD, dieldrin and heptachlor epoxide were detected in deep groundwater samples at frequencies greater than 5 percent. The maximum detected concentrations of the latter two pesticides (0.009 μg/L and 0.0105 μg/L, respectively) exceeded Region III COPC screening concentrations. Dieldrin and heptachlor epoxide were therefore retained as COPCs in the deep groundwater underlying Area B.

Of the unfiltered metals detected in the deep aquifer underlying Area B, aluminum, antimony, arsenic, barium, beryllium, cadmium, chromium, copper, manganese, nickel, and vanadium were retained as unfiltered COPCs due to prevalence and exceedances of corresponding Region III COPC screening concentrations by maximum detected concentrations. Unfiltered lead was also retained for qualitative evaluation since the maximum detected concentration (183 μg/L) exceeds the State MCL and Federal action level (50 μg/L and 15 μg/L, respectively).

Dissolved arsenic and manganese were retained as COPCs for quantitative evaluation since their filtered maximum detected concentrations exceed Region III COPC screening concentrations.

In summary, the following constituents have been retained as COPCs for deep groundwater underlying the Area B landfill:

- 1,2-Dichloroethane
- 1,2-Dichloroethene
- Benzene
- Chloroform
- Trichloroethene
- Vinyl Chloride
- Dieldrin
- Heptachlor Epoxide
- Aluminum
- Antimony
- Arsenic
- Barium
- Beryllium
- Cadmium
- Chromium
- Copper
- Lead
- Manganese
- Nickel
- Vanadium
- Dissolved Arsenic
- Dissolved Manganese

2.2.5 Surface Waters and Sediments

Surface water and sediment samples collected from both Areas A and B were analyzed for VOCs, SVOCs, pesticides, PCBs and inorganic compounds. Area B surface water and sediment samples

were collected from the landfill and pond area and the elementary school area. Surface water samples were additionally analyzed for dissolved and total inorganic constituents. However, only analytical data for organic and unfiltered inorganic constituents were considered applicable for the evaluation of potential surface water exposures in this baseline RA. Analytical sediment data were acquired for both shallow (0 to 6 inches) and deep sediments.

COPC selection summaries for surface water and sediment samples collected from Areas A and B are presented in Tables A-10 through A-18. Maximum detected surface water concentrations were compared with Federal AWQCs that are protective of human health (recalculated for IRIS). Freshwater and marine acute and chronic AWQCs were used in the absence of human health values. Maximum detected surface water concentrations were not compared with Virginia Water Quality Standards (WQSs) since these are, for the most part, similar or less conservative than the Federal AWQCs. Federal AWQCs for known or suspected carcinogens were derived from IRIS based on a target risk level of $1x10^{-06}$; whereas Virginia WQSs for known or suspected carcinogens were derived based on a target risk level of $1x10^{-05}$, thereby being less health conservative than the corresponding AWQCs. However, the WQSs are presented in the COPC selection tables in Appendix A. Only for noncarcinogens (e.g., copper and lead) are there cases of some WQSs being more health conservative than the AWQCs. Table A-10, A-13, and A-16 show that application of WQSs, rather than AWQCs, do not affect the selection of surface water COPCS, and consequently the results of this risk assessment. Therefore, Federal AWQCs were primarily used in the selection of surface water COPCS at the Camp Allen Landfill site. Maximum detected sediment concentrations, as stated previously, were compared with NOAA ER-M values, or Region III residential soil COPC screening concentrations in the absence of NOAA values. In each of the following subsections for Areas A and B, the selection of surface water COPCs is discussed first, followed by the selection of COPCs in shallow sediments, and finally deep sediments.

2.2.5.1 Area A

Surface Water

COPC selection summaries for surface water and sediment samples collected from Area A are presented in Tables A-10 through A-12.

Table A-10 shows that ten surface water samples were collected from Area A. 1,2-Dichloroethene, acetone, tetrachloroethene, trichloroethene, vinyl chloride, and total xylenes were detected in surface water samples collected from Area A at frequencies greater than 5 percent. The maximum detected concentrations of tetrachloroethene (6 µg/L), trichloroethene (20 µg/L) and vinyl chloride (6 µg/L) exceeded AWQCs for the protection of humans consuming water and organisms. These compounds were therefore retained as COPCs in Area A surface water. Currently, no Federal AWQCs or State WQCs are established for total xylenes. Because total xylenes were detected at a frequency greater than 5 percent, total xylenes were also retained as a COPC for further evaluation in the baseline RA.

Acenaphthene, bis(2-ethylhexyl)phthalate and di-n-butylphthalate were the only SVOCs detected in Area A surface water samples. Since the maximum detected concentration of bis(2-ethylhexyl)phthalate (3 µg/L) exceeded AWQCs established for the protection of humans consuming water and organisms, bis(2-ethylhexyl)phthalate was retained as a surface water COPC in Area A.

The maximum detected concentrations of 4,4'-DDE (0.069 µg/L), alpha-chlordane (0.015 µg/L), delta-BHC (0.025 µg/L), dieldrin (0.027 µg/L), gamma-chlordane (0.024 µg/L), heptachlor epoxide (0.006 µg/L), and aroclor 1254 (0.44 µg/L), exceeded corresponding Federal AWQCs and were consequently retained as surface water COPCs in Area A. Currently, no Federal AWQCs or State WQCs are established for 4,4'-DDD. However, since 4,4'-DDD (0.26 µg/L) was detected at a frequency greater than 5 percent, this pesticide was also retained as a COPC for further evaluation in the baseline RA.

The unfiltered inorganic analytes, aluminum, arsenic, barium, lead, manganese, mercury, silver, and zinc were detected in excess of AWQCs. These metals were retained as COPCs for further evaluation in the baseline RA. Due to a lack of toxicity criteria available for aluminum and lead, these metals will be qualitatively evaluated in the baseline RA.

In summary, the following constituents have been retained as COPCs for surface waters in the Area A:

- Tetrachloroethene
- Trichloroethene

- Vinyl Chloride
- Total Xylenes
- Bis(2-ethylhexyl)phthalate
- 4,4'-DDD
- 4,4'-DDE
- Alpha-Chlordane
- Aroclor-1254
- Dieldrin
- delta-BHC
- Gamma-Chlordane
- Heptachlor Epoxide
- Aluminum
- Arsenic
- Barium
- Lead
- Manganese
- Mercury
- Silver
- Zinc

Shallow Sediments

Table A-11 shows that six shallow sediment samples were analyzed for VOCs, SVOCs, pesticides, and PCBs. Any organic constituent detected in at least one sample exceeded the prevalence criteria of 5 percent frequency of detection.

1,2-Dichloroethene, 2-butanone, carbon disulfide, chlorobenzene, and trichloroethene were detected in shallow sediment samples collected from the ditch in Area A. No NOAA sediment screening values have been established for these VOCs. However, all maximum detected concentrations of these VOCs were less than Region III residential soil COPC screening concentrations, and consequently, were not retained as COPCs.

PAHs and phthalates were the only SVOCs detected in Area A shallow sediments. All maximum detected concentrations were less than NOAA ER-M sediment screening values and/or Region III residential soil COPC screening concentrations, and consequently, were not retained as COPCs.

The pesticides 4,4'-DDD and 4,4'-DDT, and the PCB aroclor-1260 were detected at maximum concentrations (110 μg/kg, 73 μg/kg and 1,500 μg/kg, respectively) exceeding corresponding NOAA

sediment screening values. All three compounds were retained as COPCs in Area A shallow sediments.

Twenty-three shallow sediment samples were analyzed for priority pollutant metals. One sample was analyzed for the full Target Analyte List (TAL) list of metals. The following metals were detected at maximum concentrations exceeding NOAA sediment screening values and were retained as COPCs: arsenic (590 mg/kg), cadmium (160 mg/kg), chromium (3,000 mg/kg), copper (553 mg/kg), lead (1,000 mg/kg), manganese (51.2 mg/kg), mercury (3 mg/kg), silver (110 mg/kg), and vanadium (180 mg/kg).

In summary, the following constituents have been retained as COPCs for shallow sediments in Area A:

- 4,4'-DDE
- 4,4'-DDT
- Aroclor-1260
- Arsenic
- Cadmium
- Chromium
- Copper
- Lead
- Manganese
- Mercury
- Silver
- Vanadium

Deep Sediments

Table A-12 shows that one deep sediment sample collected from Area A was analyzed for VOCs, SVOCs, pesticides, and PCBs. Any organic constituent detected in that sample exceeded the prevalence criteria of 5 percent frequency of detection.

1,2-Dichloroethene, 2-butanone, acetone, carbon disulfide, chlorobenzene, and trichloroethene were detected in the deep sediment samples collected from the ditch in Area A. NOAA sediment screening values have not been established for these VOCs. However, all maximum detected concentrations of these VOCs were less than Region III residential soil COPC screening concentrations, and consequently, were not retained as COPCs.

PAHs and bis(2-ethylhexyl)phthalate were the only SVOCs detected in Area A deep sediments. All maximum detected concentrations, with the exception of acenaphthene, were less than NOAA ER-M sediment screening values and/or Region III residential soil COPC screening concentrations, and consequently, were not retained as COPCs. The maximum detected concentration for acenaphthene (4,100 µg/kg) exceeded the corresponding NOAA ER-M sediment screening value (500 µg/kg), and was therefore retained as a deep sediment COPC in the Area A ditch.

The pesticides 4,4'-DDE and dieldrin, and the PCB aroclor-1254 were detected at maximum concentrations (85 µg/kg, 62 µg/kg and 980 µg/kg, respectively) exceeding corresponding NOAA sediment screening values and/or Region III residential soil COPC screening concentrations, and consequently, were retained as COPCs in Area A deep sediments.

Five deep sediment samples were analyzed for priority pollutant metals. One sample was analyzed for the full TAL list of metals. The following metals were detected at maximum concentrations exceeding NOAA sediment screening values and/or Region III residential soil COPC screening concentrations, and consequently, were retained as COPCs: arsenic (71 mg/kg), cadmium (180 mg/kg), chromium (1,700 mg/kg), lead (540 mg/kg), manganese (50.7 mg/kg), mercury (1.1 mg/kg), silver (49 mg/kg), vanadium (74 mg/kg) and zinc (542 mg/kg).

In summary, the following constituents have been retained as COPCs for deep sediments in Area A:

- Acenaphthene
- 4,4' DDE
- Aroclor-1254
- Dieldrin
- Arsenic
- Cadmium
- Chromium
- Lead
- Manganese
- Mercury
- Silver
- Vanadium
- Zinc

2.2.5.2 Area B Pond

COPC selection summaries for surface water and sediment samples collected from the Area B pond are presented in Tables A-13 through A-15.

Surface Waters

Table A-13 shows that four surface water samples were collected from the Area B pond. Any constituent detected in at least one sample exceeds the prevalence criteria of 5 percent frequency of detection. 1,1-Dichloroethane, 1,2-dichloroethane, 1,2-dichloroethene, 2-butanone, benzene, bromodichloromethane, carbon disulfide, chloroform, methylene chloride, trichloroethene, and vinyl chloride were detected in surface water samples collected from Area B pond at frequencies greater than 5 percent. The maximum detected concentrations of 1,2-dichloroethane (8 μg/L), benzene (12 μg/L), chloroform (24 μg/L), trichloroethene (45 μg/L), and vinyl chloride (22 μg/L) exceeded AWQCs for the protection of humans consuming water and organisms. These compounds were, therefore, retained as COPCs in Area B pond surface water.

2,4-Dimethylphenol, 4-methylphenol, bis(2-ethylhexyl)phthalate, di-n-butylphthalate, and phenol were the only SVOCs detected in Area B pond surface water samples. Since the maximum detected concentration of bis(2-ethylhexyl)phthalate (9 μg/L) exceeded AWQCs established for the protection of humans consuming water and organisms, bis(2-ethylhexyl)phthalate was retained as a surface water COPC in Area B pond.

The pesticides 4,4'-DDD and gamma-BHC were detected in one surface water sample. The maximum detected concentration for gamma-BHC (0.007 μg/L) was less than the corresponding Federal AWQC (0.019 μg/L) and was consequently not retained as a surface water COPC in Area B pond. Currently, no Federal AWQCs or State WQCs are established for 4,4'-DDD. Since 4,4'-DDD (0.26 μg/L) was detected at a trace concentration less than the Federal AWQC for 4,4'-DDT (46.1 μg/L), this pesticide was also eliminated as a COPC for further evaluation in the baseline RA.

Unfiltered inorganic analytes, including arsenic, lead, manganese, and zinc, were detected in excess of AWQCs. These metals were retained as COPCs for further evaluation in the baseline RA. Due

to a lack of toxicity criteria available for lead, this metal will be qualitatively evaluated in the baseline RA.

In summary, the following constituents have been retained as COPCs for surface waters in the Area B pond:

- 1,2-Dichloroethane
- Benzene
- Chloroform
- Trichloroethene
- Vinyl Chloride
- Bis(2-ethylhexyl)phthalate
- Aluminum
- Arsenic
- Manganese
- Zinc

Shallow Sediments

Table A-14 shows that five shallow sediment samples collected from the pond were analyzed for VOCs, SVOCs, pesticides, PCBs, and inorganics. Any constituent detected in at least one sample exceeded the prevalence criteria of 5 percent frequency of detection.

1,1-Dichloroethane, 1,2-Dichloroethane, 1,2-Dichloroethene, 2-butanone, acetone, benzene, carbon disulfide, methylene chloride, trichloroethene, vinyl chloride, and total xylenes were detected in shallow sediment samples collected from the pond area. NOAA sediment screening values have not been established for these VOCs. The maximum detected concentration of vinyl chloride exceeded the corresponding Region III residential soil COPC screening concentration, and consequently, was retained as a COPC.

PAHs, dichlorobenzenes, cresols, and phthalates were the SVOCs detected in pond area shallow sediments. All maximum detected concentrations were less than NOAA ER-M sediment screening values and/or Region III residential soil COPC screening concentrations, and consequently, were not retained as COPCs.

The pesticides 4,4'-DDD, 4,4'-DDE and dieldrin, and the PCB Aroclor-1254 were detected at maximum concentrations (4,200 μg/kg, 850 μg/kg, 86 μg/kg, and 7,600 μg/kg, respectively) exceeding corresponding NOAA sediment screening values and/or Region III residential soil COPC screening concentrations. All four compounds were retained as COPCs in Area B - Pond shallow sediments.

Five sediment samples were analyzed for TAL metals. The following metals were detected at maximum concentrations exceeding NOAA sediment screening values and/or Region III residential soil COPC screening concentrations and were retained as COPCs: aluminum (26,800 gm/kg), antimony (16 mg/kg), arsenic (42.7 mg/kg), beryllium (0.76 mg/kg), cadmium (41.9 mg/kg), copper (298 mg/kg), lead (497 mg/kg), manganese (246 mg/kg), mercury (0.35 mg/kg), silver (14.7 mg/kg), vanadium (130 mg/kg), and zinc (1,020 mg/kg).

In summary, the following constituents have been retained as COPCs for shallow sediments in the Area B pond:

- Vinyl Chloride
- 4,4'-DDD
- 4,4'-DDE
- Aroclor-1254
- Dieldrin
- Aluminum
- Antimony
- Arsenic
- Beryllium
- Cadmium
- Copper
- Lead
- Manganese
- Mercury
- Silver
- Vanadium
- Zinc

Deep Sediments

Table A-15 shows that one deep sediment sample collected from Area B pond was analyzed for VOCs, SVOCs, pesticides, PCBs, and inorganics. Any constituent detected in that sample therefore exceeded the prevalence criteria of 5 percent frequency of detection.

1,2-Dichloroethene, 2-butanone, acetone, tetrachloroethene, trichloroethene, and vinyl chloride were detected in the deep sediment samples collected from the Area B pond. NOAA sediment screening values have not been established for these VOCs. None of the maximum detected concentrations of VOCs exceeded the corresponding Region III residential soil COPC screening concentrations. However, since vinyl chloride is a class A (human carcinogen) compound, this VOC was retained as a COPC.

PAHs, bis(2-ethylhexyl)phthalate and 4-methylphenol were the SVOCs detected in Area B pond deep sediments. All maximum detected concentrations were less than NOAA ER-M sediment screening values and/or Region III residential soil COPC screening concentrations, and consequently, were not retained as COPCs.

The pesticides 4,4'-DDD, 4,4'-DDE and 4,4'-DDT were detected in the deep sediment sample collected from the Area B pond. The latter two pesticides were detected at maximum concentrations (60 µg/kg and 4,400 µg/kg, respectively) exceeding corresponding NOAA sediment screening values, and consequently, were retained as COPCs.

The following metals were detected at maximum concentrations exceeding NOAA sediment screening values and/or Region III residential soil COPC screening concentrations, and consequently, were retained as COPCs: beryllium (0.56 mg/kg), cadmium (12 mg/kg), and manganese (69.6 mg/kg).

In summary, the following constituents have been retained as COPCs for deep sediments in the Area B pond:

- Vinyl Chloride
- 4,4'-DDD

- 4,4'-DDE
- Beryllium
- Cadmium
- Manganese

2.2.5.3 Area B-Elementary School

COPC selection summaries for surface water and sediment samples collected from the school area are presented in Tables A-16 through A-18.

Surface Water

Table A-16 shows that two surface water samples were collected from the school area. Any constituent detected in at least one sample exceeds the prevalence criteria of 5 percent frequency of detection. No VOCs were detected in the surface water samples collected from the school area. 2,4-Dimethylphenol and diethylphthalate were the only SVOCs detected in the school area surface water samples. No AWQCs were exceeded, therefore neither of these compounds were retained as surface water COPCs in the school area.

The pesticide 4,4'-DDD was detected in both surface water samples. Since 4,4'-DDD was detected at a frequency of 100 percent, it was retained as a COPC for further quantitative evaluation in the baseline RA.

The unfiltered inorganic analytes, arsenic, manganese, lead, and zinc, were detected in excess of AWQCs. These metals were retained as COPCs for further evaluation in the baseline RA.

In summary, the following constituents have been retained as COPCs for surface water in the school area:

- 4,4'-DDD
- Arsenic
- Lead
- Manganese
- Zinc

Shallow Sediments

Table A-17 shows that no constituents were detected at maximum concentrations exceeding NOAA sediment screening values and/or Region III residential soil COPC screening concentrations. Therefore, no constituents were retained as COPCs in the shallow sediments sampled in the school area.

Deep Sediments

Table A-18 shows that lead and mercury were detected at maximum concentrations (310 mg/kg and 0.8 mg/kg, respectively) in a deep sediment sample collected from the school area that exceed NOAA sediment screening values. Consequently, lead and mercury were retained as COPCs. Due to a lack of toxicity criteria available for lead, this metal will be qualitatively evaluated in the baseline RA.

2.2.6 Air

Air sampling was performed at the Camp Allen Landfill to evaluate the potential presence of volatile COPCs in this medium. The air sampling program focused on air inside the Brig Facility, and the Camp Allen Elementary School, as well as outdoor air. Air samples were collected on three consecutive days in January 1993 at each sampling location. A total of twelve (12), five (5) and five (5) sampling locations were selected for the Brig Facility, the elementary school and outdoor air, respectively. Outdoor air samples were collected at locations upwind and downwind of the landfill. The two upwind locations were considered to be representative of site-specific background to which the remaining 20 air samples were compared. Upwind air samples displayed chemical profiles similar to those observed in the Brig Facility, elementary school, and downwind outdoor air samples. This suggests that the presence of some detected chemicals in air samples obtained from the Camp Allen Landfill Site is a result of ambient conditions. As stated previously, the selection of air COPCs for the Camp Allen Landfill consisted of comparing maximum detected air concentrations measured at the site with Region III COPC screening concentrations for ambient air, site-specific background air data. USEPA's National Ambient Volatile Organic Compound Database information was also considered. Volatile organic air concentrations specific to the Commonwealth of Virginia were obtained from the USEPA data base and used in the selection of COPCs. COPCs were

identified for Brig Facility indoor air, elementary school indoor air, outdoor ambient air around the site, and air outside of the brig facility and near the gas well. The COPC selection summaries are presented in Tables A-19 through A-22.

2.2.6.1 Brig Facility

Table A-19 shows that a total of thirty-three air samples were collected at indoor locations within the Brig Facility. Maximum detected concentrations of the following compounds exceeded corresponding Region III COPC screening concentrations, and were retained as COPCs in the baseline RA:

- 1,1,1-Trichloroethane
- 1,2,4-Trimethylbenzene
- 1,3,5-Trimethylbenzene
- 1,4-Dichlorobenzene
- Benzene
- Benzyl Chloride
- Methylene Chloride
- Bromomethane
- Chloroform
- Chloromethane
- Hexachlorobutadiene
- Meta-/Para-Xylenes
- Toluene

However, it should be noted that some of these VOCs could be present in Brig air samples for reasons other than past disposal practices at the Area A landfill. Methylene chloride and toluene are solvents commonly used by all environmental laboratories. Although methylene chloride and toluene were not detected in trip blanks, the potential exists for laboratory induced results, particularly at the levels detected. Dichlorobenzenes are commonly used as disinfectants and are present in many cleaning solutions. 1,4-Dichlorobenzene is also a primary constituent of mothballs (Merck Index, 11th Ed., 1989). Hexachlorobutadiene was detected in only one of 33 samples taken throughout the Brig (3 percent frequency of detection). Hexachlorobutadiene is an intermediate used in the production of rubber compounds. Of all of the detected VOCs, 1,1,1-trichloroethane was detected at the highest concentrations throughout the Brig (sample AA- 06A, 3,400 $\mu g/m^3$, maximum detected value). 1,1,1-Trichloroethane is widely used as a propellant in many products, and is also used for cleaning equipment and gear in the Brig (LANTDIV personnel, 1993). Its

presence in Brig air may likely be due to Brig usage and not past practices at the Area A landfill. 1,3,5-Trimethylbenzene occurs in coal tars, which are often used as disinfectants in certain cleaning agents (Merck Index, 11th Ed., 1989). Trimethylbenzenes also occur in many petroleum products. 1,3,5-Trimethylbenzene was not detected in other environmental media from Area A suggesting that the presence of the chemical in Brig air samples may not be site-related.

Freons 113 and 114 were each detected in only one sample, at a frequency of less than 5 percent. Freons are ubiquitous in the environment since they are used as coolants in air conditioning and refrigeration units. Although no toxicity or Region III COPC screening criteria are known to exist for freons, they were not retained for evaluation in the baseline RA due to the low frequency of detection in indoor air samples collected at the Brig Facility.

2.2.6.2 Elementary School

Table A-20 shows that a total of fifteen air samples were collected at indoor locations within the elementary school. Maximum detected concentrations of the following compounds exceeded corresponding Region III COPC screening concentrations, and were retained as COPCs in the baseline RA:

- 1,2,4-Trimethylbenzene
- 1,4-Dichlorobenzene
- Benzene
- Hexachlorobutadiene

The presence of some of these chemicals in school indoor air could be related to sources other than past disposal practices at the Area B Landfill. Detected concentrations of 1,2,4-trimethylbenzene, 1,4-dichlorobenzene and hexachlorobutadiene may be attributable to the possible non-site-related sources that were discussed for the Brig Facility.

2.2.6.3 Outdoor Air

Ambient Air

Table A-21 shows that a total of nine air samples were collected at outdoor locations around the Camp Allen site. Maximum detected concentrations of the following compounds exceeded corresponding Region III COPC screening concentrations, and were retained as COPCs in the baseline RA:

- 1,2,4-Trimethylbenzene
- Benzene
- Bromomethane
- Hexachlorobutadiene

Of the VOCs listed above, the maximum detected concentrations of bromomethane and hexachlorobutadiene also exceeded corresponding background ambient air concentrations.

Gas Well Location

Table A-22 shows that a total of three air samples were collected at the gas well location outside of the Brig Facility. Maximum detected concentrations of the following compounds exceeded corresponding Region III COPC screening concentrations, and were retained as COPCs in the baseline RA:

- 1,2,4-Trimethylbenzene
- Benzene
- Toluene

Of the VOCs listed above, the maximum detected concentrations of 1,2,4-trimethylbenzene and toluene also exceeded corresponding background ambient air concentrations.

Freon 114 was detected in two out of three samples collected just outside of the Brig Facility near the gas well, at a frequency exceeding 5 percent. Freons are ubiquitous in the environment since they are used as coolants in air conditioning units. Trace levels of freon may be detected in indoor

and/or outdoor air at any building that is equipped with air conditioning and/or refrigeration units. Therefore, freon 114 was not retained for evaluation in the baseline RA.

2.3 Summary of COPCs

The following presents a comprehensive list of all COPCs identified over all areas of concern and investigated environmental media at the Camp Allen Landfill:

Volatile Organic Compounds

- Benzene
- Bromomethane
- 2-Butanone
- Chlorobenzene
- Chloroform
- Chloromethane
- 1,4-Dichlorobenzene
- 1,2-Dichloroethane
- 1,1-Dichloroethene
- 1,2-Dichloroethene
- 4-Methyl-2-pentanone
- Methylene Chloride
- Tetrachloroethene
- Toluene
- 1,1,1-Trichloroethane
- Trichloroethene
- 1,2,4-Trimethylbenzene
- 1,3,5-Trimethylbenzene
- Benzyl Chloride
- Vinyl Chloride
- Total Xylenes

Semivolatile Organic Compounds

- Hexachlorobutadiene
- 2-Methylphenol
- 2,4-dimethylphenol
- 4-Methylphenol
- Bis(2-ethylhexyl)phthalate
- Bis(2-chloroethyl)ether
- Acenaphthene

Pesticides

- Aldrin
- Alpha-Chlordane
- delta-BHC
- gamma-BHC (Lindane)
- 4,4'-DDD
- 4,4'-DDE
- 4,4'-DDT
- Dieldrin
- gamma-Chlordane
- Heptachlor Epoxide

Polychorinated Biphenyls

- Aroclor-1254
- Aroclor-1260

Metals

- Aluminum
- Antimony
- Arsenic
- Barium
- Beryllium
- Cadmium
- Chromium
- Copper
- Lead
- Manganese
- Mercury
- Nickel
- Silver
- Thallium
- Vanadium
- Zinc

SECTION 2.0 TABLES

TABLE 2-1

NATIVE CONCENTRATION RANGES FOR INORGANICS IN SOILS FROM LITERATURE
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Constituent	Lindsay, 1979[1]	Tox. Profiles[2]	Tox. of Metals, 1986[3]	Dragun, 1988[4]	Modern Env. Tox. XI, 1987[5]	U.S. Geological Survey (Eastern Portion of U.S.)[6]
Aluminum	NA	NA	NA	10,000 - 300,000	NA	7,000 - 100,000
Antimony	--	--	NA	0.6 - 10	--	<1 - 8.8
Arsenic	1.0 - 50	0.1 - 80	40	1 - 40	Trace - 40	<0.1 - 73
Barium	--	--	NA	100 - 3,500	--	10 - 1,500
Beryllium	NA	0.01 - 40	NA	0.1 - 40	NA	<1.0 - 7.0
Cadmium	0.01 - 0.7	0.6 - 6.0	<1.0	0.01 - 7.0	<1.0 - 30	NA
Chromium	1 - 100	NA	Trace - 250	5.0 - 3,000	Trace - 250	1.0 - 1,000
Copper	--	--	2 - 100	2.0 - 100	--	<1 - 700
Lead	2 - 200	10 - 30	2 - 200	2.0 - 200	10 - 700	<10 - 300
Manganese	--	--	1 - 7,000	100 - 4,000	--	<2 - 7,000
Mercury	0.01 - 0.3	NA	NA	0.01 - 0.08	0.01 - 0.3	<0.01 - 3.4
Nickel	NA	5.0 - 1,000	NA	5.0 - 1,000	NA	<5.0 - 700
Selenium	0.1 - 2.0	4.0 - 8.0	NA	0.1 - 2.0	0.1 - 100	<0.1 - 3.9
Silver	10 - 5,000	NA	NA	0.1 - 5.0	NA	NA
Thallium	--	--	1	NA	--	NA
Vanadium	--	--	5 - 140	20 - 500	--	<7 - 300
Zinc	NA	10 - 3,000	NA	<10 - 2,000	NA	<5.0 - 2,900

Notes: NA - Not available in reference
-- Reference not available
All values reported in mg/kg.

(1) Lindsay, W. L. 1979. Chemical Equilibria in Soils. John Wiley and Sons, New York.

TABLE 2-1 (Continued)

NATIVE CONCENTRATION RANGES FOR INORGANICS IN SOILS FROM LITERATURE
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Notes (Continued):

(2) U.S. Environmental Protection Agency.
Draft Toxicological Profile for Arsenic, February 1992.
Draft Toxicological Profile for Beryllium, February 1992.
Draft Toxicological Profile for Cadmium, February 1992.
Draft Toxicological Profile for Lead, February 1992.
Draft Toxicological Profile for Nickel, February 1992.
Draft Toxicological Profile for Selenium, October 1987.
Draft Toxicological Profile for Zinc, December 1989.
Prepared for the Agency for Toxic Substances and Disease Registry (ATSDR).

(3) Friberg, L., Nordberg, G. F. and Vouk, V. B., editors. 1986. Handbook on the Toxicology of Metals, Volume II: Specific Metals. Elsevier Science Publishers, Amsterdam.

(4) Dragun, J. 1988. The Soil Chemistry of Hazardous Materials. The Hazardous Materials Control Research Institute, Silver Spring, Maryland.

(5) Mehlman, M. A. 1987. Series: Advances in Modern Environmental Toxicology, Volume XI, Genotoxic and Carcinogenic Metals: Environmental and Occupational Occurrence and Exposure. Princeton Scientific Publishing, Princeton, New Jersey.

(6) Schacklette, H. T. and Boerngen, J. G. 1984. "Element Concentrations in Soils and Other Surficial Materials of the Conterminous United States." U.S. Geological Survey Professional Paper 1270, U.S. Department of the Interior.

TABLE 2-2

HUMAN HEALTH BASED WATER CRITERIA AND ADVISORIES
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Constituent	Drinking Water Health Advisories (mg/L)					Federal Standards (mg/L)		State of Virginia Standards (mg/L)	
	One Day Child	Ten Day Child	Longer-Term Child	Longer-Term Adult	Lifetime Adult	MCL	MCLG	MCL	Groundwater Standards
VOCs									
Benzene	0.2	0.2	--	--	--	0.005	0.0	0.005	--
Benzyl Chloride	--	--	--	--	--	--	--	--	--
Bromomethane	0.1	0.1	0.1	0.5	0.01	--	--	--	--
2-Butanone	--	--	--	--	--	--	--	--	--
Chlorobenzene	--	--	--	--	--	--	--	--	--
Chloroform	4	4	0.1	0.4	--	0.1*/0.08+	0.0	--	--
Chloromethane	9	0.4	0.4	1	0.003	--	--	--	--
(para) 1,4-Dichlorobenzene	10	10	10	40	0.075	0.075	0.075	0.075	--
(ortho) 1,2-Dichlorobenzene	9	9	9	30	0.6	0.6	0.6		--
1,1-Dichloroethane	--	--	--	----	--	--	--	--	
1,2-Dichloroethane	0.7	0.7	0.7	2.6	--	0.005	0.0	0.005	--
1,1-Dichloroethene	2	1	1	4	0.007	0.007	0.007	0.007	--
trans-1,2-Dichloroethene	2	2	2	6	0.1	0.1	0.1		--
Ethylbenzene	30	3	1	3	0.7	0.7	0.7	--	--
4-Methyl-2-pentanone (methyl isobutyl ketone)	--	--	1	--	--	--	--	--	--
Methylene chloride	--	--	--	--	--	--	--	--	--
Tetrachloroethene	2	2	1	5	--	0.005	0.0	--	--
Toluene	20	2	2	7	1	1	1	--	--
1,1,1-Trichloroethane	100	40	40	100	0.2	0.2	0.2	0.2	--
Trichloroethene	--	--	--	--	--	0.005	0.0	0.005	--
1,2,4-Trimethylbenzene	--	--	--	--	--	--	--	--	--
1,3,5-Trimethylbenzene	--	--	--	--	--	--	--	--	--
Vinyl chloride	3	3	0.01	0.05	--	0.002	0.0	0.002	--

TABLE 2-2 (Continued)

HUMAN HEALTH BASED WATER CRITERIA AND ADVISORIES
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Constituent	Drinking Water Health Advisories (mg/L)					Federal Standards (mg/L)		State of Virginia Standards (mg/L)	
	One Day Child	Ten Day Child	Longer-Term Child	Longer-Term Adult	Lifetime Adult	MCL	MCLG	MCL	Groundwater Standards
m-Xylene	--	--	--	--	--	--	--	--	--
o-Xylene	--	--	--	--	--	--	--	--	--
p-Xylene	--	--	--	--	--	--	--	--	--
Xylenes (total)	40	40	40	100	10	10	10	--	--
SVOCs: Acenaphthene	--	--	--	--	--	--	--	--	--
bis (2-chloroethyl)ether	--	--	--	--	--	--	--	--	--
bis(2-ethylhexyl)phthalate	--	--	--	--	--	--	--	--	--
2,4-Dimethylphenol	--	--	--	--	--	--	--	--	--
Hexachlorobutadiene	0.3	0.3	0.1	0.4	0.001	--	0.001	--	--
2-Methylphenol (o-cresol)	--	--	--	--	--	--	--	--	--
4-Methylphenol (p-cresol)	--	--	--	--	--	--	--	--	--
Pesticides Aldrin	0.0003	0.0003	0.0003	0.0003	--	--	--	--	0.000003
beta-BHC	--	--	--	--	--	--	--	--	--
gamma-BHC (Lindane)	1	1	0.03	0.1	0.0002	0.0002	0.0002	0.004	0.00001
alpha-Chlordane (2)	0.06	0.06	--	--	--	0.002	0	--	0.00001
4,4'-DDD	--	--	--	--	--	--	--	--	--
4,4'-DDE	--	--	--	--	--	--	--	--	--
4,4'-DDT	--	--	--	--	--	--	--	--	0.000001
Dieldrin	0.0005	0.0005	0.0005	0.002	--	--	--	--	0.000003
Endrin	0.02	0.02	0.003	0.01	0.002	0.002	0.002	0.002	0.000004
Heptachlor epoxide	0.01	--	0.0001	0.0001	--	0.0002	0	--	0.000001
PCBs Aroclor-1254	--	--	--	--	--	0.0005	0	--	--
Aroclor-1260	--	--	--	--	--	0.0005	0	--	--

TABLE 2-2 (Continued)

HUMAN HEALTH BASED WATER CRITERIA AND ADVISORIES
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Constituent	Drinking Water Health Advisories (mg/L)					Federal Standards (mg/L)		State of Virginia Standards (mg/L)	
	One Day Child	Ten Day Child	Longer-Term Child	Longer-Term Adult	Lifetime Adult	MCL	MCLG	MCL	Groundwater Standards
Inorganics									
Aluminum	--	--	--	--	--	--	--	--	--
Antimony	0.01	0.01	0.01	0.015	0.003	0.006	0.006	--	--
Arsenic	--	--	--	--	--	0.05	--	0.05	0.05
Barium	--	--	--	--	2	2	2	1.0	1.0
Beryllium	30	30	4	20	--	0.004	0.004	--	--
Cadmium	0.04	0.04	0.005	0.02	0.005	0.005	0.005	0.010	0.0004
Chromium	1	1	0.2	0.8	0.1	0.1	0.1	0.05	0.05
Copper	--	--	--	--	--	1.3(1)	1.3	1.0	1.0
Iron	--	--	--	--	--	--	--	0.3	--
Lead	--	--	--			0.015(1)	0	0.05	0.05
Manganese	--	--	--	--	--	--	--	0.05	--
Mercury (inorganic)	--	--	--	0.002	0.002	0.002	0.002	0.002	0.00005
Nickel	1	1	0.5	1.7	0.1	0.1	0.1	--	--
Selenium	--	--	--	--	--	0.05	0.05	0.01	0.01
Silver	0.2	0.2	0.2	0.2	0.1	--	--	0.05	--
Thallium	0.007	0.007	0.007	0.02	0.0004	0.002	0.0005	--	--
Vanadium	--	--	--	--	--	--	--	--	--
Zinc	6	6	3	12	2	--	--	5.0	0.05

Notes: (1) Action level.
* Current MCL
\+ Total for all THMs combined cannot exceed this level

TABLE 2-3

STATE OF VIRGINIA AND FEDERAL AMBIENT WATER QUALITY CRITERIA FOR THE PROTECTION OF HUMAN HEALTH FROM THE CONSUMPTION OF WATER AND OF WATER AND ORGANISMS
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	State of Virginia Human Health Criteria		Federal Human Health Criteria	
	Public Water Supply[1] (μg/L)	All Other Surface Waters[2] (μg/L)	Water and Organisms (μg/L)	Organisms Only (μg/L)
VOCs				
Benzene	12	710	1.2	71
Benzyl Chloride	--	--	--	--
Bromomethane	--	--	48	4,000
Chlorobenzene	--	--	680	21,000
Chloroform	57	4,700	5.7	470
Chloromethane	57	4,700	5.7	470
1,2-dichlorobenzene	2,700	1,700	2,700	17,000
1,4-dichlorobenzene	400	2,600	400	2,600
1,1-dichloroethane	--	--	--	--
1,2-dichloroethane	3.8	990	0.38	99
1,1-dichloroethene	--	--	0.57	3.2
trans-1,2-dichloroethene	--	--	700	140,000
Ethylbenzene	3,000	29,000	3,100	29,000
4-methyl-2-pentanone (methyl isobutyl ketone)	--	--	--	--
Methylene chloride	--	--	4.7	1,600
Tetrachloroethene	--	--	--	--
Toluene	6,800	200,000	10,000	300,000
1,1,1-trichloroethane	--	--	3,100	170,000
Trichloroethene	27	807	2.7	81
1,2,4-trimethylbenzene	--	--	--	--
1,3,5-trimethylbenzene	--	--	--	--
Vinyl chloride	20	5,250	--	--
m-xylene	--	--	--	--
o-xylene	--	--	--	--

TABLE 2-3 (Continued)

STATE OF VIRGINIA AND FEDERAL AMBIENT WATER QUALITY CRITERIA FOR THE PROTECTION OF HUMAN HEALTH FROM THE CONSUMPTION OF WATER AND OF WATER AND ORGANISMS
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	State of Virginia Human Health Criteria		Federal Human Health Criteria	
	Public Water Supply[1] (μg/L)	All Other Surface Waters[2] (μg/L)	Water and Organisms (μg/L)	Organisms Only (μg/L)
p-xylene	--	--	--	--
Xylenes (Total)	--	--	--	--
SVOCs: Acenaphthene	--	--	1,200	2,700
bis (2-chloroethyl)ether	--	--	--	--
bis(2-ethylhexyl)phthalate	--	--	--	--
2,4-dimethylphenol	--	--	--	--
Hexachlorobutadiene	--	--	0.44	50
2-methylphenol(o-cresol)	--	--	--	--
4-methylphenol(p-cresol)	--	--	--	--
Pesticides Aldrin	0.0013	0.0014	0.00013	0.00014
beta-BHC	--	--	0.014	0.046
gamma-BHC (Lindane)	7	25	0.019	0.063
alpha-Chlordane[3]	0.0058	0.0059	0.00058	0.00059
4,4'-DDD	--	--	0.00083	0.00083
4,4'-DDE	--	--	0.00059	0.00059
4,4'-DDT	0.0059	0.0059	0.00059	0.00059
Dieldrin	0.0014	0.0014	0.00014	0.00014
Endrin	0.76	0.91	0.76	0.81
Heptachlor epoxide	--	--	0.00010	0.00011
PCBs Aroclor-1254	0.00044	0.00045	0.000044	0.000045
Aroclor-1260	0.00044	0.00045	0.000044	0.000045
Inorganics Aluminum	--	--	(4)	(4)
Antimony	--	--	14	4,300

TABLE 2-3 (Continued)

STATE OF VIRGINIA AND FEDERAL AMBIENT WATER QUALITY CRITERIA FOR THE PROTECTION OF HUMAN HEALTH FROM THE CONSUMPTION OF WATER AND OF WATER AND ORGANISMS
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	State of Virginia Human Health Criteria		Federal Human Health Criteria	
	Public Water Supply[1] (μg/L)	All Other Surface Waters[2] (μg/L)	Water and Organisms (μg/L)	Organisms Only (μg/L)
Arsenic	50	--	0.018	0.14
Barium	2,000	--	--	--
Beryllium	--	--	0.0076	0.131
Cadmium	16	170	10	170
Chromium	170	3,400	170	3,400
Copper	1,300	--	1,300	--
Iron	300[5]	--	--	--
Lead	15	--	--	--
Manganese	50[5]	--	--	--
Mercury	0.144	0.146	0.14	0.15
Nickel	607	4,583	510	3,800
Selenium	172	11,200	104	6,800
Silver	--	--	91	--
Thallium	--	--	1.7	6.0
Vanadium	--	--	--	--
Zinc	5,000[5]	--	--	--

Notes:
(1) Calculated for drinking water and fish consumption
(2) Calculated for fish consumption
(3) Toxicity criteria for chlordane used to evaluate alpha- and gamma-chlordane
(4) Values are pH dependent
(5) To maintain acceptable taste, odor or aesthetic quality of drinking water
(6) Chronic aquatic life values have been calculated to protect wildlife from harmful effects through ingestion of contaminated tissues. However, the standard will also protect aquatic life from toxic effects.
(7) Chronic aquatic life standard applies to methyl mercury.

TABLE 2-4

STATE OF VIRGINIA AND FEDERAL AMBIENT WATER QUALITY CRITERIA FOR THE PROTECTION OF AQUATIC LIFE.
CHEMICALS DETECTED IN SURFACE WATERS
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	State of Virginia Surface Water Standards				Federal Ambient Water Quality Criteria			
	Aquatic Life (µg/L)				Aquatic Life (µg/L)			
	Freshwater		Salt Water		Freshwater		Salt Water	
	Acute	Chronic	Acute	Chronic	Acute	Chronic	Acute	Chronic
VOCs								
Benzene	--	--	--	--	5,300**	--	5100*	700*
Benzyl Chloride	--	--	--	--	--	--	--	--
Bromomethane	--	--	--	--	--	--	--	--
Chlorobenzene	--	--	--	--	--	--	--	--
Chloroform	--	--	--	--	28,900**	1,240**	--	--
Chloromethane	--	--	--	--	--	--	--	--
1,2-dichlorobenzene	--	--	--	--	--	--	--	--
1,4-dichlorobenzene	--	--	--	--	--	--	--	--
1,1-dichloroethane	--	--	--	--	--	--	--	--
1,2-dichloroethane	--	--	--	--	118,000**	20,000**	113,000*	--
1,1-dichloroethene	--	--	--	--	--	--	--	--
trans-1,2-dichloroethene	--	--	--	--	--	--	--	--
Ethylbenzene	--	--	--	--	32,000*	--	430*	--
4-methyl-2-pentanone (methyl isobutyl ketone)	--	--	--	--	--	--	--	--
Methylene chloride	--	--	--	--	--	--	--	--
Tetrachloroethene	--	--	--	--	5,280*	840*	10,200*	450*
Toluene	--	--	--	--	17,500*	--	6,300*	5,000*
1,1,1-trichloroethane	--	--	--	--	--	--	31,200*	--
Trichloroethene	--	--	--	--	45,000**	21,900**	2,000*	--
1,2,4-trimethylbenzene	--	--	--	--	--	--	--	--
1,3,5-trimethylbenzene	--	--	--	--	--	--	--	--
Vinyl chloride	--	--	--	--	--	--	--	--
m-xylene	--	--	--	--	--	--	--	--
o-xylene	--	--	--	--	--	--	--	--

TABLE 2-4 (Continued)

STATE OF VIRGINIA AND FEDERAL AMBIENT WATER QUALITY CRITERIA FOR THE PROTECTION OF AQUATIC LIFE.
CHEMICALS DETECTED IN SURFACE WATERS
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	State of Virginia Surface Water Standards				Federal Ambient Water Quality Criteria			
	Aquatic Life (µg/L)				Aquatic Life (µg/L)			
	Freshwater		Salt Water		Freshwater		Salt Water	
	Acute	Chronic	Acute	Chronic	Acute	Chronic	Acute	Chronic
p-xylene	--	--	--	--	--	--	--	--
Xylenes (Total)	--	--	--	--	--	--	--	--
SVOCs:								
Acenaphthene	--	--	--	--	1,700*	520*	970*	710*
bis (2-chloroethyl)ether	--	--	--	--	--	--	--	--
bis(2-ethylhexyl)phthalate	--	--	--	--	--	--	--	--
2,4-dimethylphenol	--	--	--	--	2,120*	--	--	--
Hexachlorobutadiene	--	--	--	--	90*	9.3*	32*	--
2-methylphenol(o-cresol)	--	--	--	--	--	--	--	--
4-methylphenol(p-cresol)	--	--	--	--	--	--	--	--
Pesticides								
Aldrin	3.0	0.3	1.3	0.13	3.0	--	1.3	--
beta-BHC	--	--	--	--	--	--	--	--
gamma-BHC (Lindane)	2.0	0.080	0.16	0.01	2.0	0.8	0.16	--
alpha-Chlordane (2)	2.4	0.0043	0.09	0.0040	2.4	0.0043	0.09	0.004
4,4'-DDD	--	--	--	--	0.6*	--	3.6*	--
4,4'-DDE	--	--	--	--	1050*	--	14*	--
4,4'-DDT	1.1	0.001	0.13	0.0010	1.1	0.001	0.13	0.001
Dieldrin	2.5	0.0019	0.71	0.0019	2.5	0.0019	0.71	0.0019
Endrin	0.18	0.0023	0.037	0.0023	0.18	0.0023	0.037	0.0023
Heptachlor epoxide	--	--	--	--	0.52	0.0038	0.053	0.0036
PCBs								
Aroclor-1254	--	0.014	--	0.03	0.014	--	--	--
Aroclor-1260	--	0.014	--	0.03	--	--	--	--

TABLE 2-4 (Continued)

STATE OF VIRGINIA AND FEDERAL AMBIENT WATER QUALITY CRITERIA FOR THE PROTECTION OF AQUATIC LIFE.
CHEMICALS DETECTED IN SURFACE WATERS
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	State of Virginia Surface Water Standards				Federal Ambient Water Quality Criteria			
	Aquatic Life (µg/L)				Aquatic Life (µg/L)			
	Freshwater		Salt Water		Freshwater		Salt Water	
	Acute	Chronic	Acute	Chronic	Acute	Chronic	Acute	Chronic
Inorganics Aluminum	--	--	--	--	(1)	(1)	(1)	(1)
Antimony	--	--	--	--	/p/88	/p/30	/p/1,500	/p/500
Arsenic	--	--	--	--	--	--	--	--
Barium	--	--	--	--	--	--	--	--
Beryllium	--	--	--	--	130*	5.3*	--	--
Cadmium	**	**	43	9.3	3.9**	1.1**	43	9.3
Chromium	16	11	1,100	50	16	11	1,100	50
Copper	**	**	2.9	2.9	18**	12**	2.9	2.9
Iron	--	--	--	--	--	1,000	--	--
Lead	**	**	220	8.5	82**	3.2**	220	8.5
Manganese	--	--	--	--	--	--	--	--
Mercury	2.4	0.012	2.1	0.025	2.4	0.012	2.1	0.025
Nickel	**	**	75	8.3	1,400**	160**	75	8.3
Selenium	20	5.0	300	71	20	5.0	300	71
Silver	**	--	2.3	--	4.1** (/p/0.92)	--	2.3** (/p/7.2)	/p/0.92
Thallium	--	--	--	--	1,400*	40*	2,130*	--
Vanadium	--	--	--	--	--	--	--	--
Zinc	**	**	95	86	120**	110**	95	86

Notes: (1) Criteria are pH dependent
(2) Toxicity criteria used to evaluate alpha- chlordane is for gamma-chlordane
/p/ = proposed criterion
* No criteria available. Lowest observed effect level (LOEL) is presented.
** Hardness dependent. Federal values use a hardness of 100 mg/L as $CaCO_3$.
All values are in $\mu g/L$.
-- = No numerical value is currently available.

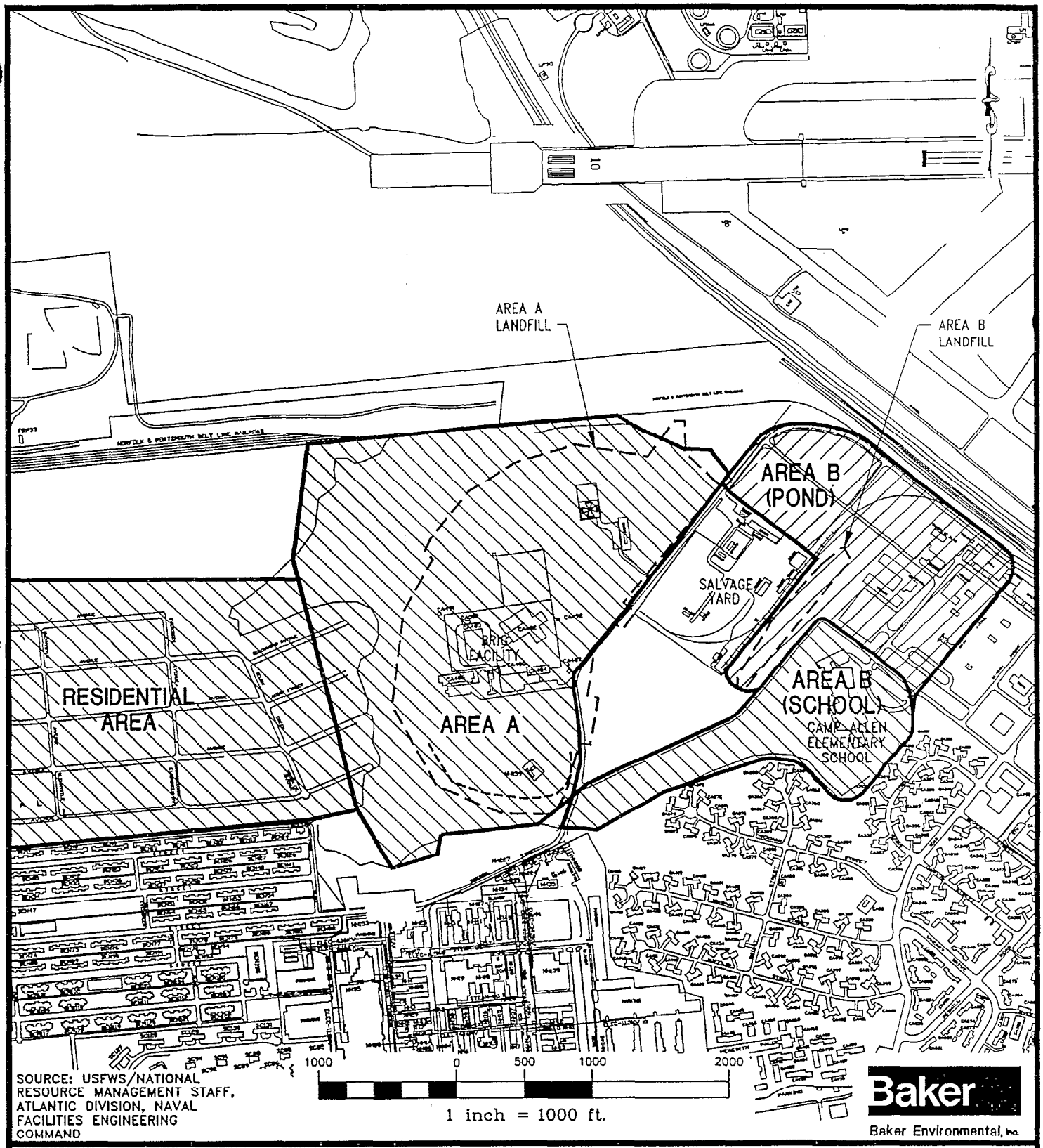
TABLE 2-5

SEDIMENT SCREENING VALUES (SSVs) FOR
SELECT CHEMICALS DETECTED IN SEDIMENTS
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	SSV[1] (mg/kg)	
	ER-L	ER-M
Trace Elements:		
Arsenic	8	70
Cadmium	1.2	9.6
Chromium	81	370
Copper	34	270
Lead	46	218
Mercury	0.15	0.71
Nickel	21	223
Silver	1.0	3.7
Zinc	150	410
Polychlorinated Biphenyls (PCBs):		
Total PCBs	0.022	0.180
DDT and Metabolites:		
DDT	0.001	0.0461
DDE	0.002	0.027
Polynuclear Aromatic Hydrocarbons (PAHs):		
Acenaphthene	0.016	0.500
Anthracene	0.085	1.100
Benzo(a)anthracene	0.261	1.600
Benzo(a)pyrene	0.430	1.600
Chrysene	0.384	2.800
Dibenz(a,h)anthracene	0.063	0.260
Fluoranthene	0.600	5.100
Fluorene	0.019	0.540
2-Methylnaphthalene	0.070	0.670
Naphthalene	0.160	2.100
Phenanthrene	0.240	1.500
Pyrene	0.665	2.600
Total PAH	4.020	44.790

Note: [1] As per USEPA comments, preference was given to MacDonald (1992), SSV over Long and Morgan (1991). However, in the absence of a MacDonald value, Long and Morgan value was used.
ER-L - Effective Range-Low
ER-M - Effective Range-Median
If sediment concentrations are above the ER-M, adverse health effects on biota are considered probable. If sediment concentrations are between ER-L and ER-M, adverse health effects on biota are possible. If contaminant concentrations are below the ER-L, adverse health effects are considered unlikely.

SECTION 2.0 FIGURES



SOURCE: USFWS/NATIONAL RESOURCE MANAGEMENT STAFF, ATLANTIC DIVISION, NAVAL FACILITIES ENGINEERING COMMAND

FIGURE 2-1
IDENTIFICATION OF STUDY AREAS
BASELINE RISK ASSESSMENT
CAMP ALLEN LANDFILL

NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

084-506

SECTION 3

3.0 EXPOSURE ASSESSMENT

The exposure assessment addresses each current and future potential exposure pathway in groundwater, surface soil, surface water, sediment, and air. To determine whether human exposure could occur at the Camp Allen Landfill site in the absence of remedial action, an exposure assessment which identifies potential exposure pathways and receptors was conducted. The following four elements were considered to determine whether a complete exposure pathway was present: a source and mechanism of chemical release; an environmental retention or transport medium; a point of potential human contact with the contaminated medium; and, an exposure route (e.g., ingestion) at the contact point.

The exposure scenarios discussed herein represent USEPA's Reasonable Maximum Exposure (RME). Relevant equations for assessing intakes and exposure factors were obtained from the Risk Assessment Guidance for Superfund. Volume I, Human Health Evaluation (RAGS) (USEPA, 1989), Exposure Factors Handbook (USEPA, 1989a), Dermal Exposure Assessment: Principles and Applications. Interim Report (USEPA, 1992), Superfund Exposure Assessment Manual (USEPA, 1988), and Standard Default Exposure Factors. Interim Final (USEPA, 1991) .

3.1 Characterization of Exposure Setting

The Camp Allen Landfill, Naval Base Norfolk is located in Norfolk, Virginia and includes an area of approximately 45 acres that is divided into two distinct study areas. Area A is the largest study area at the landfill and is approximately 43 acres. Area B comprises the remaining two acres.

The landfill is surrounded by a mixture of residential, military, and commercial settings. The Glenwood Park community is located to the west of the Camp Allen Landfill. The Armed Forces Staff College is located approximately one third mile to the south of the landfill. Residences border the eastern and southern portions of the study area.

The climate of the Norfolk area is moderate continental with mild winters and long warm summers. Average monthly temperatures range from about 40°F in January to 79°F in July. Average annual net precipitation is approximately 45 inches. Precipitation is well distributed throughout the year and the heaviest rain events tend to occur in July and August. The prevailing wind direction is from

the southwest but northeasterly winds are common. The average wind speed is approximately 11 miles per hour.

3.2 Identification of Human Receptors

Potential human receptors at the Camp Allen Landfill Site are identified below for Area A and Area B of the study area (see Section 2.0 for a detailed discussion of the areas of concern evaluated in this RA).

3.2.1 Area A

Current potential human receptors to COPCs detected in environmental media include Brig employees (military and civilian), Brig prisoners (including awardees), local children, and local adults. Area A is dominated by the presence of the Brig, which greatly restricts access to affected on-site media by local children and adults for obvious reasons. Access is, however, available to Brig employees and prisoners who would tend to be exposed with greater frequency and duration. For the sake of conservatism, the Brig employee was considered to be a civilian, having a greater exposure duration than a military employee.

Although future residential development of Area A is highly unlikely, it was considered in the event of a base closing. Future residential adults and children living on site were therefore evaluated. Future construction workers performing excavation and building activities were also evaluated as a potential receptor group.

3.2.2 Area B

Current human receptors to COPCs detected in environmental media include local children and adults particularly those working at the Camp Allen Elementary School or accessing (trespassing) the Pond area. Potential exposure in Area B Pond is, in general, restricted by the limited access, whereas potential exposure in Area B School is not. Children attending kindergarten through fifth grade could potentially be exposed to soils before school, during recess, and after school. Ditches around Area B School are fenced on the school side to prevent children from accessing the ditches. The ditches, however, are not fenced on the opposing side facing Camp Elmore base housing.

Children attending the Elementary School could, therefore, access the ditches after school, on their way home. Adults involved in maintenance of the ditches and school property could potentially be exposed to COPCs detected in Area B School soils, surface waters and sediments.

Future residential development of Area B is also unlikely; however, for the sake of conservatism, residential adult and child exposure was evaluated. Future construction workers performing excavation and building activities were also evaluated as a potential receptor.

3.3 Identification of Potential Exposure Pathways

3.3.1 Chemical Fate and Transport

This section discusses the potential release and migration of COPCs between or within media. The potential for a chemical to migrate spatially and persist in environmental media is important in the estimation of exposure.

The distribution relationships for a chemical between the environmental compartments of air, water and soil can be evaluated using a series of equilibrium constants. By utilizing the physiochemical properties of a constituent, it is possible to estimate a chemical's expected environmental distribution and its ultimate environmental fate.

The environmental mobility and persistence of a chemical will be influenced primarily by its physical and chemical properties and the chemistry of the medium in which it occurs. Table 3-1 presents the physical and chemical properties associated with the organic COPCs including specific gravity, vapor pressure, water solubility, octanol water partition coefficient, soil sediment adsorption coefficient, Henry's Law constant, and mobility index. Calculated values, obtained using approximation methods, are presented when literature values are unavailable. A discussion of the environmental significance of each of these properties follows.

> Vapor pressure is an indication of the rate at which a chemical will volatilize. It is of primary significance as a removal mechanism at environmental interfaces such as surface soil air and surface water air. Volatilization is not a significant removal mechanism when evaluating groundwater in an aquifer and subsurface soils. Vapor pressures for monocyclic aromatics, such as benzene, toluene and xylenes, are

higher than vapor pressures for PCBs. Chemicals with higher vapor pressures are expected to enter the atmosphere much more readily than chemicals with lower vapor pressures. Volatilization is a significant loss process for volatile organic compounds in surface soils and surface water.

Water solubility is used to determine the rate at which a chemical can be solubilized and potentially leached from soil by infiltrating precipitation. In general, more soluble chemicals are more readily leached than less soluble chemicals. The water solubilities presented in Table 3-1 indicate that the volatile organic chemicals are several orders of magnitude more soluble than PCBs.

The octanol water partition coefficient (K_{ow}) is a measure of the equilibrium partitioning of chemicals between octanol and water. A linear relationship between the octanol water partition coefficient and the uptake of chemicals by fatty tissues of animal and human receptors (the bioconcentration factor, BCF) has been determined (Lyman et al., 1982). The coefficient is also useful in characterizing the sorption of compounds by organic soils where experimental values are not available. The octanol water partition coefficient also is used to estimate bioconcentration factors in aquatic organisms.

The soil sediment adsorption coefficient (K_{oc}) is an indication of the tendency of a chemical to adhere to soil particles containing organic carbon. Chemicals with high soil sediment adsorption coefficients generally have low water solubilities and vice versa. This parameter may be used to infer the relative rates at which the more mobile chemicals (monocyclic aromatics) are transported in the aqueous media. Chemicals such as PCBs are relatively immobile in the environment and are preferentially bound to the soil. These compounds are not subject to aqueous transport to the extent as compounds with higher water solubilities.

Specific gravity is the ratio of the weight of a given volume of pure chemical at a specified temperature to the weight of the same volume of water at a given temperature. Its primary use is to determine whether a constituent will have a tendency to float or sink (as an immiscible liquid) in water if it is present as a pure compound or at concentrations which exceed its water solubility.

Both vapor pressure and water solubility are of use in determining volatilization rates from surface water bodies and from groundwater. The ratio of these two parameters (Henry's Law constant) is used to calculate the equilibrium constituent concentrations in the vapor (air) phase versus the liquid (water) phase for the dilute solutions commonly encountered in environmental settings.

A quantitative assessment of mobility has been developed (Laskowski, 1983) that uses water solubility (S), vapor pressure (VP), and the organic carbon partition coefficient (K_{oc}). This value is referred to as the Mobility Index (MI). It is calculated as follows:

$$MI = \log\ [(S \times VP)/K_{oc}]$$

A scale to evaluate MI is presented by Ford and Gurba (1984) as the following:

Relative MI	Mobility Description
MI > 5	Extremely mobile
0 < MI < 5	Very mobile
-5 < MI < 0	Slightly mobile
-10 < MI < -5	Immobile
MI <-10	Very immobile

The MIs for the potential COPCs are also presented in Table 3-1.

The following paragraphs summarize the site-specific fate and transport data for the potential COPCs at the Camp Allen Landfill site.

3.3.1.1 Volatile Organic Compounds

Predominant volatile organic COPCs at the Camp Allen Landfill site can be divided into two distinct classes: (1) volatile aromatics and (2) chlorinated ethanes and ethenes.

Volatile aromatics include benzene, toluene, xylenes, and ethylbenzene. The common structure of these chemicals is the benzene ring. Toluene is a methyl substituted benzene ring. Ethylbenzene is an ethyl substituted benzene ring. Xylenes are dimethyl substituted benzenes with ortho, meta and para isometric forms.

Chlorinated ethanes and ethenes include trichloroethene, tetrachloroethene, tetrachloroethane, vinyl chloride, 1,2-dichloroethane, total 1,2-dichloroethene and 1,1,1-trichloroethane. Vinyl chloride, 1,2-dichloroethene and 1,2-dichloroethane are most likely present as a result of the degradation of higher chlorinated ethenes and ethanes.

Volatile organics tend to be very mobile in environmental media as indicated by their presence in groundwaters throughout Areas A and B and surface waters which receive groundwaters. Their inherent mobility and relatively high MIs result from high water solubilities, high vapor pressures, low K_{oc} and K_{ow} values. Volatile organics do not tend to persist in environmental media because

photolysis, oxidation and biodegradation figure significantly in their removal. They are seldom detected in surface soils (0 to 6 inches) where volatilization and other removal processes predominate. This is the case at the Camp Allen Landfill site.

3.3.1.2 Semivolatile Organic Compounds

In general, SVOCs are somewhat less mobile than the VOCs by virtue of their lower vapor pressures and lower water solubilities. K_{oc} and K_{ow} values for SVOCs are generally greater in magnitude than those for the VOCs, indicating the tendency for this class of compounds to adsorb strongly to soils and sediments. Table 3-1 shows that MI values for semivolatile COPCs at the Camp Allen Landfill site range from -15 to 2.7. These indicate that the mobilities of the semivolatile organic COPCs at the Camp Allen Landfill range from very immobile to very mobile in environmental media.

3.3.1.3 Pesticides/PCBs

Pesticides/PCBs are extremely persistent and immobile chemicals in environmental media. These chemicals are also bioaccumulated and biomagnified in the food chain.

PCBs have low vapor pressures, low water solubilities and high K_{oc} and K_{ow} values (Clement, 1985). Adsorption to organic material in soils or sediments is probably the major fate of these contaminants in the environment.

PCBs are degraded by soil microorganisms and photolysis. Heavily chlorinated PCBs like Aroclor-1260 can be photolyzed by ultraviolet light, which is an extremely slow process. Photolysis of the heavier chlorinated PCBs might be the most important degradation process for these persistent contaminants.

Technical DDT is a mixture of DDT and two primary isomers DDD and DDE. Volatilization is probably the most important transport process from soils and waters, as evidenced by the ubiquitous nature of DDT, DDD and DDE in the environment (Clement, 1985). In addition, sorption, bioaccumulation, photolysis, and biodegradation are other fate processes contributing to environmental transport at the Camp Allen Landfill site.

3.3.1.3 Inorganics

Different inorganic species behave differently in various environmental media. In general, inorganics can be transported through air, adhering to blowing dusts, or move through surface water and groundwater as dissolved salts. Inorganics can also be carried with flowing waters on suspended solids or attached to colloidal materials.

The most complicated pathway for inorganic chemicals is migration in subsurface soils and groundwater, where oxidation reduction potential (Eh) and pH play critical roles. Table 3-2 presents an assessment of relative inorganic environmental mobilities as a function of Eh and pH. Soils at the Camp Allen Landfill site are relatively neutral, therefore, inorganics in the subsurface should be relatively immobile.

3.3.2 Potential Migration Pathways

This section identifies the potential migration routes of COPCs at Areas A and B. These mechanisms were identified through an evaluation of the analytical results and known site characteristics.

3.3.2.1 Groundwater

Contaminants which come into contact with groundwater can migrate off site under the influence of groundwater flow. Migration through groundwater is dependent on the chemical nature of the contaminant and the chemical and physical nature of the aquifer. Groundwater flow velocity (a function of hydraulic gradient and conductivity), groundwater chemistry, and the chemical make up of the aquifer are all factors which affect contaminant migration. Mobility of a contaminant in groundwater is particularly influenced by it's solubility and the organic carbon partition coefficient. Compounds that have high solubility and low K_{oc} values such as vinyl chloride tend to be more mobile in groundwater than those with low solubility and high K_{oc} values such as PCBs.

3.3.2.2 Soil

Inorganic and organic compounds were detected in the soils at Areas A and B. The factors which control contaminant migration through soil is dependent on the chemical and physical nature of the contaminants and of the soil and site hydrology. Some of the factors which influence the migration of chemicals in soil include pH, oxidation reduction reactions (Eh), particle size distribution, pore size distribution, lime content, content of organic matter, concentration of ions or salts, aerobic and anaerobic conditions, presence or absence of hydrous oxides, vegetative cover, topography and climate.

3.3.2.3 Surface Water Sediment

Most of Areas A and B are surrounded by drainage ditches which collect surface water runoff and could potentially be recharged by shallow groundwaters. These ditches appear to drain to the Bousch Creek remnant which ultimately flows to Willoughby Bay approximately 2 miles north of the landfill.

Migratory pathways associated with surface water and sediment include the transport of contaminants via surface water movement, adsorption/desorption of contaminants to sediments, and discharge to groundwater. The adsorption/desorption process, from surface water to sediments, can create contaminant "sinks" in which benthic macroinvertebrates and water column organisms (i.e., fish) may be subject to contaminant exposure via the food chain. Adsorption/desorption mechanisms involve complex chemical and biochemical reactions. As chemicals are desorbed from sediment, they are then available for uptake to organisms in the water column; the higher the solubility of the chemical, the more bioavailable it is to aquatic organisms, but the less it tends to bioaccumulate.

3.3.2.4 Air

There are two potential release mechanisms to be considered in evaluating the atmospheric pathway: the release of COPCs adsorbed onto dust particulates (i.e., fugitive dust emissions) and volatilization of contaminants from soil and groundwater. The transport mechanism is the air, and the potential exposure points are the areas of human activity on and adjacent to the Camp Allen Landfill site.

Fugitive Dust Emissions.

This air pathway was evaluated as a source of exposure at outdoor locations at the site. Evaluation of this pathway introduces a large degree of conservatism into the baseline RA since much of the site is either paved or grass-covered. These conditions would normally preclude the generation and emissions of fugitive dusts.

Air exposure may occur when surface soils become airborne due to wind erosion or vehicular traffic. It is assumed that potential human receptors at the site may inhale soil particulates while engaging in outdoor activities. This assumption was applied to both current and future scenarios.

Volatilization

The results of the air sampling investigation performed at the Camp Allen Landfill site indicated detectable concentrations of VOCs in both indoor and outdoor air. Detected outdoor air concentrations can occur from volatilization of VOCs from surface soils. Detected indoor air concentrations can occur from the volatilization of organic COPCs from soil and/or groundwater, followed by intrusion through cracks in building foundations. Indoor air concentrations of some VOCs can result from other sources, as discussed in Section 2.2.6 of this report (i.e., cleaning products, solvents and coolant for refrigeration and air conditioning units). It is assumed that potential human receptors at the site may inhale soil particulates while engaging in indoor activities. This assumption was applied only to current scenarios.

3.3.3 Conceptual Site Model

Development of a conceptual site model of potential exposure is critical in evaluating all potential exposures for the aforementioned human receptors. The conceptual site model describes the area of concern in terms of suspected sources of contamination, the affected media and all potential routes of migration of the contaminants present. Conceptual site models for Areas A and B are presented in Figures 3-1 and 3-2, respectively.

The primary sources of contamination have been identified as the landfills in Areas A and B. The primary release mechanisms at these sites are stormwater runoff and contaminant migration through

groundwater from the landfills. In reality, fugitive dust generation may not be a significant potential release mechanism in Areas A and B since the areas are grass covered and are not traversed or disturbed by vehicles. However, for the sake of conservatism, potential exposures via fugitive dust emissions were included in the conceptual site model.

3.3.3.1 Potential Exposure Pathways

Tables 3-3 through 3-5 summarize the potential receptors and exposure routes evaluated at the site for current and future potential land use scenarios. In addition, pathway selection criteria is included. The following paragraphs present the rationale for the selection of potential exposure pathways for human receptors at the Camp Allen Landfill.

The shallow aquifer is not used as a drinking water source. However, the deep aquifer (Yorktown) is used as a potable source of drinking water in the Norfolk area, although not in the vicinity of the site. Because the confining clay layer is missing, or is not well developed between the two aquifers in portions of both Areas A and B, the shallow and deep aquifers are potentially interconnected. Therefore, there is the potential for cross contamination of the shallow and deep aquifers. Private wells are present to the west of the Camp Allen Landfill in the Glenwood Park Residential Area that are reportedly screened in the shallow aquifer. These wells are reportedly used for lawn watering and car washing and not for potable purposes due to the general water quality of the shallow aquifer. Only two residential wells located to the west of the Camp Allen Landfill contained volatile organic COPCs.

As part of the baseline RA, potential current exposures to local adult and child receptors includes dermal contact with and ingestion of volatile organic COPCs (1,2-dichloroethane and tetrachloroethene) detected in samples collected from two residential wells screened in the shallow aquifer. Since shallow groundwater is used by some local residents for non-potable purposes, these potential exposures could occur during activities such as watering lawns, washing cars, and filling swimming pools. Local residents may also inadvertently contact groundwater at discharge locations in the ditch. However, an evaluation of the residential nonpotable uses of groundwater provides a more conservative exposure scenario. Therefore, potential shallow groundwater exposures to local residents (using groundwater for nonpotable purposes) via the pathways of accidental ingestion and dermal contact were retained for quantitative evaluation in the baseline RA. Since only trace levels

of 1,2-dichloroethane and tetrachloroethene were detected, it is highly unlikely that outdoor exposures to these COPCs, as they volatilize from water emanating from hoses during lawn watering and car washing activities, will be significant due to the infinite dilution potential of the outdoor air. Local resident adults and children will, however, be quantitatively evaluated for potential outdoor inhalation exposures to VOCs detected in the ambient air during the air sampling investigation at the Camp Allen Landfill site.

Limited access to the Area A ditch is possible through holes in the chain link fence which separates the residential area west of the Brig from the landfill. Older children and adults could potentially be exposed to surface waters and sediments within the Area A ditch via the pathways of accidental ingestion and dermal contact. Access to areas beyond the ditch will not occur due to established security measures around the Brig.

Brig prisoners and employees could potentially be exposed to COPCs in Area A by incidental ingestion of soils, dermal contact with soils, inhalation of fugitive dusts, and inhalation of COPCs that may volatilize from soils while working on site or during recreational activities. In addition, Brig prisoners and employees could also potentially be exposed to volatile organic COPCs that were detected during the Brig indoor air sampling investigation. Indoor air VOC concentrations may have resulted from potential intrusion of vapors (emanating from potentially affected underlying groundwater) through cracked foundations, volatilization from soils to ambient air, or from other sources as discussed in Section 2.2.6 of this report (i.e., cleaning products, solvents and coolant for refrigeration and air conditioning units that are used in the Brig Facility).

Brig employees could also be subject to exposure to COPCs in surface waters and sediments by dermal contact and accidental ingestion. Stormwater runoff flows into the ditches which surround both Areas A and B of the Camp Allen Landfill. Brig employees are responsible for maintaining these ditches.

Future residential development of the Camp Allen Landfill is highly unlikely and current property use of the landfill will continue in the foreseeable future. Future property use, in the event of base closure, would in all likelihood be for commercial/industrial purposes because of deed restrictions concerning former landfills. However, future potential residential exposure was evaluated for the sake of conservatism. If the Camp Allen Landfill were to be developed residentially, adult and child

residents could potentially be exposed to COPCs identified in on-site soils, surface waters, sediments and groundwaters by ingestion and dermal contact. Outdoor volatile emissions of VOCs through soils, and fugitive dust emissions of COPCs adhering to entrained particles were also evaluated, even though landscaping features and the vegetative cover found in residential areas may preclude these types of releases.

Potable use of groundwater is unlikely, even in the event of future residential development, because of the availability of municipal water and the general groundwater quality in the vicinity of the site. As a conservative approach to estimating the risk associated with the shallow and deep aquifers in this area, future potential exposure to groundwater by ingestion, dermal contact and the inhalation of volatiles while showering have been retained as exposure pathways.

Local adults and children cannot currently gain access to Area B Pond because of the fence which is actively maintained by Camp Allen personnel. Access is further limited by security efforts around the Camp Allen Salvage Yard. However, access is available to affected soils, surface waters and sediments in the school area for adult employees and children of the elementary school, thereby allowing for the occurrences of potential ingestion and dermal contact exposures to COPCs identified in these media. Inhalation of COPCs adhered to fugitive dust particulates emanating from the school area soils, volatile organic COPCs detected in outdoor ambient air, and volatile organic COPCs detected in the indoor air of the elementary school are other potential exposure pathways evaluated for adult employees and children of the elementary school. Indoor air VOC concentrations may have resulted from potential intrusion of vapors (emanating from potentially affected underlying groundwater) through cracked foundations, volatilization from soils to ambient air, or from other sources as discussed in Section 2.2.6 of this report (i.e., cleaning products, solvents and coolant for refrigeration and air conditioning units that are used in the elementary school building).

Potential exposures to future construction workers engaged in digging activities could occur by accidental ingestion and dermal contact of COPCs in subsurface soils. Inhalation of fugitive dusts being released from subsurface soils during excavation and construction activities would also be expected to affect construction workers. Therefore, potential construction worker exposures to subsurface soil COPCs in both Areas A and B via ingestion, dermal contact and inhalation of fugitive dusts were evaluated as significant pathways to this receptor group.

3.4 Quantification of Exposure

3.4.1 Concentrations Used in the Estimate of Exposure

The chemical concentrations used in the estimation of chronic daily intakes (CDIs) for each medium are representative of the types of exposure. Exposure can occur discretely or at a number of sampling locations depending on the types of scenarios considered for a given receptor. Furthermore, certain environmental media such as groundwater, surface water, sediment and air are transitory and chemical concentrations detected in these media change frequently over time. Soils are by nature less transitory than the aforementioned media.

The manner in which environmental data are represented depends on the number of samples and sampling locations available for a given area and a given medium. The 95-percent upper confidence limit for the arithmetic mean concentration (95%UCL) or the maximum detected concentration of a COPC was used as the concentration term for surface soils, subsurface soils, surface water, and sediments in order to represent the spacial nature of potential exposures to COPCs in those media. The maximum detected concentration of a COPC was used if the 95%UCL exceeded the maximum detected concentration of that COPC in a given medium.

The 95%UCL was calculated using the following equation (USEPA, 1992):

$$95\%\ UCL = \bar{x} + t(s/\sqrt{n})$$

Where: 95%UCL = 95th percent upper confidence limit for the arithmetic mean concentration

$\bar{x}$ = *mean*

s = standard deviation
t = Student t statistic
n = number of samples

As a conservative measure, only maximum detected concentrations were used to quantify chronic daily intakes due to indoor and outdoor air VOC inhalation exposures.

Exposure concentrations in Area A and Area B groundwater, in both the shallow and deep zones, were obtained from data for individual monitoring wells. Wells were selected to best represent exposures at locations that are approximately centrally situated within groundwater plumes, and that

reflect high levels of concentrations for conservative evaluations. However, this method of selecting exposure concentrations may result in some COPCs not being evaluated quantitatively in the risk assessment since not all COPCs may be present in the selected well locations. This will be discussed as an uncertainty, in greater detail, in Section 7.0. For Area A shallow groundwater, data from wells B-20W and B-20WSS were used as exposure concentrations. For Area A deep groundwater, data from well A-MW1B were used as exposure concentrations. For Area B shallow and deep groundwater, exposure concentrations were obtained from analytical data acquired for monitoring wells B-MW11A and B-MW19, respectively.

Evaluations of groundwater in Areas A and B were performed for each well location to also separately assess potential public health impacts as indicated by the summed risks resulting from exposures to groundwater containing organic and unfiltered inorganic COPCs, and the summed risks resulting from exposures to groundwater containing organic and filtered (dissolved) inorganic COPCs.

For results reported as "nondetect" (e.g., ND, U, etc.), a value of one half of the sample-specific detection limit was used to calculate the 95%UCL. A value of half the detection limit was assigned to nondetects when estimating the 95%UCL because the actual value could be between zero and a value just below the detection limit. Ninety-five percent UCLs were calculated only for the constituents detected in at least one sample collected from the environmental medium of interest.

Reported concentrations that were less than the detection limit were used to calculate the mean and the 95%UCL concentration used in the quantitative risk assessment. Typically, these values are qualified with a " J" meaning that the value is estimated. Data qualified " J" for any reason were used in the calculation of the 95th percent UCL. Reported concentrations qualified with an "R" were rejected from the data set.

Soil, surface water, and sediment analytical data were segregated into three areas within the confines of the Camp Allen Site. These were the Area A landfill (Area A), Area B landfill pond (Area B Pond), and elementary school (Area B School). These locations represent distinct areas of potential exposure because of current access.

Groundwater data were segregated into Area A shallow, Area A deep, Area B shallow, and Area B deep because of site hydrology. Two distinct plumes of groundwater contamination were identified in Area A. These plumes are discussed in detail in the RI report. From a potential exposure standpoint, no one particular area in Area A is more or less likely to undergo residential development. Therefore, groundwater data in Area A was not further divided for risk assessment purposes. Groundwater in Area A may be divided into two distinct zones in the Feasibility Study, where corresponding potential human health risks for Area A subunits will be discussed for remediation purposes.

In order to quantitatively assess the inhalation of contaminants volatilized from shower water, the model developed by Andelman (1985) was utilized. Contaminant concentrations in air due to VOCs while showering were modeled by estimating the rate of VOC releases into air based on the following: the initial concentration of the VOC in the shower water; the known rate of release of trichloroethene (TCE) from water; Henry's Law coefficient for TCE; Henry's Law coefficient for the VOC; the asymptotic concentration of the chemical in air; flow rates of air and water in the shower; the volume of the shower room; and the assumed duration of shower time. Relevant equations and spreadsheets that were used to estimate VOC shower air concentrations are presented in Appendix B (Spreadsheets B-1 through B-4).

3.4.2 Estimation of Daily Chronic Intake

The equations for estimating exposure to site contaminants for the various identified exposure pathways are as follows.

3.4.2.1 Groundwater and Surface Water

Ingestion of Potable Groundwater

The daily intake associated with the direct potential ingestion of the chemicals of concern in groundwater under a potable use scenario was calculated using the following equation (USEPA, 1989):

$$CDI = \frac{Cw \ x \ IR \ EF \ ED}{BW \ x \ AT}$$

Where:	CDI	=	Chronic Daily Intake, mg/kg/day
	Cw	=	Chemical concentration in water, mg/L
	IR	=	Ingestion rate, L/day
	EF	=	Frequency of exposure, days/year
	ED	=	Exposure duration, years
	BW	=	Average body weight, kg
	AT	=	Averaging time, days

Accidental Ingestion of Non-Potable Groundwater and Surface Water

The daily intake associated with the accidental ingestion of COPCs in groundwater under a non-potable use scenario (e.g., washing cars and water lawns) and surface water was calculated using the following equation (USEPA, 1989a):

$$CDI = \frac{Cw \ x \ IR \ x \ ET \ x \ EF \ x \ ED}{BW \ x \ AT}$$

Where:	CDI	=	Chronic Daily Intake, mg/kg/day
	Cw	=	Chemical concentration in water, mg/L
	IR	=	Ingestion rate, L/day
	ET	=	Exposure time, hours/event
	EF	=	Frequency of exposure, events/year
	ED	=	Exposure duration, years
	BW	=	Average body weight, kg
	AT	=	Averaging time, days

Dermal Contact with Groundwater and Surface Water

The dermally absorbed dose associated with potential dermal contact with COPCs in groundwater (showering or bathing) and surface water was calculated using the following equation (USEPA, 1992):

$$DAD = \frac{DA \ x \ A \ x \ EV \ x \ EF \ x \ ED}{BW \ x \ AT}$$

Where:	DAD	=	Dermally absorbed dose, mg/kg/day
	DA	=	Absorbed dose per event, mg/cm^2-event

A	=	Surface area of exposed skin, cm^2
EV	=	Event frequency, events/day
EF	=	Exposure frequency, days/year
ED	=	Exposure duration, years
BW	=	Average body weight, kg
AT	=	Averaging time, days

DA was calculated for organics and inorganic COPCs in accordance with USEPA's guidance entitled: Dermal Exposure Assessment: Principles and Applications, Interim Report (USEPA, 1992). The steady-state and non-steady-state models were used in estimating DA for inorganic and organic COPCs, respectively. Relevant equations and factors required for estimating DA were calculated in Appendix B (Spreadsheet B-5).

Inhalation Volatilized Organic COPCs during Showering

The chronic daily intake resulting from the inhalation of COPCs that volatilize from water during showering was calculated using the following equation (USEPA, 1989):

$$CDI = \frac{Ca \ x \ RR \ x \ ET \ x \ EF \ x \ ED}{BW \ x \ AT}$$

Where:	CDI	=	Chronic Daily Intake, mg/kg/day
	Ca	=	Chemical concentration in air, mg/m^3, as determined from Andelman Shower Model (Andelman, 1985)
	RR	=	Respiration rate, m^3/day
	EF	=	Frequency of exposure, days/year
	ED	=	Exposure duration, years
	BW	=	Average body weight, kg
	AT	=	Averaging time, days

3.4.2.2 Surface Soils, Subsurface Soils, and Sediments

Accidental Ingestion of Soils and Sediments

The daily intake associated with the potential accidental ingestion of COPCs detected in soils (surface and subsurface) and sediments was calculated using the following equation (USEPA, 1989):

$$CDI = \frac{Cs \ x \ IR \ x \ CF \ x \ FI \ x \ EF \ x \ ED}{BW \ x \ AT}$$

Where:	CDI	=	Chronic Daily Intake, mg/kg/day
	Cs	=	Chemical concentration in soil/sediment mg/kg
	IR	=	Ingestion rate, mg/day
	FI	=	Fraction of soil/sediment ingested from site, unitless
	CF	=	Conversion Factor, 10^{-6} kg/mg
	EF	=	Frequency of exposure, days/year
	ED	=	Exposure duration, years
	BW	=	Average body weight, kg
	AT	=	Averaging time, days

Dermal Contact with Soils and Sediments

The dermally absorbed dose associated with the potential dermal contact of COPCs in soils (surface and subsurface) and sediments was calculated using the following equation (USEPA, 1992):

$$DAD = \frac{DA \ x \ CF \ x \ A \ x \ EF \ x \ ED}{BW \ x \ AT}$$

Where:	DAD	=	Dermally absorbed dose, mg/kg/day
	DA	=	Absorbed dose per event, mg/cm^2-event
		=	Cs*ABS*AF (USEPA, 1992)
			where: Cs = Chemical concentration in soil/sediment, mg/kg
			ABS = Absorbance factor, unitless
			AF = Soil to Skin Adherence Factor, mg/cm^2
	CF	=	Conversion factor, 10^{-6} kg/mg
	A	=	Surface area of exposed skin, cm^2
	EV	=	Event frequency, events/day
	EF	=	Exposure frequency, days/year
	ED	=	Exposure duration, years
	BW	=	Average body weight, kg
	AT	=	Averaging time, days

Inhalation of Fugitive Dusts Emanating from Surface Soils and Excavated Subsurface Soils

The chronic daily intake resulting from the inhalation of COPCs adsorbed onto fugitive dust particulates was estimated using the following equation (USEPA, 1989):

$$CDI = \frac{Ca \ x \ RR \ x \ EF \ x \ ED}{BW \ x \ AT}$$

Where:	CDI	=	Chronic Daily Intake, mg/kg/day
	Ca	=	Chemical concentration in air as fugitive dusts, mg/m^3
	RR	=	Respiration rate, m^3/day
	EF	=	Frequency of exposure, days/year

ED	=	Exposure duration, years
BW	=	Average body weight, kg
AT	=	Averaging time, days

The air concentration (Ca) of a chemical in fugitive dust emissions was estimated by multiplying the soil concentration of the chemical (Cs) by the default particulate emission factor (PEF = 4.63×10^9 m^3/kg) as determined by Cowherd (1985), and provided by USEPA (1991a).

3.4.2.3 Indoor and Outdoor Ambient Air

The chronic daily intake resulting from the inhalation of a volatile COPC in indoor and outdoor ambient air was estimated using the following equation:

$$CDI = \frac{Ca \times RR \times ET \times EF \times ED}{BW \times AT}$$

Where:	CDI	=	Chronic Daily Intake, mg/kg/day
	Ca	=	Chemical concentration measured in ambient air, mg/m^3
	RR	=	Respiration rate, m^3/day
	EF	=	Frequency of exposure, days/year
	ED	=	Exposure duration, years
	BW	=	Average body weight, kg
	AT	=	Averaging time, days

3.4.3 Exposure Factors Used To Derive Chronic Daily Intakes

Tables 3-6 through 3-12 present the exposure factors used in the estimation of potential chronic daily intakes for COPCs retained for each environmental medium. USEPA promulgated exposure factors are used in conjunction with USEPA standard default exposure factors. When USEPA exposure factors are not available, best professional judgment and site-specific information are used to derive a conservative and defensible value. The following paragraphs present the rationale for the selection of exposure factors for each receptor group evaluated in the Residential Area, Area A, and Area B Pond and Area B School.

3.4.3.1 Area A and the Residential Area

Local Adults

Local adults living in the residential area west of Area A could potentially be exposed to COPCs detected in shallow groundwater obtained from residential wells, surface water and shallow sediments. Table 3-6 presents the exposure factors used to quantify the potential exposure to local adults.

On-site groundwater is not currently being used for any purpose; however, residential wells exist in the Glenwood neighborhood. Residential wells are reportedly screened in the shallow zone and adults could potentially be exposed to COPCs in shallow groundwater while washing cars, watering lawns, or swimming. It was conservatively assumed that activities (primarily car washing) involving non-potable groundwater usage could occur 28 days per year (EF), or 4 times per month during April through October. The USEPA exposure time (ET) for surface water, 2.6 hours, was assumed for each event. COPCs in groundwater could potentially be ingested accidentally and contacted dermally during each event. The USEPA ingestion rate (IR) for surface water of 50 ml per hour (derived for swimming exposures) was used as a conservative estimate of accidental groundwater ingestion. The adult skin surface area (SA) available for dermal contact was estimated to be 20,000 cm^2, representing total body exposure. Equations and chemical-specific permeability constants (Kp) presented by USEPA (1992) were used to estimate the non-steady-state absorption of organic COPCs and the steady-state absorption of inorganic COPCs by skin exposed to groundwater. The exposure duration assumed for the adult was 30 years.

Potential local adult exposures to surface waters and sediments, present in the Area A ditch would be limited. The ditch is not large enough for recreational uses such as swimming or fishing and could go dry during the summer months. Nevertheless, local adults could gain access to the ditches through holes in the chain link fence. It was assumed that the EF for local adults was 7 days per year which corresponds to the national average swimming frequency value. Potential dermal contact and accidental ingestion of surface water and sediment were evaluated. IR values of 50 ml of surface water per hour and 50 mg per day of sediment were used. These represent USEPA's accidental ingestion rate for swimming exposures and the default residential adult accidental soil ingestion rate. An adult SA value of 5,300 cm^2, representing surface area available for contact for an adult wearing

a short sleeve shirt, shorts and shoes, was used. Equations and chemical-specific permeability constants (Kp) presented in by USEPA (1992) were used to estimate the non-steady-state absorption of organic COPCs and the steady-state absorption of inorganic COPCs by skin exposed to surface water. A dermal adherence factor (AF) of 1 mg/cm^2 was used to estimate the adherence of affected sediments to the skin. Experimentally derived absorbance values for classes of chemicals were used in determining CDIs for local resident older child and adult trespassers that would result from potential dermal exposures to sediments. The following summarizes these values as the percent of chemical estimated to be absorbed through the skin and into the bloodstream.

VOCs - 25% (Ryan, et al., 1987)
SVOCs - 10% (Ryan, et al, 1987)
Pesticides - 10% (Ryan, et al., 1987)
PCBs - 6% (USEPA, 1992)
Arsenic - 3% (Wester, et al., 1993)
Inorganics - 1% (Ryan, et al., 1987)

Each of these values represent the upper limits of their respective absorbance ranges as presented in the cited references. Local adults could also inhale volatilized COPCs that have been detected in outdoor ambient air. A respiration rate of 0.83 m^3/hour is assumed for potential inhalation exposures.

Local Children

Local young children (ages 1-6 years) living in the residential area in the vicinity of Area A could potentially be exposed to COPCs detected in groundwaters, surface waters and sediments. Table 3-6 presents the exposure factors used to quantify potential exposure by local children.

Groundwater is not currently used as a potable water source in Area A. Residential wells screened in the shallow zone west of Area A are used for lawn watering, washing cars and could possibly be used to fill swimming pools in which children swim. Young children could potentially be exposed to COPCs in residential area shallow groundwaters during nonpotable uses such as swimming. An EF of 28 days per year (corresponding to four times the national average swimming frequency of 7 days per year) was used to evaluate the potential current human health risks associated with dermal contact and accidental ingestion of affected groundwater. A young child weighing 15 kg was used in the evaluation of current potential residential area groundwater exposure. A skin SA value of

8,023 cm^2 was used to represent the 95th percentile whole body surface area. An IR value of 0.05 liters per hour was used to represent the rate of potential accidental groundwater ingestion. An exposure time of 2.6 hours was used which represents the national average swimming duration. Equations and chemical-specific permeability constants (Kp) presented by USEPA (1992) were used to estimate the non-steady-state absorption of organic COPCs and the steady-state absorption of inorganic COPCs by skin exposed to groundwater.

Older local children (ages 6 through 15) living in the vicinity of Area A could also access the ditches through holes in the chain link fence, and potentially contact surface waters and sediments therein. It was assumed that younger children (ages 1 to 6 years of age) could not gain access to the surface waters or sediments surrounding Area A because of the proximity of the ditches to the residential area and the relatively inaccessible nature of the ditch location. It was assumed that local children could potentially access ditch surface waters and sediments 28 days per year (EF) over the course of a nine year period. Exposure to COPCs detected in surface water and sediment could potentially occur by dermal contact and accidental ingestion. An IR value of 50 ml per hour for surface water, in conjunction with an exposure time of 2.6 hours per day and an IR of 100 mg per day for sediments were used to evaluate potential exposure. Dermal exposure was estimated to occur over 3,683 cm^2 of skin. Equations and chemical-specific permeability constants (Kp) presented by USEPA (1992) were used to estimate the non-steady-state absorption of organic COPCs and the steady-state absorption of inorganic COPCs by skin exposed to surface water. The same experimentally derived dermal absorption values used for local adults were also used to estimate shallow sediment exposures to older children. Local older children could also inhale volatilized COPCs that have been detected in outdoor ambient air (USEPA, 1992, 1994b). A respiration rate of 0.83 m^3/day is assumed for potential inhalation exposures. The older children were assumed to weigh 37 kg (BW).

Brig Employees and Prisoners

Military personnel and civilians are employed at the Area A Brig. The Brig houses awardees and prisoners who may be assigned there. Employees could potentially contact COPCs present in Area A surface soils, ditch surface waters, and sediments via accidental ingestion and dermal contact. Prisoners could potentially contact COPCs present in Area A surface soils via accidental ingestion and dermal contact. Both employees and prisoners could potentially inhale COPCs in air

that have either volatilized from soils or that have adsorbed onto fugitive dust particulates. Table 3-7 provides a summary of the exposure factors used to evaluate potential exposure in the Brig area.

Civilian employees could potentially be subjected to the longest employment times because military employees are usually assigned to three year rotations (Askew, R.. Personal correspondence, 1993). Civilian Brig employees would, therefore, have the longest potential ED and were selected as the most conservative receptor for evaluating potential employee exposure.

USEPA's default exposure factors for evaluating commercial industrial scenarios were used to evaluate the potential dermal contact and accidental ingestion of Area A soils by civilian employees. Potential employee exposure to ditch sediments were evaluated using the commercial industrial default values with the exception of EF. Brig employees routinely supervise or take part in maintenance of the vegetation which grows along the ditches. This activity may result in potential exposures to surface water and sediments in the ditch. An EF value of 12 days per year was derived assuming that the vegetation is cut twice monthly, May through October. An SA value of 4,100 cm^2 was used, which assumes that dermal contact could occur to the head, forearms, and hands during ditch maintenance. This corresponds to an individual wearing a short sleeve shirt, long pants and shoes. IR values of 50 ml per hour and 50 mg per day were assumed for surface water and sediment, respectively. An ET of 2.6 hours per day was assumed for the estimation of chronic daily intake resulting from ingestion and dermal exposures to surface water.

Prisoners, awardees, and employees could potentially be exposed to COPCs in Brig area soils on a daily basis. Prisoners can be assigned to the Brig for durations of 2 years while awardees are generally assigned to the Brig for 6 months or less. Brig prisoners would, therefore, provide the most conservative estimate of exposure and were selected for the evaluation of potential human health effects. Brig employees were assumed to be exposed to soils for a 25 year period (ED), 250 days per year (EF). Brig prisoners were assumed to be exposed to soils for a 2 year period (ED), 350 days per year (EF). An SA value of 4,100 cm^2 was assumed for dermal exposure with soils. An ingestion rate of 50 mg per day, corresponding to a commercial industrial soil ingestion rate, was used.

Equations and chemical-specific permeability constants (Kp) presented by USEPA (1992) were used to estimate the non-steady-state dermal absorption of organic COPCs and the steady-state absorption of inorganic COPCs by skin exposed to surface water. Experimentally derived absorbance values for classes of chemicals were used in determining CDIs for Brig employees and Brig prisoners that would result from potential dermal exposures to soil/sediments. The following summarizes these values as the percent of chemical estimated to be absorbed through the skin and into the bloodstream.

VOCs - 25% (Ryan, et al., 1987)
SVOCs - 10% (Ryan, et al, 1987)
Pesticides - 10% (Ryan, et al., 1987)
PCBs - 6% (USEPA, 1992)
Arsenic - 3% (Wester, et al., 1993)
Inorganics - 1% (Ryan, et al., 1987)

Each of these values represent the upper limits of their respective absorbance ranges as presented in the cited references.

Inhalation exposures to COPCs that have volatilized to indoor and outdoor air, and to COPCs that are adsorbed onto fugitive dust particulates could occur to employees and prisoners. For prisoners, these exposures can occur for 2 hours and 22 hours per day for outdoor and indoor air, respectively. For employees stationed at indoor and outdoor locations, inhalation exposures were assumed to occur for 10 hours per day. An inhalation rate of 0.83 m^3/hour was assumed for both Brig prisoners and employees.

Future Resident Adults and Children

Future residential development of the Camp Allen Landfill, is highly unlikely. For the sake of conservatism, the potential exposure pathways associated with future potential residential development was evaluated. Table 3-8 presents the exposure factors used in the future potential residential development scenarios. Future adult and young child (ages 1-6 years) residents were evaluated for potential exposures due to ingestion and dermal contact with groundwater used for potable purposes, surface soils, surface water and shallow and deep sediments. Future residents were also evaluated for potential exposures due to inhalation of fugitive dusts, COPCs that may volatilize from shower water, and volatilized COPCs detected in indoor and outdoor air samples.

Inhalation rates of 0.83 m^3/hour and 0.6 m^3/hour were assumed for the adult and young child residents, respectively.

USEPA's standard default exposure factors were used in the evaluation of COPCs detected in soils and groundwaters for future potential adult and child residents of Area A. Default exposure factors concerning frequency and duration of exposure, rate of ingestion and adherence rates are not currently available for surface waters or sediments. Therefore, professional judgment in conjunction with local meteorological data was used to establish exposure frequency, exposure duration, adherence and rate of ingestion.

Equations and chemical-specific permeability constants (Kp) presented by USEPA (1992) were used to estimate the non-steady-state dermal absorption of organic COPCs and the steady-state absorption of inorganic COPCs by skin exposed to groundwater and surface water. Experimentally derived absorbance values for classes of chemicals were used in determining CDIs for future resident children and adults that would result from potential dermal exposures to soils and sediments. The following summarizes these values as the percent of chemical estimated to be absorbed through the skin and into the bloodstream.

VOCs - 25% (Ryan, et al., 1987)
SVOCs - 10% (Ryan, et al, 1987)
Pesticides - 10% (Ryan, et al., 1987)
PCBs - 6% (USEPA, 1992)
Arsenic - 3% (Wester, et al., 1993)
Inorganics - 1% (Ryan, et al., 1987)

Each of these values represent the upper limits of their respective absorbance ranges as presented in the cited references.

3.4.3.2 Area B

Adult Workers

Adult workers (Brig employees and prisoners) cut the grass and maintain the fencing in Area B Pond. Through these activities, Brig employees could potentially contact COPCs by dermal contact and accidentally ingesting surface soils, surface waters, and shallow sediments. Adult workers

could also be exposed to COPCs in fugitive dusts and COPCs that have volatilized from soils via the inhalation pathway. Table 3-9 presents the exposure factors used to quantify potential intakes by Brig employees potentially exposed in Area B Pond.

Local Adults

Local adults living in the vicinity of Area B could potentially be exposed to COPCs present in soils, surface waters and shallow sediments in Area B School. Table 3-10 presents the exposure factors used to quantify potential exposure to local adults.

Local adults working at the Camp Allen Elementary School could potentially be exposed to COPCs detected in Area B soils during routine maintenance duties. USEPA's default commercial industrial exposure factors were used to evaluate potential adult soil exposure to Area B soils. Local adults working at the school could also potentially be exposed to surface waters and shallow sediments in Area B School ditches. These ditches are not large enough for recreational activities such as swimming and fishing. The ditches also go dry periodically, therefore, potential exposure would be limited to maintenance related exposure when water pools in these ditches.

Adult employees of the Elementary School could potentially be exposed, via the inhalation pathway, to COPCs in fugitive dusts, as well as COPCs that have been detected in indoor and outdoor air.

Potential local adult exposure to soils, surface waters, and sediments in Area B Pond is limited. Area B Pond is fenced, and the integrity of the fence is routinely maintained. Furthermore, security efforts around the Camp Allen Salvage Yard, adjacent to Area B Pond, further limits the potential for access in this area.

Groundwater is not currently used for any purpose in Area B and residential wells do not exist in the vicinity of the Area B landfill. Groundwater, therefore, was not evaluated using current potential exposure scenarios.

Local Children

Local children living in the vicinity of Area B could also potentially be exposed to COPCs detected in soils, surface waters and shallow sediments. Table 3-10 presents the exposure factors used to quantify potential exposure by local children.

Local children attending the Camp Allen Elementary School could potentially be exposed to surface soils in the vicinity of the school. Children attending the Elementary School were assumed to be age 6 to 12 years of age (grades K through 6). These children could potentially be exposed for an EF of 180 days per year for 6 years (ED). Exposure to COPCs detected in surface water and sediment could potentially occur by dermal contact and accidental ingestion. An IR value of 50 ml per hour for surface water, in conjunction with an exposure time of 2.6 hours per day and an IR of 100 mg per day for sediments were used to evaluate potential exposure. Dermal exposure was estimated to occur over 3,242 cm^2 of skin (head, arms, hands, and lower legs). The older children were assumed to weigh 31 kg (BW).

Children attending the Elementary School could potentially be exposed, via the inhalation pathway, to COPCs in fugitive dusts, as well as COPCs that have been detected in indoor and outdoor air.

Future Resident Adults and Children

Future residential development of the Camp Allen Landfill, Area B is highly unlikely. For the sake of conservatism and in the event of a base closure, the potential exposure pathways associated with future potential residential development was evaluated. Table 3-11 presents the exposure factors used in the future potential residential development scenarios.

USEPA's standard default exposure factors were used in the evaluation of COPCs detected in soils and groundwaters for future potential adult and child residents of Area B. Residential default exposure factors concerning frequency of exposure, rate of ingestion and sediment adherence rates are not currently available for surface waters or sediments. Therefore, professional judgment in conjunction with meteorological data was used to establish exposure frequency, adherence and rate of ingestion.

3.4.3.3 Subsurface Soils

Potential exposures to subsurface soil COPCs in both Areas A and B may occur to construction workers while performing soil excavation and construction activities. Exposure pathways evaluated include accidental ingestion, dermal contact and inhalation of fugitive dusts. Table 3-12 presents values for exposure input parameters used in estimating intakes for construction workers. Exposure was assumed to occur 8 hours per day, 250 days per year, for a construction period of 1 year. USEPA default values for the soil ingestion rate (480 mg/day) and the inhalation rate (0.83 m^3/hour) were also assumed for the construction worker. A skin surface area of 4,100 cm^2 was evaluated for dermal contact with subsurface soils. Experimentally derived absorbance values for classes of chemicals were used in determining CDIs for future construction workers that would result from potential dermal exposures to subsurface soils. The following summarizes these values as the percent of chemical estimated to be absorbed through the skin and into the bloodstream.

VOCs - 25% (Ryan, et al., 1987)
SVOCs - 10% (Ryan, et al, 1987)
Pesticides - 10% (Ryan, et al., 1987)
PCBs - 6% (USEPA, 1992)
Arsenic - 3% (Wester, et al., 1993)
Inorganics - 1% (Ryan, et al., 1987)

Each of these values represent the upper limits of their respective absorbance ranges as presented in the cited references.

SECTION 3.0 TABLES

TABLE 3-1

PHYSICAL AND CHEMICAL PROPERTIES OF CHEMICALS OF POTENTIAL CONCERN
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Constituents	Vapor Pressure (mm Hg)	Water Solubility (mg/L)	K_{oc} (ml/g)	K_{ow} log	Specific Gravity (g/cm^3)	Henry's Law Constant (atm-m^3/mole)	Mobility Index
VOCs: benzene	9.52E+01	1.75E+03	83	2.12	0.879	5.59E-03	3.3
benzyl chloride	1.00	3.30E+03	50	2.63	1.10	5.06E-05	1.8
bromomethane (methyl bromide)	1.63E+03	1.75E+04	126	1.19	1.73	6.24E-03	5.3
2-butanone	7.75E+01	2.68E+05	4.5	0.26	0.805	2.74E-05	6.7
chlorobenzene	1.71E+01	4.66E+02	330	2.84	1.107	3.72E-03	1.4
chloroform	1.51E+02	8.20E+03	31	1.97	1.489	2.87E-03	4.6
chloromethane (methyl chloride)	4.31E+03	6.50E+03	35	0.95	--	4.40E-02	5.9
1,4-dichlorobenzene	1.18	7.90E+01	1700	3.60	1.458	2.89E-03	-1.3
1,1-dichloroethane	180	5,500	43	1.79	1.250	4.31E-03	4.4
1,2-dichloroethane	6.40E+01	8.52E+03	14	1.48	1.250	9.78E-04	4.6
1,1-dichloroethene	6.00E+02	2.25E+03	65	1.84	1.218	3.40E-02	4.3
1,2-dichloroethene	3.24E+02	6.30E+03	59	0.48	1.260	6.56E-03	4.5
4-methyl-2-pentanone (methyl isobutyl ketone)	14.5	2.04E+04	65	1.19	0.802	9.40E-05	3.6
methylene chloride (dichloromethane)	3.62E+02	2.00E+04	8.8	1.30	1.327	2.03E-03	5.9
tetrachloroethene	1.78E+01	1.50E+02	364	2.6	1.626	2.59E-02	0.86
toluene	2.81E+01	5.35E+02	300	2.73	0.867	6.37E-03	1.7
1,1,1-trichloroethane	1.23E+02	1.50E+03	152	2.5	1.350	1.44E-02	3.1
trichloroethene	5.79E+01	1.10E+03	126	2.38	1.46	9.10E-03	2.7
1,2,4-trimethylbenzene	--	--	--	--	0.89	--	--
1,3,5-trimethylbenzene	--	--	--	--	0.86	--	--
vinyl chloride	2.66E+03	2.67E+03	57	1.38	0.912	8.19E-02	5.1
m-xylene	1.00E+01	1.30E+02	166	3.26	0.88	7.68E-03	0.9
o-xylene	1.00E+01	1.75E+02	129	2.95	0.88	5.10E-03	1.1

TABLE 3-1 (Continued)

PHYSICAL AND CHEMICAL PROPERTIES OF CHEMICALS OF POTENTIAL CONCERN
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE NORFOLK, VIRGINIA

Constituents	Vapor Pressure (mm Hg)	Water Solubility (mg/L)	K_{oc} (ml/g)	K_{ow} log	Specific Gravity (g/cm^3)	Henry's Law Constant (atm-m^3/mole)	Mobility Index
p-xylene	1.00E+01	1.98E+02	204	3.15	0.86	7.68E-03	1.0
xylenes (total)	1.00E+01	1.98E+02	240	3.26	0.870	7.04E-03	0.9
SVOCs: acenaphthene	1.00E-03	3.42	2.14E+04	4.22	0.899	--	-6.8
Bis(2-chloroethyl)ether	7.10E-01	1.02E+04	13.9	1.50	--	1.31E-05	2.7
Bis(2-ethylhexyl)phthalate	2.70E-07	1.3	5.37E+08	3.57	0.99	1.07E-07	-15
2,4-dimethylphenol	--	--	--	--	--	--	--
hexachlorobutadiene	2.00	1.50E-01	29000	4.18	--	4.57	-4.9
2-methylphenol (o-cresol)	3.10E-01	3.08E+04	22	1.95	1.041	1.60E-06	2.6
4-methylphenol (p-cresol)	1.30E-01	2.26E+04	49	1.94	1.035	9.60E-07	1.8
Pesticides: Aldrin	6.00E-06	1.80E-01	9.60E+04	5.30	--	1.60E-05	-11
beta-BHC	1.70E-05	17	1.38E+04	3.29	1.87	4.47E-07	-6.8
gamma-BHC (Lindane)	1.60E-04	7.80	1080	3.90	1.87	7.85E-06	-5.4
alpha-Chlordane	1.00E-05	5.60E-01	1.40E+05	3.32	--	9.63E-06	-7.9
4,4'-DDD	1.89E-06	1.00E-01	7.70E+05	6.20	--	7.96E-06	-12
4,4'-DDE	6.50E-06	4.00E-02	4.40E+06	7.00	--	6.80E-05	-10
4,4'-DDT	5.50E-06	5.00E-03	2.43E+05	6.19	--	5.13E-04	-14
Dieldrin	1.78E-07	1.95E-01	1700	3.50	1.75	4.58E-07	-12
Heptachlor epoxide	3.00E-04	3.50E-01	220	2.70	--	4.39E-04	-7.9
PCBs: Aroclor-1254	7.70E-05	3.00E-02	1.07E+06	4.59	1.50	2.80E-03	-10
Aroclor-1260	4.10E-05	3.00E-03	1.29E+06	4.87	1.58	7.1E-03	-12

Notes: -- = Value not available.

TABLE 3-2

RELATIVE MOBILITIES OF INORGANICS AS A FUNCTION OF ENVIRONMENTAL CONDITIONS (Eh, pH)
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Relative Mobility	Environmental Conditions			
	Oxidizing	Acidic	Neutral/ Alkaline	Reducing
Very high			Se	
High	Se, Zn	Se, Zn, Cu, Ni, Hg, Ag		
Medium	Cu, Ni, Hg, Ag, As, Cd	As, Cd	As, Cd	
Low	Pb, Ba, Se	Pb, Ba, Be	Pb, Ba, Be	
Very Low	Fe, Cr	Cr	Cr, Zn, Cu, Ni, Hg, Ag	Cr, Se, Zn, Cu, Ni, Hg, Pb, Ba, Be, Ag

Notes:

Se =	Selenium	Cd =	Cadmium
Zn =	Zinc	Ba =	Barium
Cu =	Copper	Pb =	Lead
Ni =	Nickel	Fe =	Iron
Hg =	Mercury	Cr =	Chromium
Ag =	Silver	Be =	Beryllium
As =	Arsenic	Zn =	Zinc

Source: Swartzbaugh, et al. "Remediating Sites Contaminated with Heavy Metals." Hazardous Materials Control, November/December 1992.

TABLE 3-3

POTENTIAL EXPOSURE PATHWAY SUMMARY AND SELECTION RATIONALE FOR AREA A AND THE RESIDENTIAL AREA, CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Exposure Route	Selected Pathway for Evaluation?	Reason for Selection or Exclusion
Brig Employees	Ingestion of on-site groundwater	No	Wells on site are not used as a drinking water source; potable water is supplied by the municipal water authority.
	Inhalation with volatile constituents while showering	No	Wells on site are not used as a drinking water source; potable water is supplied by the municipal water authority.
	Dermal contact with groundwater while showering/bathing	No	Wells on site are not used as a drinking water source; potable water is supplied by the municipal water authority.
	Ingestion of and dermal contact with surface soil while working on site	Yes	Contaminated soil is in an area accessible to employees during routine maintenance duties.
	Inhalation of fugitive dusts from the surface soil while working on site	Yes	Area is grass covered, limiting the potential for dust emission; however, fugitive dusts will be evaluated for conservatism.
	Ingestion of and dermal contact with surface water while working on site	Yes	Surface water is accessible to Brig employees during routine maintenance activities.
	Ingestion of and dermal contact with shallow sediments while working on site	Yes	Sediment is accessible to Brig employees during routine maintenance activities.
	Inhalation of volatile constituents in indoor and outdoor air	Yes	VOCs were detected in indoor and outdoor air samples.

TABLE 3-3 (Continued)

POTENTIAL EXPOSURE PATHWAY SUMMARY AND SELECTION RATIONALE FOR AREA A AND THE RESIDENTIAL AREA, CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Exposure Route	Selected Pathway for Evaluation?	Reason for Selection or Exclusion
Brig Prisoners	Ingestion of on-site groundwater	No	Wells on site are not used as a drinking water source; potable water is supplied by the municipal water authority.
	Inhalation of volatile constituents while showering	No	Wells on site are not used as a drinking water source; potable water is supplied by the municipal water authority.
	Dermal contact with groundwater during showering/bathing	No	Wells on site are not used as a drinking water source; potable water is supplied by the municipal water authority.
	Ingestion of and dermal contact with on-site surface soil	Yes	Soils are accessible to Brig awardees on a limited basis.
	Inhalation of fugitive dusts from the on-site surface soil	Yes	Area is grass covered, limiting the potential for dust emission; however, fugitive dusts will be evaluated for conservatism.
	Ingestion of and dermal contact with surface water and shallow sediments	No	Surface water and sediments are not accessible to Brig awardees or prisoners.
	Inhalation of volatile constituents in indoor and outdoor air	Yes	VOCs were detected in indoor and outdoor air samples.

TABLE 3-3 (Continued)

POTENTIAL EXPOSURE PATHWAY SUMMARY AND SELECTION RATIONALE FOR AREA A AND THE RESIDENTIAL AREA, CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Exposure Route	Selected Pathway for Evaluation?	Reason for Selection or Exclusion
Local Children	Ingestion of Residential Area groundwater	Yes	Area A groundwater can be used for nonpotable purposes such as washing cars, watering lawns, and swimming pools.
	Dermal contact with Residential Area groundwater	Yes	Area groundwater can be used for nonpotable purposes such as washing cars, watering lawns, and swimming pools.
	Inhalation of volatilized organics from nonpotable water	No	Area groundwater can be used for nonpotable purposes such as washing cars, watering lawns, and swimming pools. However, trace levels of VOCs would undergo infinite dilution in outdoor air making this pathway insignificant.
	Ingestion of and dermal contact with on-site surface soils	No	Access to on-site soils is restricted in the Brig Area for obvious reasons.
	Ingestion of and dermal contact with surface waters and shallow sediments	Yes	Older children (ages 6 to 15) could access surface waters and sediments in Area A on a limited basis.
	Inhalation of fugitive dusts emanating from on-site surface soils	No	Access to on-site soils is restricted in the Brig Area for obvious reasons. Residential area is not situated downwind of the site.
	Inhalation of outdoor air	Yes	VOCs were detected in outdoor ambient air samples.

TABLE 3-3 (Continued)

POTENTIAL EXPOSURE PATHWAY SUMMARY AND SELECTION RATIONALE FOR AREA A AND THE RESIDENTIAL AREA, CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Exposure Route	Selected Pathway for Evaluation?	Reason for Selection or Exclusion
Local Adults	Ingestion of Residential Area groundwater	Yes	Area A groundwater can be used for nonpotable purposes such as watering lawns, washing cars, and swimming pools.
	Dermal contact with Residential Area groundwater	Yes	Area A groundwater can be used for nonpotable purposes such as watering lawns, washing cars, and swimming pools.
	Inhalation of volatilized organics from Residential Area groundwater	No	Area groundwater can be used for nonpotable purposes such as washing cars, watering lawns, and swimming pools. However, trace levels of VOCs would undergo infinite dilution in outdoor air making this pathway insignificant.
	Ingestion of and dermal contact with on-site surface soils	No	Access to on-site soils is restricted in the Brig Area for obvious reasons.
	Ingestion of and dermal contact with surface water and shallow sediments	Yes	Adults could access surface waters and sediments in Area A on a limited basis.
	Inhalation of fugitive dusts emanating from on-site surface soils	No	Access to on-site soils is restricted in the Brig Area for obvious reasons. Residential Area is not selected downwind of the site.
	Inhalation of outdoor air	Yes	VOCs were detected in outdoor ambient air samples.

TABLE 3-3 (Continued)

POTENTIAL EXPOSURE PATHWAY SUMMARY AND SELECTION RATIONALE FOR AREA A AND THE RESIDENTIAL AREA, CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Exposure Route	Selected Pathway for Evaluation?	Reason for Selection or Exclusion
Future Resident Children	Ingestion of on-site groundwater	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Inhalation of volatile constituents while showering	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Dermal contact with groundwater while showering/bathing	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Ingestion of and dermal contact with on-site surface soils	Yes	Future residential children could contact soils during outdoor activities.
	Inhalation of fugitive dusts emanating from on-site surface soils	Yes	Future residential children could inhale fugitive dust during outdoor activities.
	Ingestion of and dermal contact with surface waters and sediments	Yes	Residential children could contact surface waters and sediments during outdoor activities.
	Inhalation of outdoor air	Yes	VOCs were detected in outdoor ambient air samples.

TABLE 3-3 (Continued)

POTENTIAL EXPOSURE PATHWAY SUMMARY AND SELECTION RATIONALE FOR AREA A AND THE RESIDENTIAL AREA, CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Exposure Route	Selected Pathway for Evaluation?	Reason for Selection or Exclusion
Future Resident Adult	Ingestion of on-site groundwater	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Inhalation of volatile constituents while showering	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Dermal contact with groundwater while showering/bathing	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Ingestion of and dermal contact with on-site soils	Yes	Future residential adults could contact soils during various outdoor and gardening activities.
	Inhalation of fugitive dusts emanating from on-site soils	Yes	Future residential adults could contact soils during various outdoor and gardening activities.
	Ingestion of and dermal contact with surface waters and sediments	Yes	Residential adults could access surface waters and sediments during outdoor activities.
	Inhalation of outdoor air	Yes	VOCs were detected in outdoor ambient air samples.

TABLE 3-3 (Continued)

POTENTIAL EXPOSURE PATHWAY SUMMARY AND SELECTION RATIONALE FOR AREA A AND THE RESIDENTIAL AREA, CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Exposure Route	Selected Pathway for Evaluation?	Reason for Selection or Exclusion
Future Construction Workers	Ingestion and dermal contact with on-site groundwater	No	Excavation and construction activities assumed to occur above water table.
	Ingestion and dermal contact with on-site surface soils	No	Most direct contact exposures will occur with excavated subsurface soils.
	Inhalation of fugitive dusts emanating from on-site surface soils	No	Most direct contact exposures will occur with excavated subsurface soils.
	Ingestion and dermal contact with on-site subsurface soil	Yes	Most direct contact exposures will occur with excavated subsurface soils.
	Inhalation of fugitive dusts emanating from on-site subsurface soils	Yes	Most direct contact exposures will occur with excavated subsurface soils.

TABLE 3-4

POTENTIAL EXPOSURE PATHWAY SUMMARY AND SELECTION RATIONALE
AREA B POND, CAMP ALLEN LANDFILL
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Exposure Route	Selected Pathway for Evaluation?	Reason for Selection or Exclusion
Adult Workers	Ingestion of on-site groundwater	No	Employees and Brig prisoners/awardees are not expected to contact groundwater during maintenance activities.
	Inhalation of volatile constituents while showering	No	Employees and Brig prisoners/awardees are not expected to contact groundwater during maintenance activities.
	Dermal contact with on-site groundwater while showering or bathing	No	Employees and Brig prisoners/awardees are not expected to contact groundwater during maintenance activities.
	Ingestion of and dermal contact with surface soil while working on site	Yes	Contaminated soils in area are accessible to employees and Brig prisoners/awardees during routine maintenance activities.
	Inhalation of fugitive dusts emanating from surface soils while working on site	Yes	Area is grass-covered, limiting the potential for dust emission; however, fugitive dusts will be evaluated for conservatism.
	Ingestion of and dermal contact with surface water while working on site	Yes	Surface water is accessible to employees and Brig prisoners/ awardees performing routine maintenance activities.
	Ingestion of and dermal contact with shallow sediment while working on site	Yes	Shallow sediments are accessible to employees and Brig prisoners/awardees performing routine maintenance activities.
	Inhalation of volatile constituents in outdoor air	Yes	VOCs were detected in outdoor air samples.

TABLE 3-4 (Continued)

POTENTIAL EXPOSURE PATHWAY SUMMARY AND SELECTION RATIONALE
AREA B POND, CAMP ALLEN LANDFILL
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Exposure Route	Selected Pathway for Evaluation?	Reason for Selection or Exclusion
Local Adults and Children	Ingestion of on-site groundwater	No	On-site groundwater is not used as a drinking water source. Potable water is supplied by the municipal water authority.
	Inhalation of volatile constituents while showering in residences	No	On-site groundwater is not used as a drinking water source. Potable water is supplied by the municipal water authority.
	Dermal contact with groundwater during showering/bathing	No	On-site groundwater is not used as a drinking water source. Potable water is supplied by the municipal water authority.
	Ingestion of and dermal contact with on-site surface soils while trespassing	No	Contaminated soil in Area B Pond is not accessible to local residents.
	Inhalation of fugitive dusts emanating from on-site surface soils	No	Soil is grass covered, limiting the potential for dust emissions.
	Ingestion of and dermal contact with surface waters and shallow sediments	No	Surface waters and sediments in Area B Pond are not accessible to local residents.
	Outdoor air inhalation of volatile constituents	No	Trace levels of outdoor air VOCs detected on site would be infinitely diluted before reaching residential areas southeast of Area B Pond.
Future Resident Children	Ingestion of on-site groundwater	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Inhalation of volatile constituents while showering	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Dermal contact with groundwater while showering/bathing	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Ingestion of and dermal contact with on-site surface soils	Yes	Future residential children could contact soils during recreational activities.
	Inhalation of fugitive dusts emanating from on-site surface soils	Yes	Residential development would require landscaping and lawn care which would limit the potential for dust emissions. However, fugitive dusts will be evaluated for conservatism.
	Ingestion of and dermal contact with surface waters and sediments	Yes	Residential children could contact surface waters and sediments during recreational activities.
	Inhalation of outdoor air	Yes	VOCs were detected in outdoor ambient air samples.

TABLE 3-4 (Continued)

POTENTIAL EXPOSURE PATHWAY SUMMARY AND SELECTION RATIONALE
AREA B POND, CAMP ALLEN LANDFILL
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Exposure Route	Selected Pathway for Evaluation?	Reason for Selection or Exclusion
Future Resident Adult	Ingestion of on-site groundwater	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Inhalation of volatile constituents while showering	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Dermal contact with groundwater while showering/bathing	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Ingestion of and dermal contact with on-site surface soils	Yes	Future residential adults could contact soils during maintenance and gardening activities.
	Inhalation of fugitive dusts emanating from on-site surface soils	Yes	Future residential adults could contact soils during maintenance and gardening activities.
	Ingestion of and dermal contact with surface waters and sediments	Yes	Ditches are not sufficiently large enough for future resident adult recreational activities.
	Inhalation of outdoor air	Yes	VOCs were detected in outdoor ambient air samples.
Future Construction Workers(1)	Ingestion and dermal contact with on-site groundwater	No	Excavation and construction activities assumed to occur above water table.
	Ingestion and dermal contact with on-site surface soils	No	Most direct contact exposures will be with excavated subsurface soils.
	Inhalation of fugitive dusts emanating from on-site surface soils	No	Most direct contact exposures will be with excavated subsurface soils.
	Ingestion and dermal contact with on-site subsurface soil	Yes	Most direct contact exposures will be with excavated subsurface soils.
	Inhalation of fugitive dusts emanating from on-site subsurface soils	Yes	Most direct contact exposures will be with excavated subsurface soils.

(1) Evaluated for all of Area B.

TABLE 3-5

POTENTIAL EXPOSURE PATHWAY SUMMARY AND SELECTION RATIONALE
AREA B SCHOOL, CAMP ALLEN LANDFILL
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Exposure Route	Selected Pathway for Evaluation?	Reason for Selection or Exclusion
Adult Employees and Children in Elementary School	Ingestion of on-site groundwater	No	On-site groundwater is not used as a drinking water source. Potable water is supplied by the municipal water authority.
	Inhalation of volatile constituents while showering in residences	No	On-site groundwater is not used as a drinking water source. Potable water is supplied by the municipal water authority.
	Dermal contact with groundwater during showering/bathing	No	On-site groundwater is not used as a drinking water source. Potable water is supplied by the municipal water authority.
	Ingestion of and dermal contact with on-site surface soils	Yes	Contaminated soil is accessible to employees and school children.
	Inhalation of fugitive dusts emanating from on-site surface soils	Yes	Soil is grass covered, limiting the potential for dust emissions; however, fugitive dusts will be evaluated for conservatism.
	Ingestion of and dermal contact with surface waters and shallow sediments	Yes	Surface waters and sediments are accessible to employees and school children in Area B School.
	Indoor and outdoor air inhalation of volatile constituents	Yes	VOCs were detected in indoor and outdoor air samples.
Future Resident Children	Ingestion of on-site groundwater	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Inhalation of volatile constituents while showering	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Dermal contact with groundwater while showering/bathing	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Ingestion of and dermal contact with on-site surface soils	Yes	Future residential children could contact soils during recreational activities.
	Inhalation of fugitive dusts emanating from on-site surface soils	Yes	Residential development would require landscaping and lawn care which would limit the potential for dust emissions. However, fugitive dusts will be evaluated for conservatism.
	Ingestion of and dermal contact with surface waters and sediments	Yes	Residential children could contact surface waters and sediments during recreational activities.
	Inhalation of outdoor air	Yes	VOCs were detected in outdoor ambient air samples.

TABLE 3-5 (Continued)

POTENTIAL EXPOSURE PATHWAY SUMMARY AND SELECTION RATIONALE
AREA B SCHOOL, CAMP ALLEN LANDFILL
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Exposure Route	Selected Pathway for Evaluation?	Reason for Selection or Exclusion
Future Resident Adult	Ingestion of on-site groundwater	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Inhalation of volatile constituents while showering	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Dermal contact with groundwater while showering/bathing	Yes	Although use of groundwater is highly unlikely, groundwater could be used as a potable source in the future.
	Ingestion of and dermal contact with on-site surface soils	Yes	Future residential adults could contact soils during maintenance and gardening activities.
	Inhalation of fugitive dusts emanating from on-site surface soils	Yes	Residential development would require landscaping and lawn care which would limit the potential for dust emissions. However, fugitive dusts will be evaluated for conservatism.
	Ingestion of and dermal contact with surface waters and sediments	Yes	Ditches are not sufficiently large enough for future resident adult recreational activities.
	Inhalation of outdoor air	Yes	VOCs were detected in outdoor ambient air samples.
Future Construction Workers(1)	Ingestion and dermal contact with on-site groundwater	No	Excavation and construction activities assumed to occur above water table.
	Ingestion and dermal contact with on-site surface soils	No	Most direct contact exposures will occur with excavated subsurface soils.
	Inhalation of fugitive dusts emanating from on-site surface soils	No	Most direct contact exposures will occur with excavated subsurface soils.
	Ingestion and dermal contact with on-site subsurface soil	Yes	Most direct contact exposures will occur with excavated subsurface soils.
	Inhalation of fugitive dusts emanating from on-site subsurface soils	Yes	Most direct contact exposures will occur with excavated subsurface soils.

(1) Evaluated for all of Area B.

TABLE 3-6

EXPOSURE INPUT PARAMETERS FOR LOCAL ADULTS AND CHILDREN POTENTIALLY EXPOSED TO COPCs IN THE AREA A AND THE RESIDENTIAL AREA ,CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Input Parameter	Media	Units	Current Receptor		Comments/References
			Child[1]	Adult	
ED, Exposure Duration	Sediment	year	9	30	Professional Judgment and USEPA, 1991
	Groundwater	year	6	30	USEPA, 1991
	Surface Water	year	9	30	Professional Judgment and USEPA, 1991
	Outdoor Air	year	9	30	Professional Judgment and USEPA, 1991
EF, Exposure Frequency	Sediment	day/year	28	7	Professional Judgment
	Groundwater	day/year	28	28	Professional Judgment
	Surface Water	day/year	28	7	Professional Judgment
	Outdoor Air	day/year	150	150	Professional Judgment
ET, Exposure Time	Surface Water	hr/day	2.6	2.6	USEPA, 1989
	Groundwater	hr/day	2.6	2.6	USEPA, 1989
	Outdoor Air	hr/day	2	1	Professional Judgment
IR, Ingestion Rate	Sediment	mg/day	100	50	USEPA, 1991
	Groundwater	L/day	1	2	USEPA, 1989
	Surface Water	L/hr	0.05	0.05	USEPA, 1989
A, Surface Area	Sediment	cm^2	3,683	5,300	USEPA, 1992
	Groundwater	cm^2	8,023	20,000	USEPA, 1992
	Surface Water	cm^2	3,683	5,300	USEPA, 1992
AT, Averaging Time AT_{nc}, noncarcinogenic	Sediment	day	3,285	10,950	USEPA, 1989
	Groundwater	day	3,285	10,950	USEPA, 1989
	Surface Water	day	3,285	10,950	USEPA, 1989
	Outdoor Air	day	3,285	10,950	USEPA, 1989
AT_c, carcinogenic	All Media	day	25,550	25,550	USEPA, 1989
ABS, Absorbance Factor	Soil/Sediment	NA	Chemical-specific[2]	Chemical-specific[2]	USEPA, 1992 Ryan, et al., 1987 Wester, et al., 1993
AF, Adherence Factor	Soil/Sediment	mg/cm^2	1	1	USEPA, 1992
BW, Body Weight	All Media	Kg	[3]	70	USEPA, 1989

TABLE 3-6 (Continued)

EXPOSURE INPUT PARAMETERS FOR
LOCAL ADULTS AND CHILDREN POTENTIALLY EXPOSED TO COPCs IN THE
AREA A AND THE RESIDENTIAL AREA ,CAMP ALLEN LANDFILL
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Input Parameter	Media	Units	Current Receptor		Comments/References
			Child[1]	Adult	
Kp, Permeability Constant	All Media	cm/hr	Chemical-specific	Chemical-specific	see Table B-5
RR, Respiration Rate	Air	m^3/hr	0.83	0.83	USEPA, 1989a

Notes: [1] Older child (ages 7 to 15 years old) was evaluated for potential exposures to all media, except groundwater. Young child (ages 1 to 6 years old) was evaluated for nonpotable exposure to groundwater (e.g., swimming).

[2] The following absorbance factors will be applied to estimate chronic daily intakes due to dermal exposures to chemical in soils:

Experimentally Derived (USEPA, 1992): PCBs - 0.06
Cadmium - 0.01

Default Values (Ryan, et al., 1987): VOCs - 0.25
SVOCs - 0.10
Pesticides - 0.10
Inorganics - 0.01
Arsenic - 0.03 (Wester, et al., 1993)

[3] Child body weights: 1 to 6 years old = 15 kg; 7 to 15 years old = 37 kg.

NA - Not Applicable

References:

Ryan, E.A., Hawkins, E.T., et al. 1987. Assessing Risk from Dermal Exposure at Hazardous Waste Sites. Bennet, G. and Bennet, J. editors. Superfund '87: Proceedings of the 8th National Conference.

USEPA, 1992. Dermal Exposure Assessment: Principles and Applications - Interim Report.

USEPA, 1991. Risk Assessment Guidance for Superfund, Volume I - Human Health Evaluation Manual Supplemental Guidance. "Standard Default Exposure Factors." Interim Final.

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TABLE 3-7

EXPOSURE INPUT PARAMETERS FOR BRIG PRISONERS AND EMPLOYEES POTENTIALLY EXPOSED TO COPCs IN AREA A, CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Input Parameter	Media	Units	Current Receptor		Comments/References
			Brig Prisoner	Brig Employee	
ED, Exposure Duration	Sediment	year	NA	25	Site-specific values
	Surface Water	year	NA	25	Site-specific values
	Soil	year	2	25	Site-specific values
	Outdoor Air	year	2	25	Site-specific values
	Indoor Air	year	2	25	Site-specific values
EF, Exposure Frequency	Sediment	day/year	NA	12	Professional Judgment and Site-specific values
	Surface Water	day/year	NA	12	Professional Judgment and Site-specific values
	Soil	day/year	350	250	USEPA, 1991
	Outdoor Air	day/year	350	250	USEPA, 1991
	Indoor Air	day/year	350	250	USEPA, 1991
ET, Exposure Time	Surface Water	hr/day	NA	2.6	Professional Judgment
	Outdoor Air	hr/day	2	10	Professional Judgment and Site-specific values
	Indoor Air	hr/day	22	10	Professional Judgment and Site-specific values
IR, Ingestion Rate	Sediment	mg/day	NA	50	USEPA, 1991
	Surface Water	L/hr	NA	0.05	USEPA, 1989
	Soil	mg/day	50	50	USEPA, 1989
A, Surface Area	Sediment	cm^2	NA	4100	USEPA, 1989a
	Surface Water	cm^2	NA	4100	USEPA, 1989a
	Soil	cm^2	4,100	4100	USEPA, 1989a
AT, Averaging Time AT_{nc}, noncarcinogenic	Sediment	day	NA	9,125	USEPA, 1989
	Surface Water	day	NA	9,125	USEPA, 1989
	Soil	day	730	9,125	USEPA, 1989
	Outdoor Air	day	730	9,125	USEPA, 1989
	Indoor Air	day	730	9,125	USEPA, 1989
AT_c, carcinogenic	All Media	day	25,550	25,550	USEPA, 1989

TABLE 3-7 (Continued)

EXPOSURE INPUT PARAMETERS FOR
BRIG AWARDEES AND EMPLOYEES POTENTIALLY EXPOSED TO
COPCs IN AREA A, CAMP ALLEN LANDFILL
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Input Parameter	Media	Units	Current Receptor		Comments/References
			Brig Prisoner	Brig Employee	
ABS, Absorbance Factor	Soil/Sediment	NA	Chemical-specific[1]	Chemical-specific[1]	USEPA, 1992 Ryan, et al., 1987 Wester, et al., 1993
AF, Adherence Factor	Soil/Sediment	mg/cm^2	1	1	USEPA, 1992a
BW, Body Weight	All Media	Kg	70	70	USEPA, 1989
Kp, Permeability Constant	Surface Water	cm/hr	NA	Chemical-specific	See Table B-5
RR, Respiration Rate	Air	m^3/hr	0.83	0.83	USEPA, 1989a

Notes: [1] The following absorbance factors will be applied to estimate chronic daily intakes due to dermal exposures to chemical in soils:

Experimentally Derived (USEPA, 1992): PCBs - 0.06
Cadmium - 0.01

Default Values (Ryan, et al., 1987): VOCs - 0.25
SVOCs - 0.10
Pesticides - 0.10
Inorganics - 0.01
Arsenic - 0.03 (Wester, et al., 1993)

NA - Not Applicable

References:

Ryan, E.A., Hawkins, E.T., et al. 1987. Assessing Risk from Dermal Exposure at Hazardous Waste Sites. Bennet, G. and Bennet, J. editors. Superfund '87: Proceedings of the 8th National Conference.

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Wester, R.C., Maibach, H.I., et al. 1993. *In Vivo and In Vitro* Percutaneous Absorption and Skin Decontamination of Arsenic from Water and Soil. Fundamental and Applied Toxicology 20, pp. 336-340.

TABLE 3-8

EXPOSURE INPUT PARAMETERS FOR FUTURE RESIDENT CHILDREN AND ADULTS POTENTIALLY EXPOSED TO COPCs IN AREA A , CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Input Parameter	Media	Units	Future Receptor		Comments/References
			Child (1 to 6 years)	Adult	
ED, Exposure Duration	Sediment	year	6	30	USEPA, 1991
	Groundwater	year	6	30	USEPA, 1991
	Surface Water	year	6	30	USEPA, 1991
	Soil	year	6	30	USEPA, 1991
	Air, Shower	year	6	30	USEPA, 1991
	Outdoor Air	year	6	30	USEPA, 1991
	Indoor Air	year	6	30	USEPA, 1991
EF, Exposure Frequency	Sediment	day/year	137	34	Professional Judgment
	Groundwater	day/year	350	350	USEPA, 1991
	Surface Water	day/year	137	34	Professional Judgment
	Soil	day/year	350	350	USEPA, 1991
	Air, Shower	day/year	350	350	USEPA, 1991
	Outdoor Air	day/year	150	150	6 mos. fair weather
	Indoor Air	day/year	350	350	USEPA, 1991
ET, Exposure Time	Surface Water	hr/day	2.5	2.5	Professional Judgment
	Air, Shower	hr/day	0.2	0.2	USEPA, 1991
	Groundwater	hr/day	0.2	0.2	USEPA, 1991
	Outdoor Air	hr/day	2	1	Professional Judgment
	Indoor Air	hr/day	14(1)	13(2)	Professional Judgment
IR, Ingestion Rate	Sediment	mg/day	200	100	USEPA, 1989
	Groundwater	L/day	1	2	USEPA, 1991
	Surface Water	L/hr	0.05	0.05	USEPA, 1989
	Soil	mg/day	200	100	USEPA, 1989
A, Surface Area	Sediment	cm^2	2,000	5,300	USEPA, 1992
	Groundwater	cm^2	8,023	20,000	USEPA, 1992
	Surface Water	cm^2	2,000	5,300	USEPA, 1992
	Soil	cm^2	2,006	5,300	USEPA, 1992
AT, Averaging Time					
AT_{nc}, noncarcinogenic	Sediment	day	2,190	10,950	USEPA, 1989
	Groundwater	day	2,190	10,950	USEPA, 1989
	Surface Water	day	2,190	10,950	USEPA, 1989
	Soil	day	2,190	10,950	USEPA, 1989
	Air, Shower	day	2,190	10,950	USEPA, 1989
	Outdoor Air	day	2,190	10,950	USEPA, 1989
	Indoor Air	day	2,190	10,950	USEPA, 1989
AT_c, carcinogenic	All Media	day	25,550	25,550	USEPA, 1989a

TABLE 3-8 (Continued)

EXPOSURE INPUT PARAMETERS FOR FUTURE RESIDENT CHILDREN AND ADULTS POTENTIALLY EXPOSED TO COPCs IN AREA A , CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Input Parameter	Media	Units	Future Receptor		Comments/References
			Child (1 to 6 years)	Adult	
ABS, Absorbance Factor	Soil/Sediment	NA	Chemical-specific(3)	Chemical-specific(3)	USEPA, 1992 Ryan, et al., 1987 Wester, et al., 1993
AF, Adherence Factor	Soil/Sediment	mg/cm^2	1	1	USEPA, 1992
BW, Body Weight	All Media	Kg	(4)	70	USEPA, 1989
Kp, Permeability Constant	Groundwater/ Surface Water	cm/hr	Chemical-specific	Chemical-specific	See Table B-5
RR, Respiration Rate	Air	m^3/hr	0.6	0.83	USEPA, 1989, 1989a

Notes: (1) 24 hrs (1 day) - 8 hrs (school) - 2 hrs (outdoors) = 14 hrs (during school year)

(2) 24 hrs (1 day) - 10 hrs (work) - 1 hr (outdoors) = 13 hrs

(3) The following absorbance factors will be applied to estimate chronic daily intakes due to dermal exposures to chemical in soils:

Experimentally Derived (USEPA, 1992): PCBs - 0.06
Cadmium - 0.01

Default Values (Ryan, et al., 1987): VOCs - 0.25
SVOCs - 0.10
Pesticides - 0.10
Inorganics - 0.01
Arsenic - 0.03 (Wester, et al., 1993)

(4) Child body weights: 1 to 6 years = 15 kg; 7 to 15 years = 37 kg

NA = Not Applicable

References:

Ryan, E.A., Hawkins, E.T., et al. 1987. Assessing Risk from Dermal Exposure at Hazardous Waste Sites. Bennet, G. and Bennet, J. editors. Superfund '87: Proceedings of the 8th National Conference.

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Wester, R.C., Maibach, H.I., et al. 1993. *In Vivo and In Vitro* Percutaneous Absorption and Skin Decontamination of Arsenic from Water and Soil. Fundamental and Applied Toxicology 20, pp. 336-340.

TABLE 3-9

EXPOSURE INPUT PARAMETERS FOR
ADULT WORKERS POTENTIALLY EXPOSED TO COPCs IN AREA B - POND
CAMP ALLEN LANDFILL
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Input Parameter	Media	Units	Adult Employee	Comments/References
ED, Exposure Duration	Sediment	year	25	USEPA, 1991
	Soil	year	25	USEPA, 1991
	Surface Water	year	25	USEPA, 1991
	Outdoor Air	year	25	USEPA, 1991
	Indoor Air	year	25	USEPA, 1991
EF, Exposure Frequency	Sediment	day/year	12	Professional Judgment
	Soil	day/year	250	USEPA, 1991
	Surface Water	day/year	12	Professional Judgment and USEPA, 1992
	Outdoor Air	day/year	250	USEPA, 1991
	Indoor Air	day/year	250	USEPA, 1991
ET, Exposure Time	Surface Water	hr/day	2.6	USEPA, 1989
	Outdoor Air	hr/day	4	Professional Judgment
	Indoor Air	hr/day	4	Professional Judgment
IR, Ingestion Rate	Sediment	mg/day	50	USEPA, 1991
	Soil	mg/day	50	USEPA, 1991
	Surface Water	L/hr	0.05	USEPA, 1991
A, Surface Area	Sediment	cm^2	4,100	USEPA, 1989a
	Soil	cm^2	4,100	USEPA, 1989a
	Surface Water	cm^2	4,100	USEPA, 1989a
AT, Averaging Time AT_{nc}, noncarcinogenic	Sediment	day	9,125	USEPA, 1989
	Soil	day	9,125	USEPA, 1989
	Surface Water	day	9,125	USEPA, 1989
	Outdoor Air	day	9,125	USEPA, 1989
	Indoor Air	day	9,125	USEPA, 1989
AT_c, carcinogenic	All Media	day	25,550	USEPA, 1989
ABS, Absorbance Factor	Soil/Sediment	NA	Chemical-specific(1)	USEPA, 1992 Ryan, et al., 1987 Wester, et al., 1993

TABLE 3-9 (Continued)

EXPOSURE INPUT PARAMETERS FOR
ADULT WORKERS POTENTIALLY EXPOSED TO COPCs IN AREA B - POND
CAMP ALLEN LANDFILL
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Input Parameter	Media	Units	Adult Employee	Comments/References
AF, Adherence Factor	Soil/Sediment	mg/cm^2	1	USEPA, 1992
BW, Body Weight	All Media	Kg	70	USEPA, 1989
Kp, Permeability Constant	All Media	cm/hr	Chemical-specific	See Table B-5
RR, Respiration Rate	Air	m^3/hr	0.83	USEPA, 1989a

Notes: (1) The following absorbance factors will be applied to estimate chronic daily intakes due to dermal exposures to chemical in soils:

Experimentally Derived (USEPA, 1992): PCBs - 0.06
Cadmium - 0.01

Default Values (Ryan, et al., 1987): VOCs - 0.25
SVOCs - 0.10
Pesticides - 0.10
Inorganics - 0.01
Arsenic - 0.03 (Wester, et al., 1993)

NA - Not Applicable

References:

Ryan, E.A., Hawkins, E.T., et al. 1987. Assessing Risk from Dermal Exposure at Hazardous Waste Sites. Bennet, G. and Bennet, J. editors. Superfund '87: Proceedings of the 8th National Conference.

USEPA, 1992. Dermal Exposure Assessment: Principles and Applications - Interim Report.

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USEPA, 1989. Risk Assessment Guidance for Superfund, Volume I - Human Health Evaluation Manual (Part A) Interim Final.

USEPA, 1989a. Exposure Factors Handbook.

Wester, R.C., Maibach, H.I., et al. 1993. *In Vivo and In Vitro* Percutaneous Absorption and Skin Decontamination of Arsenic from Water and Soil. Fundamental and Applied Toxicology 20, pp. 336-340.

TABLE 3-10

EXPOSURE INPUT PARAMETERS FOR
SCHOOL CHILDREN AND ADULT EMPLOYEES POTENTIALLY EXPOSED TO
COPCs IN AREA B - ELEMENTARY SCHOOL
CAMP ALLEN LANDFILL
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Input Parameter	Media	Units	Current Receptor		Comments/References
			Child (7 to 12 years)	Adult	
ED, Exposure Duration	Sediment	year	6	25	USEPA, 1991
	Surface Water	year	6	25	USEPA, 1991
	Soil	year	6	25	USEPA, 1991
	Outdoor Air	year	6	25	USEPA, 1991
	Indoor Air	year	6	25	USEPA, 1991
EF, Exposure Frequency	Sediment	day/year	137	12	Professional Judgment
	Surface Water	day/year	137	12	Professional Judgment
	Soil	day/year	180	250	USEPA, 1991
	Outdoor Air	day/year	180	250	USEPA, 1991
	Indoor Air	day/year	180	250	USEPA, 1991
ET, Exposure Time	Surface Water	hr/day	2.6	2.6	Professional Judgment
	Outdoor Air	hr/day	1	1	Professional Judgment
	Indoor Air	hr/day	7	9	Professional Judgment
IR, Ingestion Rate	Sediment	mg/day	100	50	USEPA, 1991
	Surface Water	L/hr	0.05	0.05	USEPA, 1989
	Soil	mg/day	200	50	USEPA, 1991
A, Surface Area	Sediment	cm^2	3,242	4,100	USEPA, 1989a
	Surface Water	cm^2	3,242	4,100	USEPA, 1989a
	Soil	cm^2	3,242	4,100	USEPA, 1989a
AT, Averaging Time AT_{nc}, noncarcinogenic	Sediment	day	2,190	9,125	USEPA, 1989
	Surface Water	day	2,190	9,125	USEPA, 1989
	Soil	day	2,190	9,125	USEPA, 1989
	Outdoor Air	day	2,190	9,125	USEPA, 1989
	Indoor Air	day	2,190	9,125	USEPA, 1989
AT_c, carcinogenic	All Media	day	25,550	25,550	USEPA, 1989
ABS, Absorbance Factor	Soil/Sediment	NA	Chemical-specific(1)	Chemical-specific(1)	USEPA, 1992 Ryan, et al., 1987 Wester, et al., 1993
AF, Adherence Factor	Soil/Sediment	mg/cm^2	1	1	USEPA, 1992
BW, Body Weight	All Media	Kg	31	70	USEPA, 1989, 1989a
Kp, Permeability Constant	All Media	cm/hr	Chemical-specific	Chemical-specific	See Table B-5

TABLE 3-10 (Continued)

EXPOSURE INPUT PARAMETERS FOR
SCHOOL CHILDREN AND ADULT EMPLOYEES POTENTIALLY EXPOSED TO
COPCs IN AREA B - ELEMENTARY SCHOOL
CAMP ALLEN LANDFILL
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Notes: (1) The following absorbance factors will be applied to estimate chronic daily intakes due to dermal exposures to chemical in soils:

Experimentally Derived (USEPA, 1992):	PCBs - 0.06 Cadmium - 0.01
Default Values (Ryan, et al., 1987):	VOCs - 0.25 SVOCs - 0.10 Pesticides - 0.10 Inorganics - 0.01 Arsenic - 0.03 (Wester, et al., 1993)

NA - Not Applicable

References:

Ryan, E.A., Hawkins, E.T., et al. 1987. Assessing Risk from Dermal Exposure at Hazardous Waste Sites. Bennet, G. and Bennet, J. editors. Superfund '87: Proceedings of the 8th National Conference.

USEPA, 1992. Dermal Exposure Assessment: Principles and Applications - Interim Report.

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USEPA, 1989a. Exposure Factors Handbook.

Wester, R.C., Maibach, H.I., et al. 1993. *In Vivo and In Vitro* Percutaneous Absorption and Skin Decontamination of Arsenic from Water and Soil. Fundamental and Applied Toxicology 20, pp. 336-340.

TABLE 3-11

EXPOSURE INPUT PARAMETERS FOR FUTURE RESIDENT ADULTS AND CHILDREN POTENTIALLY EXPOSED TO COPCs IN AREA B - POND AND ELEMENTARY SCHOOL CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Input Parameter	Media	Units	Future Receptor		Comments/References
			Child (1 to 6 years)	Adult	
ED, Exposure Duration	Sediment	year	6	30	USEPA, 1991
	Groundwater	year	6	30	USEPA, 1991
	Surface Water	year	6	30	USEPA, 1991
	Soil	year	6	30	USEPA, 1991
	Air, Shower	year	6	30	USEPA, 1991
	Outdoor Air	year	6	30	USEPA, 1991
	Indoor Air	year	6	30	USEPA, 1991
EF, Exposure Frequency	Sediment	day/year	137	34	Professional Judgment
	Groundwater	day/year	350	350	USEPA, 1991
	Surface Water	day/year	137	34	Professional Judgment
	Soil	day/year	350	350	USEPA, 1991
	Air, Shower	day/year	350	350	USEPA, 1991
	Groundwater	day/year	350	350	USEPA, 1991
	Outdoor Air	day/year	150	150	6 mos. fair weather
	Indoor Air	day/year	350	350	USEPA, 1991
ET, Exposure Time	Surface Water	hr/day	2.6	2.6	Professional Judgment
	Air, Shower	hr/day	0.2	0.2	USEPA, 1991
	Outdoor Air	hr/day	2	1	Professional Judgment
	Indoor Air	hr/day	14(1)	13(2)	Professional Judgment
IR, Ingestion Rate	Sediment	mg/day	200	100	USEPA, 1991
	Groundwater	L/day	1	2	USEPA, 1991
	Surface Water	L/hr	0.05	0.05	USEPA, 1989
	Soil	mg/day	200	100	USEPA, 1991
A, Surface Area	Sediment	cm^2	2,006	5,300	USEPA, 1992
	Groundwater	cm^2	2,006	20,000	USEPA, 1992
	Surface Water	cm^2	2,006	5,300	USEPA, 1992
	Soil	cm^2	2,006	5,300	USEPA, 1992
AT, Averaging Time AT_{nc}, noncarcinogenic	All Media	day	2,190	10,950	USEPA, 1989
AT_c, carcinogenic	All Media	day	25,550	25,550	USEPA, 1989
ABS, Absorbance Factor	Soil/Sediment	NA	Chemical-specific(3)	Chemical-specific(3)	USEPA, 1992 Ryan, et al., 1987 Wester, et al., 1993
AF, Adherence Factor	Soil/Sediment	mg/cm^2	1	1	USEPA, 1992

TABLE 3-11 (Continued)

EXPOSURE INPUT PARAMETERS FOR
FUTURE RESIDENT ADULTS AND CHILDREN POTENTIALLY EXPOSED TO
COPCs IN AREA B - POND AND ELEMENTARY SCHOOL
CAMP ALLEN LANDFILL
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Input Parameter	Media	Units	Future Receptor		Comments/References
			Child (1 to 6 years)	Adult	
BW, Body Weight	All Media	Kg	15	70	USEPA, 1989
Kp, Permeability Constant	All Media	cm/hr	Chemical-specific	Chemical-specific	See Table B-5
RR, Respiration Rate	Air	m^3/hr	0.6	0.83	USEPA, 1989, 1989a

Notes:
(1) 24 hrs (1 day) - 8 hrs (school) - 2 hrs (outdoors) = 14 hrs (during school year)
(2) 24 hrs (1 day) - 10 hrs (work) - 1 hr (outdoors) = 13 hrs
(3) The following absorbance factors will be applied to estimate chronic daily intakes due to dermal exposures to chemical in soils:

Experimentally Derived (USEPA, 1992): PCBs - 0.06
Cadmium - 0.01

Default Values (Ryan, et al., 1987): VOCs - 0.25
SVOCs - 0.10
Pesticides - 0.10
Inorganics - 0.01
Arsenic - 0.03 (Wester, et al., 1993)

NA = Not Applicable

References:

Ryan, E.A., Hawkins, E.T., et al. 1987. Assessing Risk from Dermal Exposure at Hazardous Waste Sites. Bennet, G. and Bennet, J. editors. Superfund '87: Proceedings of the 8th National Conference.

USEPA, 1992. Dermal Exposure Assessment: Principles and Applications - Interim Report.

USEPA, 1991. Risk Assessment Guidance for Superfund, Volume I - Human Health Evaluation Manual Supplemental Guidance. "Standard Default Exposure Factors." Interim Final.

USEPA, 1989. Risk Assessment Guidance for Superfund, Volume I - Human Health Evaluation Manual (Part A) Interim Final.

USEPA, 1989a. Exposure Factors Handbook.

Wester, R.C., Maibach, H.I., et al. 1993. *In Vivo and In Vitro* Percutaneous Absorption and Skin Decontamination of Arsenic from Water and Soil. Fundamental and Applied Toxicology 20, pp. 336-340.

TABLE 3-12

EXPOSURE INPUT PARAMETERS FOR CHRONIC DAILY INTAKE VIA SUBSURFACE SOIL INGESTION, DERMAL CONTACT, AND INHALATION CONSTRUCTION WORKERS IN AREAS A AND B CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Input Parameter	Units	Adult Construction Worker	Comments/Reference
CF, Conversion Factor	kg/mg	1×10^{-6}	NA
IR, Ingestion Rate	mg/day	480	USEPA, 1991
EF, Exposure Frequency	days/year	250	USEPA, 1991
AF, Adherence Factor	mg/cm^2	1.0	USEPA, 1992 USEPA, 1991
ABS, Dermal Absorption Factor	unitless	Chemical-specific(1)	USEPA, 1992 Ryan, et al., 1987 Wester, et al., 1993
ET, Exposure Time	hrs/day	8	Professional Judgement/USEPA, 1991
RR, Respiration Rate	m^3/hr	0.83	USEPA, 1991
A, Exposed Surface Area	cm^2/day	4,100	USEPA, 1992
ED, Exposure Duration	years	1	USEPA, 1991
BW, Body Weight	kg	70	USEPA, 1989
AT, Averaging Times ATnc, Noncarcinogenic	days	365	USEPA, 1989a
ATc, Carcinogenic	days	25,550	USEPA, 1989a

Notes: (1) The following absorbance factors will be applied to estimate chronic daily intakes due to dermal exposures to chemical in soils:

Experimentally Derived (USEPA, 1992): PCBs - 0.06
Cadmium - 0.01

Default Values (Ryan, et al., 1987): VOCs - 0.25
SVOCs - 0.10
Pesticides - 0.10
Inorganics - 0.01
Arsenic - 0.03 (Wester, et al., 1993)

NA = Not Applicable

TABLE 3-12 (Continued)

EXPOSURE INPUT PARAMETERS FOR CHRONIC DAILY INTAKE VIA SUBSURFACE SOIL INGESTION, DERMAL CONTACT, AND INHALATION CONSTRUCTION WORKERS IN AREAS A AND B CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

References:

Ryan, E.A., Hawkins, E.T., et al. 1987. Assessing Risk from Dermal Exposure at Hazardous Waste Sites. Bennet, G. and Bennet, J. editors. Superfund '87: Proceedings of the 8th National Conference.

USEPA, 1992. Dermal Exposure Assessment: Principles and Applications - Interim Report.

USEPA, 1991. Risk Assessment Guidance for Superfund, Volume I - Human Health Evaluation Manual Supplemental Guidance. "Standard Default Exposure Factors." Interim Final.

USEPA, 1989. Risk Assessment Guidance for Superfund, Volume I - Human Health Evaluation Manual (Part A). Interim Final.

USEPA, 1989a. Exposure Factors Handbook.

Wester, R.C., Maibach, H.I., et al. 1993. *In Vivo and In Vitro* Percutaneous Absorption and Skin Decontamination of Arsenic from Water and Soil. Fundamental and Applied Toxicology 20, pp. 336-340.

SECTION 3.0 FIGURES

FIGURE 3-1
CONCEPTUAL SITE MODEL
CAMP ALLEN LANDFILL - AREA A
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

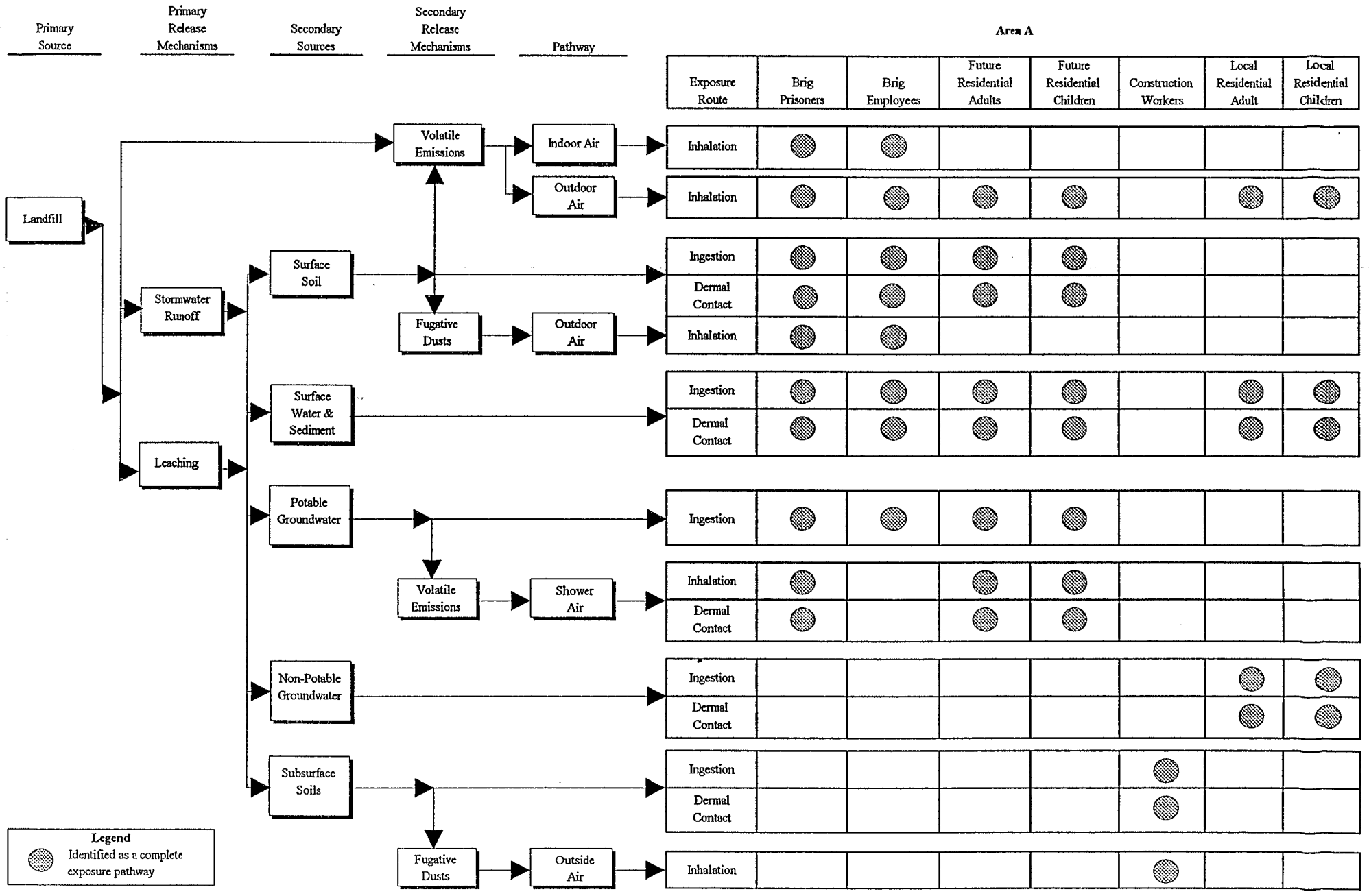
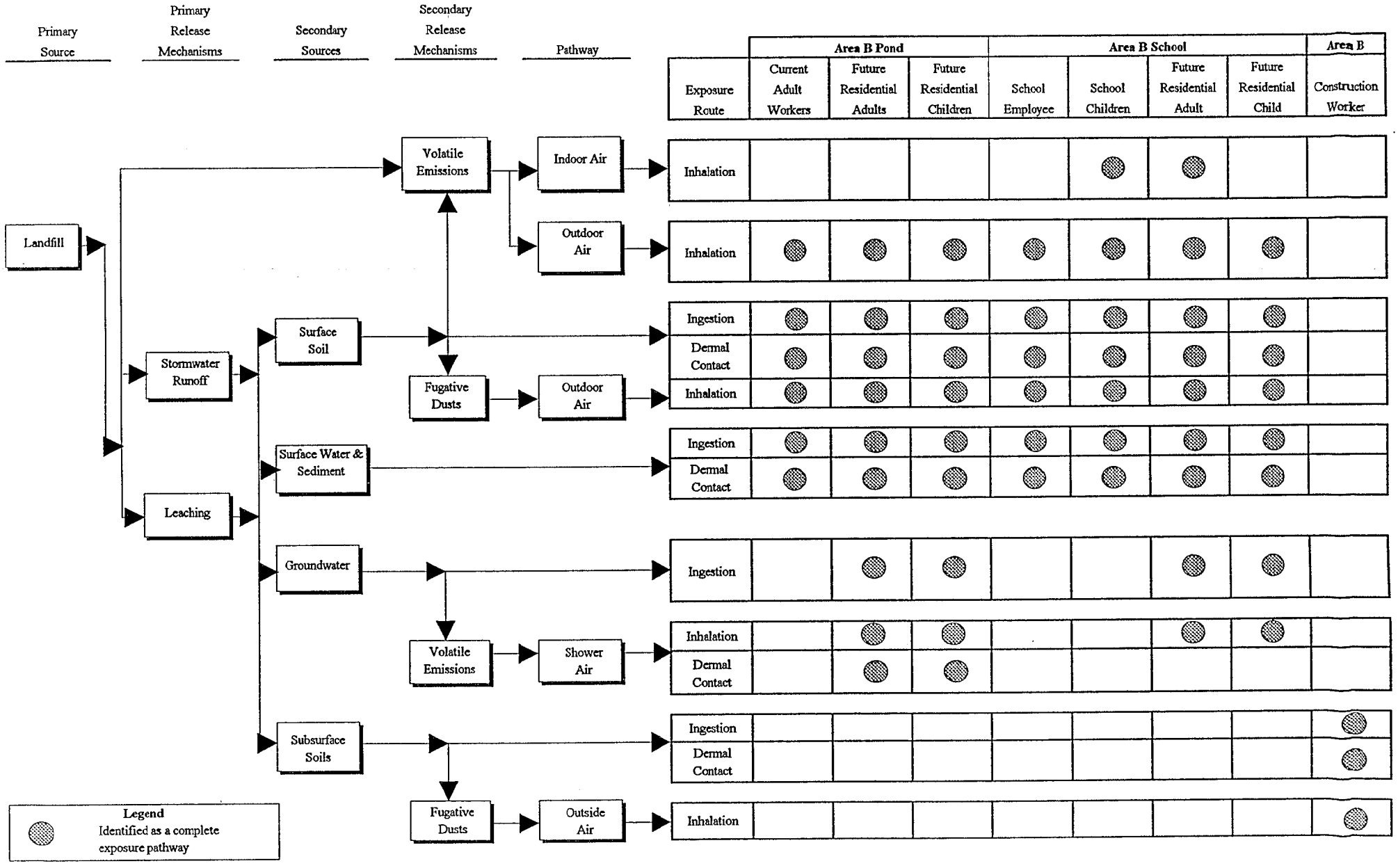


FIGURE 3-2
CONCEPTUAL SITE MODEL
CAMP ALLEN LANDFILL - AREA B, AREA B POND, AREA B SCHOOL
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA



SECTION 4

4.0 TOXICITY ASSESSMENT

Section 3.0 presented potential exposure pathways and receptors for this baseline risk assessment. This section will review the available toxicological information for COPCs retained for quantitative evaluation.

4.1 Toxicological Evaluation

The purpose of this section is to identify the potential health and environmental effects associated with potential exposure to the COPCs. A toxicological evaluation characterizes the inherent toxicity of a compound. It consists of the review of scientific data to determine the nature and extent of the potential human health and environmental effects associated with potential exposure to the various chemicals. The end product is a collection of toxicological profiles for the COPCs. These toxicological profiles provide the qualitative weight-of-evidence that demonstrate whether facility COPCs pose any actual or potential health and environmental effects.

Toxicological profiles addressing the COPCs at the Camp Allen Landfill are presented in Appendix C. In these toxicological profiles, the available human and animal data are presented. Human data from occupational exposures are often insufficient for determining quantitative indices of toxicity because of uncertainties in exposure estimates, and inherent difficulties in determining causal relationships established by epidemiological studies. For this reason, animal bioassays are conducted under controlled conditions and their results are extrapolated to humans. There are several stages to this extrapolation. First, to account for species differences, conversion factors are used to extrapolate from test animals to humans. Second, the relatively high doses administered to test animals must be extrapolated to the lower doses more typical of human exposures. For potential noncarcinogens, safety factors and modifying factors are applied to animal results when developing acceptable human doses. For potential carcinogens, mathematical models are used to extrapolate effects at high doses to effects at lower doses. Epidemiological data can then be used for inferential purposes to establish the credibility of the experimentally derived indices.

Toxic effects considered in these profiles include noncarcinogenic (toxic) and potentially carcinogenic health effects as well as environmental effects. Toxicological endpoints, routes of exposure, and doses in humans and/or animal studies are discussed. Potential carcinogenic health

effects are associated with exposure to a potential carcinogen. Routes of exposure and doses in humans and/or animal studies are provided. Also considered is the USEPA's weight-of-evidence of a compound's carcinogenicity (i.e., Group A, known human carcinogens; Group-B, probable human carcinogens; Group C, possible human carcinogens; Group D, not classifiable as to its carcinogenicity). Environmental effects include acute and chronic toxic effects observed in aquatic biota and terrestrial wildlife.

The available toxicological information indicates that many of the COPCs have both noncarcinogenic and potential carcinogenic health effects in humans and/or in experimental animals. Although the COPCs may potentially cause adverse health and environmental impacts, dose-response relationships and the potential for exposure must be evaluated before the risk to receptors can be determined. Dose-response relationships correlate the magnitude of the dose with the probability of toxic effects, as discussed in the following section.

4.2 Dose-Response Evaluation

An important component of the risk assessment process is the relationship between the dose of a compound (amount to which an individual or population is potentially exposed) and the potential for adverse health effects resulting from exposure to that dose. Dose-response relationships provide a means by which potential public health impacts may be evaluated. Standard reference doses and/or carcinogenic slope factors have been developed for many of the COPCs. This section provides a brief description of these parameters.

Reference (Doses and Reference Concentrations (RfCs) - The RfDs and RfCs are developed for chronic and/or subchronic human exposure to chemicals and are based solely on the noncarcinogenic effects of chemical substances. These values are defined as an estimate of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of adverse effects during a lifetime. The RfD is usually expressed as dose (mg) per unit body weight (kg) per unit time (day). The RfC is expressed as dose (mg) per cubic meter of air (m^3). They are generally derived by dividing a no-observed-(adverse)-effect-level (NOAEL or NOEL) or a lowest observed-adverse-effect-level (LOAEL) for the critical toxic effect by an appropriate "uncertainty factor (UF)." Effect levels are determined from laboratory or epidemiological studies. The uncertainty factor is based on the availability of toxicity data.

Uncertainty factors usually consist of multiples of 10, where each factor represents a specific area of uncertainty naturally present in the extrapolation process. These uncertainty factors are presented below and were taken from the RAGs.

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

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

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

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

In addition to UFs, a modifying factor is applied to each reference dose and is defined as:

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

Thus, the RfD incorporates the certainty of the evidence for chronic human health effects. Even if applicable human data exist, the RfD still maintains a margin of safety such that chronic human health effects are not underestimated.

Carcinogenic Slope Factor (CSF) - Carcinogenic slope factors are used to estimate an upper-bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen (USEPA, 1989). This factor is generally reported in units of $(mg/kg/day)^{-1}$ and is derived through an assumed low-dosage linear multistage model and an extrapolation from

high to low dose-responses determined from animal studies. The value used in reporting the slope factor is the upper 95 percent confidence limit.

CSFs can also be derived from USEPA promulgated unit risk values for air and/or water. CSFs derived from unit risks cannot however be applied to environmental media other than the medium considered in the unit risk estimate.

These slope factors are also accompanied by weight-of-evidence classifications which designate the strength of the evidence that the COPC is a potential human carcinogen.

Quantitative indices of toxicity and USEPA weight-of-evidence classifications are presented in Table 4-1 for the identified COPCs. The hierarchy (USEPA, 1989) for choosing these values was:

- Integrated Risk Information System (IRIS)
- Health Effects Assessment Summary Table (HEAST)
- Region III Specific Directives

The IRIS data base is updated monthly and contains both verified RfDs, RfCs and CSFs. The USEPA has formed an RfD work group to review existing data used to derive RfDs and RfCs. Once this task has been completed the verified RfD appears in IRIS. Like the RfD Work Group, the USEPA has also formed the Carcinogen Risk Assessment Verification Endeavor (CRAVE) Work group to review and validate toxicity values used in developing CSFs. Once the slope factors have been verified via extensive peer review, they also appear in the IRIS data base.

HEAST, on the other hand, provides both interim (unverified) and verified RfDs, RfCs and CSFs. This document is published quarterly and incorporates any applicable changes to its data base.

SECTION 4.0 TABLES

TABLE 4-1

TOXICITY FACTORS
CAMP ALLEN LANDFILL RISK ASSESSMENT

Constituents	Oral CSF $(mg/kg/day)^{-1}$	Inhal. CSF $(mg/kg/day)^{-1}$	Oral RfD (mg/kg/day)	Inhal. RfD (mg/kg/day)	WOE	Target	Critical Effect
Volatile Organic Compounds: benzene	2.90E-02 (i)	2.90E-02 (i)	--	1.71E-03 (e)	A	Blood	Hematological impairment
benzyl chloride	1.70E-01 (i)	--	--	--	B2	--	--
bromomethane	--	--	1.40E-03 (i)	1.43E-03 (i)	D	Cells	Epithelial hyperplasia of the forestomach/nasal cavity
2-butanone	--	--	6.00E-01 (i)	2.86E-01 (i)	D	Fetus	Decreased birth rate
chlorobenzene	--	--	2.00E-02 (i)	5.71E-03 (a)	D	Liver	Histopathologic changes in liver
chloroform	6.10E-03 (i)	8.05E-02 (i)	1.00E-02 (i)	--	B2	Liver	Lesions
chloromethane	1.30E-02 (h)	6.30E-03 (h)	--	--	C	--	--
1,4-dichlorobenzene	2.40E-02 (h)	--	--	2.29E-01 (i)	C	Liver	Increased weight
1,1-dichloroethane	--	--	1.00E-01 (h)	1.43E-01 (a)	C	--	None observed
1,2-dichloroethane	9.10E-02 (i)	9.10E-02 (i)	--	2.86E-03 (e)	B2	--	--
1,1-dichloroethene	6.00E-01 (i)	1.75E-01 (i)	9.00E-03 (i)	--	C	Liver	Lesions
1,2-dichloroethene	--	--	9.00E-03 (h)	--	D	Liver	Lesions
4-methyl-2-pentanone (methyl isobutyl ketone)	--	--	8.00E-02 (h)	2.29E-02 (a)	--	Liver and Kidney	Increased weight
methylene chloride	7.50E-03 (i)	1.64E-03 (i)	6.00E-02 (i)	8.57E-01 (h)	B2	Liver	Liver toxicity

TABLE 4-1 (Continued)

TOXICITY FACTORS
CAMP ALLEN LANDFILL RISK ASSESSMENT

Constituents	Oral CSF $(mg/kg/day)^{-1}$	Inhal. CSF $(mg/kg/day)^{-1}$	Oral RfD (mg/kg/day)	Inhal. RfD (mg/kg/day)	WOE	Target	Critical Effect
tetrachloroethene	5.20E-02 (e)	2.03E-03 (e)	1.00E-02 (i)	--	B2/C[1]	Liver	Hepatotoxicity
toluene	--	--	2.00E-01 (i)	1.14E-01 (i)	D	Liver and Kidney	Altered weight
1,1,1-trichloroethane	--	--	9.00E-02 (w)	2.86E-01 (w)	D	CNS/Whole Body	Hyperactivity/decreased weight
trichloroethene	1.10E-02 (w)	6.00E-03 (e)	6.00E-03 (e)	--	B2	--	--
1,2,4-trimethylbenzene	--	--	5.40E-04 (e)	--	--	--	--
1,3,5-trimethylbenzene	--	--	4.00E-04 (e)	--	--	--	--
vinyl chloride	1.90 (h)	3.00E-01 (h)	--	--	A	--	--
m-xylene	--	--	2.00 (h)	2.00E-01 (w)	D	CNS/Whole Body	Hyperactivity/decreased weight
o-xylene	--	--	2.00 (h)	2.00E-01 (w)	D	CNS/Whole Body	Hyperactivity/decreased weight
p-xylene	--	--	--	8.57E-02 (w)	D	CNS/Whole Body	Hyperactivity/decreased weight
xylenes (total)	--	--	2.00 (i)	--	D	CNS/Whole Body	Hyperactivity/decreased weight

TABLE 4-1 (Continued)

TOXICITY FACTORS
CAMP ALLEN LANDFILL RISK ASSESSMENT

Constituents	Oral CSF $(mg/kg/day)^{-1}$	Inhal. CSF $(mg/kg/day)^{-1}$	Oral RfD (mg/kg/day)	Inhal. RfD (mg/kg/day)	WOE	Target	Critical Effect
Semivolatile Organic Compounds: acenaphthene	--	--	6.00E-02 (i)	--	--	Liver	Hepatotoxicity
bis(2-chloroethyl)ether	1.10 (i)	1.16 (i)	--	--	B2	--	--
bis(2-ethylhexyl)phthalate	1.40E-02 (i)	--	2.00E-02 (i)	--	B2	Liver	Increased weight
2,4-dimethylphenol	--	--	2.00E-02 (i)	--	--	CNS/Blood	Clinical signs/ hematological changes
hexachlorobutadiene	7.80E-02 (i)	7.70E-02 (i)	2.00E-04 (h)	--	C	Renal tubules (kidney)	Regeneration (increased weight)
2-methylphenol (o-cresol)	--	--	5.00E-02 (i)	--	C	Whole Body/CNS	Decreased body weight and neurotoxicity
4-methylphenol (p-cresol)	--	--	5.00E-03 (h)	--	C	Whole Body/ Respiratory Sys./ CNS	Maternal death/distress/ hyperactivity
Pesticides: aldrin	17.0 (i)	17.1 (i)	3.00E-05 (i)	--	B2	Liver	Liver toxicity
beta-BHC	1.80 (i)	1.79 (i)	--	--	C	--	--
gamma-BHC (Lindane)	1.30 (h)	--	3.00E-04 (i)	--	C	Liver and Kidney	Liver and kidney toxicity
alpha-chlordane[2]	1.30 (i)	1.29 (i)	6.00E-05 (i)	--	B2	Liver	Regional liver hypertrophy
4,4'-DDD	2.40E-01 (i)	--	--	--	B2	--	--

TABLE 4-1 (Continued)

TOXICITY FACTORS
CAMP ALLEN LANDFILL RISK ASSESSMENT

Constituents	Oral CSF $(mg/kg/day)^{-1}$	Inhal. CSF $(mg/kg/day)^{-1}$	Oral RfD (mg/kg/day)	Inhal. RfD (mg/kg/day)	WOE	Target	Critical Effect
4,4'-DDE	3.40E-01 (i)	--	--	--	B2	--	--
4,4'-DDT	3.40E-01 (i)	3.40E-01 (i)	5.00E-04 (i)	--	B2	Liver	Lesions
dieldrin	16.0 (i)	16.1 (i)	5.00E-05 (i)	--	B2	Liver	Lesions
heptachlor epoxide	9.10 (i)	9.10 (i)	1.30E-05 (i)	--	B2	Liver	Increased weight
PCBs: Aroclor-1254(3)	7.70 (i)	--	--	--	B2	--	--
Aroclor-1260(3)	7.70 (i)	--	--	--	B2	--	--
Inorganics: aluminum	--	--	1.00E+00 (o)	--	--	--	--
antimony	--	--	4.00E-04 (i)	--	D	Whole Body/Blood	Increased mortality/ altered chemistry
arsenic	1.75 (i)	15.1 (i)	3.00E-04 (i)	--	A	Skin	Keratosis/hyperpigmentation
barium	--	--	7.00E-02 (i)	1.43E-04 (a)	D	Cardiovascular system	Increased blood pressure
beryllium	4.30 (i)	8.40 (i)	5.00E-03 (i)	--	B2	--	None observed
cadmium	--	6.30 (i)	5.00E-04 (i)	--	B1	Renal cortex	Significant proteinuria
chromium	--	42.0 (i)	5.00E-03 (i)	--	A	--	None observed

TABLE 4-1 (Continued)

TOXICITY FACTORS
CAMP ALLEN LANDFILL RISK ASSESSMENT

Constituents	Oral CSF $(mg/kg/day)^{-1}$	Inhal. CSF $(mg/kg/day)^{-1}$	Oral RfD (mg/kg/day)	Inhal. RfD (mg/kg/day)	WOE	Target	Critical Effect
copper	--	--	3.71E-02 (h)	--	D	Gastrointestinal system	Irritation
lead	--	--	--	--	B2	--	--
manganese	--	--	5.00E-03 (i)	1.43E-05 (i)	D	CNS/lung	Effects
mercury (inorganic)	--	--	3.00E-04 (h)	8.57E-05 (h)	D	Kidney/ nervous system	Effects/neurotoxicity
nickel (refinery dusts)	--	8.40E-01 (i)	--	--	A	--	--
nickel (soluble salts)	--	--	2.00E-02 (i)	--	C	Whole body	Decreased body and organ weights
silver	--	--	5.00E-03 (i)	--	D	Skin	Argyria
thallium[4]	--	--	8.00E-05 (i)	--	D	Liver/Blood/Hair	Increased SGOT/Increased Serum LDH/Adopecia
vanadium	--	--	7.00E-03 (h)	--	D	--	--
zinc	--	--	3.00E-01 (i)	--	D	Blood	Decreased blood enzyme

Notes: [1] Under review
[2] Toxicity factors for chlordane used.
[3] Toxicity factor for polychlorinated biphenyls.
[4] Reference dose applies to thallium carbonate, chloride or sulfate.
i = Integrated Risk Information System (IRIS), 1994
e = Environmental Criteria and Assessment Office (ECAO) (as cited from 4th quarter USEPA, Region III RBC Tables)
h = Health Effects Assessment Summary Tables (HEAST), 1994
a = HEAST Alternative Method, 1994
w = Withdrawn from IRIS or HEAST
o = Other EPA Document (as cited from 4th quarter USEPA, Region III RBC tables)

SECTION 5

5.0 RISK CHARACTERIZATION

This section presents quantitative estimates of potential human health and ecological effects associated with the Camp Allen Landfill, Areas A and B. Sections 5.1 through 5.4 present and discuss the estimated incremental lifetime cancer risks (ILCRs) and hazard indices (HIs) for identified potential receptor groups which could be exposed to COPCs by the exposure pathways presented previously in Section 3.0.

Results of the ecological investigation and potential ecological effects are presented in Section 5.2.

5.1 Potential Human Health Effects

Quantitative risk calculations for potentially carcinogenic compounds estimate (inferentially) the potential incremental lifetime cancer risk levels for an individual. This unit of risk refers to a potential cancer risk that is above the background cancer risk in unexposed individuals. For example, an incremental lifetime cancer risk level of 1×10^{-06} indicates that an exposed individual has an increased probability of developing cancer of 0.000001, subsequent to exposure, over the course of a lifetime. A simpler way of understanding an inferential, incremental cancer risk is to apply the value of a community of exposed individuals. For example, an inferential incremental lifetime cancer risk level of 1×10^{-06} also indicates that one additional case of cancer may occur in a population of one million exposed individuals.

The potential incremental lifetime cancer risk level to an individual is estimated from the following relationship:

$$ICR = \sum_{i=1}^{n} CDI_i \; x \; CSF_i$$

where CSF_i is the cancer slope factor [(mg/kg/day^{-1}] for compound i, and CDI_i is the chronic daily intake (mg/kg/day) for compound i. The cancer slope factor is defined in most instances as an upper 95th percentile confidence limit of the probability of a carcinogenic response based on experimental animal data; the CDI is defined as the exposure expressed as mass of a substance contacted per unit body weight per unit time, averaged over a long period of time (i.e., seven years to a lifetime). The

above equation was derived assuming that cancer is a non-threshold process and that the potential excess risk level is proportional to the cumulative intake over a lifetime.

The above equation is only valid at relatively low risk levels. When estimated risks associated with an exposure pathway are high (i.e., greater than 0.01), the following non-linear equation is used.

$$ICR = \sum_{i=1}^{n} [1-\exp(-CDI \times CSF)]$$

Estimated ICR values will be compared to the target risk range of 1×10^{-04} to 1×10^{-06} which represents USEPA's opinion on what are generally acceptable levels (USEPA, 1990).

In contrast to the above approach for potentially carcinogenic effects, quantitative risk calculations for noncarcinogenic compounds assume that a threshold toxicological effect exists. Therefore, the potential for noncarcinogenic effects are calculated by comparing chronic daily intake levels with reference doses.

Noncarcinogenic effects are estimated by calculating the Hazard Index (HI) which is defined as:

$$HI = \sum_{i=1}^{n} HQ_i$$

where: $HQ_i = CDI_i/RfD_i$

where HQ_i is the hazard quotient for chemical i. CDI_i is the chronic daily intake (mg/kg/day) of compound i, and RfD_i is the reference dose (mg/kg/day) of compound i over a prolonged period of exposure.

A value of 1.0 (unity) is used for examination of the HI. The hazard index is calculated by comparing estimated chronic daily intakes with threshold levels below which there is minimal potential for noncarcinogenic health effects. Any HI equal to or exceeding 1.0 suggests that noncarcinogenic health effects are possible. HI values below 1.0 suggest that systemic adverse health effects will not occur subsequent to exposure.

The estimated potential risks and hazard indices for potential human exposure pathways presented in Section 3.0 are discussed by area in the following sections. Quantitative risk calculations for specific COPCs are provided in Appendix D.

5.2 Area A

The following subsections describe the risk calculations for potential current and future receptors in Area A of the Camp Allen Landfill.

5.2.1 Current Local Adult and Child Residents

Local adults and younger children (1-6 yrs.) living in the vicinity of Area A could potentially contact COPCs through the non-potable use (accidental ingestion/dermal contact) of shallow residential area groundwater. Local adults and older children (7-15 yrs.) could potentially be exposed to surface waters (assumed to be unfiltered, via accidental ingestion and dermal contact), shallow sediments (accidental ingestion and dermal contact) present in the Area A ditch, and outdoor air (inhalation). Access to the ditch (site) is gained through holes in the chain link fence which separates the residential area from Camp Allen. Table 5-1 present ILCR and HI values associated with each of the affected media by exposure pathway.

5.2.1.1 Adult Residents

Potential non-potable use of groundwater by local adult residents exhibited a total ILCR of 6.4×10^{-07}. This value is below USEPA's generally acceptable target risk range of 10^{-06} to 10^{-04}. A total HI value of 1.3×10^{-03} was also calculated for current potential potable shallow groundwater usage. This HI value is well below 1.0, suggesting that potential adverse noncarcinogenic health effects will not occur subsequent to exposure.

Potential accidental ingestion and dermal contact of surface waters from the Area A ditch produced a total ILCR value of 2.7×10^{-05}, which is within USEPA's target risk range. This is due primarily to the presence of Aroclor-1254 which contributed 93 percent of the total risk to this medium. A total HI value of 1.1×10^{-02} was also derived, which is well below 1.0, suggesting that adverse noncarcinogenic health effects are not likely to occur subsequent to exposure.

A total ILCR value of 6.6×10^{-06} was derived for local adults potentially ingesting and dermally contacting shallow ditch sediments. This value is within USEPA's target risk range. The corresponding total HI value was 3.9×10^{-02}, which is well below 1.0, suggesting that systemic health effects will not occur subsequent to exposure.

An evaluation of potential risk subsequent to exposure from outdoor (ambient) air was also conducted. An ILCR value of 9.1×10^{-08} and an HI value of 7.6×10^{-03} were derived. Each of these values falls within the acceptable criteria.

The current total site risk to a local adult potentially exposed to COPCs detected in residential area groundwater, Area A ditch surface water and shallow sediments, and outdoor air is an ILCR of 3.4×10^{-05} (due predominantly to Aroclor-1254 in the surface water) and an HI of 5.9×10^{-02}. Each of these values fall below or within the acceptable criteria, indicating that excess cancers or adverse health effects will not occur subsequent to exposure.

5.2.1.2 Child Residents

Local children living in the vicinity of Area A could also potentially be exposed to COPCs in residential area shallow groundwater used for non-potable purposes, to surface waters and sediments upon accessing the ditch surrounding Area A, and to outdoor air.

Potential exposure by young children (1-6 yrs.) to residential groundwater resulted in a total ILCR value of 3.8×10^{-07}. This value is below the target risk range of 10^{-06} to 10^{-04}, which USEPA considers to be generally protective of human health. The corresponding total HI value of 3.0×10^{-03}, is below 1.0. As a result, noncarcinogenic adverse health effects are not expected in young children subsequent to non-potable groundwater exposure.

Older children who could potentially contact or accidentally ingest Area A ditch surface waters exhibited a total ILCR value of 4.2×10^{-05}, which is within USEPA's target risk range. This risk level is due primarily to the presence of Aroclor-1254, which contributed 95 percent of the total risk associated with this medium. A corresponding total HI value of 4.0×10^{-02} was also derived, indicating that noncarcinogenic adverse health effects are not expected in older children subsequent to exposure to surface waters.

Older children could also be exposed to Area A shallow ditch sediments by gaining access to the site. The corresponding total ILCR value for potential Area A ditch sediment exposure was 1.8×10^{-05}, which falls within USEPA's target risk range. This is due primarily to the presence of arsenic in the sediments which contributed over 90 percent of the total risk from this medium. The HI value of 3.8×10^{-01} is below 1.0.

An evaluation of potential risk subsequent to exposure from outdoor (ambient) air was also conducted. An ILCR value of 1.0×10^{-07} and an HI value of 2.9×10^{-02} was derived. These values fall outside the target risk range and 1.0, respectively.

The current total site risk to a young child potentially exposed to COPCs in residential area groundwater is an ILCR of 3.8×10^{-07} and an HI of 3.0×10^{-03}. The current total site risk to an older child potentially exposed to COPCs in the surface water, shallow sediments, and outdoor air is an ILCR of 6.0×10^{-05} and an HI of 4.5×10^{-01}. The ILCR values are below or within the acceptable risk range. The HI for both the younger and older child is well below unity, indicating the potential for adverse health effects subsequent to exposure is unlikely.

5.2.2 Current Brig Employees and Prisoners

Brig employees could be exposed to COPCs in Area A, that could include: surface soils (accidental ingestion, dermal contact, and inhalation of fugitive dusts); surface waters (accidental ingestion and dermal contact); shallow sediments (accidental ingestion and dermal contact), and indoor and outdoor air (inhalation). As a conservative approach, Brig employees were considered to be civilian, and working at the facility for a duration of 25 years.

Brig prisoners were assumed to be assigned to the Brig for a maximum duration of two years with potential exposure to COPCs in Area A surface soils in the prison yard, the air within the confines of the Brig buildings (indoor air), and ambient (outdoor) air during recreation activities. Table 5-2 presents the ILCR and HI values associated with each of the affected media by exposure pathway and receptor.

5.2.2.1 Brig Employee

A Brig employee potentially exposed to COPCs in surface soils exhibited an ILCR of 7.0×10^{-05}, due primarily to the presence of arsenic which contributed over 95 percent of the risk to this medium. The HI value, via exposure to surface soils, was 5.9×10^{-01}. The ILCR is within USEPA's risk range while the HI value is below the acceptable value of 1.0.

Brig employees involved in the maintenance of the ditch surrounding the Brig could also potentially contact surface waters and shallow sediments. The total ILCR from potential exposure to surface waters was 2.9×10^{-05}. This ILCR value is within USEPA's target risk range of 1×10^{-06} to 1×10^{-04}. The HI value associated with accidental ingestion and dermal contact of ditch surface waters was 1.5×10^{-02}, suggesting that noncarcinogenic adverse health effects are not expected. The ILCR and HI for accidental ingestion and dermal contact of shallow sediments was 6.4×10^{-06} and 4.3×10^{-02} respectively. The ILCR was within USEPA's target risk range. The HI did not exceed 1.0 for sediments, suggesting that noncarcinogenic adverse health effects would not be expected subsequent to exposure to sediments.

An evaluation of potential risk subsequent to exposure from indoor and outdoor air was also conducted. An ILCR value of 1.3×10^{-06} and an HI of 8.2×10^{-02} for indoor air, and an ILCR value of 1.3×10^{-06} and an HI value of 6.0×10^{-01} for outdoor air was derived for the Brig employee. Each of these values falls within the acceptable criteria.

The total site ILCR for a current Brig employee potentially exposed to COPCs in surface soils, surface water, shallow sediment, indoor air, and outdoor air is 1.1×10^{-04}. The ILCR value for the Brig employee exceeds USEPA's acceptable risk range due to the presence of arsenic in surface soils, and arsenic and Aroclor-1254 in surface water. A total site HI is 1.3 which exceeds the acceptable value of unity, due primarily to the presence of toluene (targeting the liver and kidney) in the outdoor air, which in itself, contributed 36 percent of the total site risk. Other contributing media included: surface soil containing arsenic (targeting the skin) and cadmium (targeting the renal cortex); surface water containing arsenic and manganese (targeting the central nervous system and lung); and shallow sediment containing arsenic.

5.2.2.2 Brig Prisoners

Brig prisoners potentially exposed to COPCs in Area A soils displayed an ILCR of 7.8×10^{-06} and an HI value of 8.2×10^{-01}. All values were within or below acceptable criteria. An evaluation of potential risk subsequent to exposure from indoor and outdoor air was also conducted. An ILCR value of 3.1×10^{-07} and an HI of 2.5×10^{-01} for indoor air, and an ILCR value of 2.8×10^{-08} and an HI of 3.6×10^{-02} for outdoor air were derived for the Brig prisoner. All of these values were within generally acceptable criteria.

The current total site ILCR and total HI for a Brig prisoner potentially exposed to COPCs in the surface soil, indoor air, and outdoor air was 8.1×10^{-06} and 1.1, respectively. Both the ILCR and the HI for the Brig prisoner are within generally acceptable levels.

5.2.3 Future Adult and Child On-Site Residents

If Area A is developed for residential purposes in the future, there is a potential for resident adults and young children (age 1 to 6 years) to be exposed to COPCs in shallow and deep groundwater (ingestion, dermal contact, and VOCs showering), surface soils (accidental ingestion and dermal contact), surface waters (accidental ingestion and dermal contact), shallow and deep sediments (accidental ingestion and dermal contact), and outdoor air (inhalation). Potable use of shallow and deep groundwater was evaluated for the sake of conservatism. Potential groundwater exposures were evaluated using organic and total (unfiltered) inorganic concentrations, and organic and dissolved (filtered) inorganic concentrations. ILCRs and HI values are presented for future potential residential land use by well location in Tables 5-3 (shallow well B-20W), 5-4 (shallow well B-20WSS) and 5-5 (deep well A-MW1B).

Potable use of Area A shallow groundwater (Well B-20W) by future resident adults exhibited a total ILCR value of 2.7×10^{-01} using organic and total inorganic values. Potable use by future children (0-6 yrs.) produced a total ILCR value of 1.8×10^{-01} using organic and total inorganics. These values exceed USEPA's target risk range due to the presence of vinyl chloride which comprised in excess of 96 percent of the risk level for this medium for both the future adult and child resident. Adult HI values of $3.0 \times 10^{+02}$, and child HI values of $6.2 \times 10^{+02}$ for organic and total inorganics, were also derived for potable groundwater use. These values exceed 1.0 due to the presence of

4-methylphenol (targeting the whole body, respiratory system and central nervous system) which contributed 61 percent of the total HI for this medium. Overall, no significant reduction to the ILCR or the HI value was apparent as a result of sample filtration. Refer to Table 5-3.

Future potential potable use of Area A shallow groundwater (Well B-20WSS) by resident adults produced a total ILCR value of 5.4×10^{-03} using organic and total inorganics. Evaluation of future potential potable use by young children (1-6 yrs.) resulted in total ILCR value of 3.0×10^{-03} using organic and total inorganics. These values exceed USEPA's target risk range because of the presence of arsenic and tetrachloroethene which comprised in excess of 71 percent of the risk level for this medium. Adult HI values of $6.6 \times 10^{+01}$, and child HI values of $1.4 \times 10^{+02}$ were derived using organic and total inorganics. These values exceed 1.0 due primarily to the presence of trichloroethene (20 percent), arsenic (15 percent), and manganese (27 percent), in the shallow groundwater ingestion HI. No significant reduction in human health effects was observed in evaluating dissolved inorganic concentrations. Refer to Table 5-4.

Future potential potable use of deep groundwater (Well A-MW1B) exhibited an ILCR and an HI for future adult residents of 8.9×10^{-03} and 7.5, respectively, using organics and total inorganics. An ILCR value of 8.5×10^{-03} and an HI of 3.8 were derived for adults using organics and dissolved inorganics. The ILCR was impacted primarily by the presence of vinyl chloride. A two-fold reduction was observed for noncarcinogenic effects when evaluating filtered inorganics; however, ILCRs and HI values still remained in exceedance of unity. The most significant contributor for the dissolved inorganics was manganese. Thallium was the most significant contributor to risk for the total inorganics.

Future potential potable use of deep groundwater (Well A-MW1B) by children exhibited ILCRs of 5.4×10^{-03} (total) and 5.2×10^{-03} (dissolved), and HIs of $1.2 \times 10^{+01}$ (total) and 6.0 (dissolved). The carcinogenic risk for the child exceeded the target risk range due to the presence of vinyl chloride, which accounted for greater than 95 percent of the risk from deep groundwater. Similar to the adult scenario, a reduction in the noncarcinogenic effects was observed using dissolved inorganics. The HI for unfiltered groundwater was impacted primarily by 1,2-dichloroethene, trichloroethene, arsenic and manganese. The HI for filtered groundwater was impacted primarily by 1,2 dichloroethene, trichloroethene and dissolved manganese, which were detected in the deep aquifer. Refer to Table 5-5.

Future residential adults contacting Area A surface soils exhibited an ILCR of 1.8×10^{-04}, which exceeds USEPA's target risk range of 10^{-06} to 10^{-04}. This exceedance is due primarily to the presence of arsenic, which contributed approximately 46 percent of the risk associated with accidental ingestion and 95 percent of the risk associated with dermal contact. An HI of 1.3 was also derived for residential adult surface soil exposure. The HI exceeds unity, suggesting that systemic health effects may occur, subsequent to exposure. Arsenic and cadmium contributed the most predominantly to the total site HI; however, the HQs estimated for these inorganics were less than unity. Future residential children contacting Area A surface soils exhibited an ILCR of 1.4×10^{-04} which exceeds the upper end of USEPA's target risk range of 1×10^{-04}, because of the presence of arsenic which accounted for almost 100 percent of the risk from surface soils. Young residential children exhibited an HI of 6.4 which exceeds 1.0, due to the presence of arsenic and cadmium which combined contribute over 75 percent of the risk from surface soils.

Potential exposure to Area A ditch surface water resulting from future residential property development produced an ILCR value of 1.2×10^{-04} for adults and 2.0×10^{-04} for children. These values exceed the USEPA's target risk range due to the presence of Aroclor-1254. The HI values for adults and children exposed to Area A ditch surface waters were 4.1×10^{-02} and 6.4×10^{-01}, respectively, suggesting that noncarcinogenic adverse health effects should not occur subsequent to exposure.

Residential adults contacting shallow ditch sediments in Area A produced a total ILCR of 3.2×10^{-05} and an HI value of 1.9×10^{-01}. Deep ditch sediments produced a total ILCR of 2.1×10^{-05} and an HI value of 2.0×10^{-01}. Both the shallow and deep ditch sediments did not exceed USEPA's target risk range for the ILCR or 1.0 for the HI value.

Young residential children contacting Area A shallow sediments displayed a total ILCR value of 1.2×10^{-04} and an HI of 4.0. Deep sediments produced a total ILCR of 7.5×10^{-05} and an HI of 4.8. The ILCRs were within USEPA's acceptable target risk range, the HIs exceeded 1.0 due to the presence of arsenic.

If future resident adults and children were to obtain groundwater in close proximity to shallow well B-20W, the total site ILCR would be 2.7×10^{-01} for adults, and 1.8×10^{-01} for children, using organics and total inorganics. The total site HI value would be $3.0 \times 10^{+02}$ for adults and $6.4 \times 10^{+02}$

for children, using organics and total inorganics. Potable use of shallow Area A groundwater contributed more than 98 percent to the total risk and 90 percent to the total hazard index. Overall, no significant reduction in cancer risk or human health effects was apparent using dissolved inorganic analytical results over total. Refer to Table 5-3.

If future resident adults and children were to obtain groundwater in close proximity to shallow well B-20WSS, the total site ILCR values for adults and children would be 5.8×10^{-03} and 3.5×10^{-03}, respectively, using organics and total inorganics. Total site HI values for adults and children would be, $6.8 \times 10^{+01}$ and $1.6 \times 10^{+02}$, respectively, using organics and total inorganics. Potable use of shallow Area A groundwater contributed 93 percent to the total risk for the adult and 85 percent for the child. Potable use of shallow Area A groundwater contributed more than 87 percent to the total hazard index for the adult, and 81 percent of the total hazard index for the child.

For the deep well A-MW1B (Yorktown Aquifer) scenario, the total site ILCR values for future Area A resident adults and children were 9.3×10^{-03} and 5.9×10^{-03}, respectively, using organics and total inorganics. Total site HI values for adults and children were, 9.2 and $2.8 \times 10^{+01}$, respectively, using organic and total inorganic results. Potable use of deep Area A groundwater contributed more than 95 percent to the total risk for the adult and 91 percent to the total risk for the child. Potable use of deep Area A groundwater contributed 81 percent of the total hazard index for the adult and 42 percent of the total hazard index for the child. Overall, no significant reduction in human health effects was apparent using dissolved inorganics versus total inorganics. Refer to Table 5-5.

5.2.4 Future Construction Workers

Although subsurface soil excavations in the Area A landfill is unlikely, as a conservative measure, future construction workers were considered to potentially be exposed to subsurface soil COPCs in Area A during soil excavations. The routes of exposure considered include: accidental ingestion, dermal contact and inhalation of fugitive dust. The corresponding risk value and hazard index is presented in Table 5-6.

Construction workers potentially exposed to COPCs in subsurface soils exhibited a total ILCR value of 1.3×10^{-06}, and a total HI value of 8.0×10^{-02}. The ILCR was well within USEPA's target risk

range and the HI value was well below unity; therefore, there appears to be little risk of adverse health effects subsequent to exposure.

5.3 Area B

The following subsections describe the risk calculations for potential current and future receptors in Area B of the Camp Allen Landfill.

5.3.1 Current Adult Workers, Area B Pond and Landfill

Current adult workers at both the Area B pond and landfill could potentially be exposed to COPCs, including the accidental ingestion of surface soils, surface water and shallow sediments; dermal contact with surface soils, surface water, and shallow sediments; and inhalation of surface soil (fugitive dusts) and outdoor air. Table 5-7 presents the ILCR and HI values derived for the various pathways.

Adult workers exposed to COPCs in surface soils exhibited an ILCR of 1.9×10^{-05} and an HI value of 1.3×10^{-01}. Potential exposure to surface water yielded an ILCR of 2.1×10^{-06} and an HI value of 7.4×10^{-03}, while potential exposure to shallow sediments yielded an ILCR of 4.4×10^{-06} and an HI value of 2.0×10^{-02}. Outdoor air yielded an ILCR of 1.3×10^{-06} and an HI value of 1.3×10^{-01}.

The total site risk and hazard index for the adult worker was 2.7×10^{-05} and 2.9×10^{-01}, respectively. These values are within or less than USEPA acceptable criteria; therefore, no adverse carcinogenic or noncarcinogenic risks are expected to occur subsequent to exposure.

5.3.2 Current Adult Employees and Children, Area B School

Current adult employees and older children (6-12 yrs.) occupying the Camp Allen Elementary School in Area B of the Camp Allen Landfill, could potentially be exposed to COPCs in surface soil and surface water by accidental ingestion and dermal contact. In addition, exposure to surface soil (fugitive dusts), indoor, and outdoor air could occur via the inhalation pathway. Table 5-8 presents the ILCR and HI values derived from the aforementioned pathways.

Adult employees with the potential for exposure to surface soil, surface water, indoor and outdoor air for Area B School exhibited a total ILCR and HI of 2.9×10^{-05} and 4.4×10^{-01}, respectively. The site ILCR value falls within USEPA's target risk range, and the HI value is less than 1.0; therefore, adverse health effects are not likely to occur, subsequent to exposure.

Children potentially exposed to surface soil, ditch surface waters, indoor air, and outdoor air pathways exhibited a total ILCR and HI value of 1.5×10^{-05} and 8.6×10^{-01}, respectively. The ILCR value falls within the USEPA target risk range, which is generally considered to be acceptable by the USEPA. The HI value is below 1.0, suggesting that noncarcinogenic adverse health effects will not occur to potentially exposed children.

5.3.3 Future Adult and Child Residents, Area B Pond and Landfill

Future adult and young child (1-6 yrs.) residents living in the vicinity of Area B Pond or Area B Landfill, could potentially be exposed to COPCs in affected media, including groundwaters, if the area is developed for residential purposes in the future. Because of the availability of municipal water, potable use of groundwater is highly unlikely even in the event of residential development, however, potable groundwater usage by future residents was considered for the sake of conservatism. Both a shallow well and deep well were considered for this scenario. Tables 5-9 and 5-10 present the ILCR and HI values derived considering the shallow well and deep well, respectively.

5.3.3.1 Future Adult Residents

Future adult residents living in the near vicinity of Area B Pond could potentially be exposed to COPCs detected in: shallow and deep groundwater (ingestion, dermal contact, and inhalation of VOCs); surface soils (ingestion, dermal contact, and inhalation of fugitive dusts); surface water (ingestion, dermal contact); shallow and deep sediments (ingestion, dermal contact); and outdoor air (inhalation). Tables 5-9 and 5-10 present ILCR and HI values associated with each of the affected media by exposure pathway.

Adults exposed to COPCs through the future potential potable use of shallow and deep groundwaters in Area B Pond exhibit ILCR values of 2.9×10^{-02} and 8.0×10^{-05}, respectively. The ILCR value for

the shallow zone exceeds the upper end of USEPA's target risk range of 10^{-04} due to the presence of vinyl chloride which accounted for over 95 percent of the overall risk in the shallow groundwater medium. The ILCR value for deeper groundwater falls within USEPA's generally acceptable target risk range. This generally acceptable risk value is produced by the presence of arsenic which accounted for approximately 85 percent of the overall risk in the deep groundwater medium via the ingestion exposure pathway.

In addition to cancer risks, systemic health effects are also apparent when shallow and deep Area B Pond groundwaters are evaluated as potable water supplies. HI values of $1.9 \times 10^{+01}$ and 2.8 are derived due to the presence of 1,2-dichloroethene (28 percent) and manganese (28 percent) in shallow groundwater and the presence of manganese (83 percent) in the deep groundwater medium. The use of dissolved groundwater inorganic analytical results produces an HI decrease of approximately 47 percent in the shallow aquifer but does not effect the deep groundwater HI value.

Future potential adult resident exposure to COPCs in surface soils by dermal contact, accidental ingestion and the inhalation of fugitive dusts produces an ILCR of 4.5×10^{-05} and an HI of 2.9×10^{-01}. The ILCR value falls within USEPA's generally acceptable target risk range. The HI value is below 1.0 , suggesting the potential for systemic adverse health effects in adults will not occur subsequent to surface soil exposure.

The HI values and ILCR values for surface water, shallow sediments, deep sediments and outdoor air were all within acceptable values and constitute less than 1 percent of either the total ILCR value or the total HI value for this scenario. The total site ILCR and total site HI estimate for future adult residents obtaining groundwater from shallow well location B-MW11A were 2.9×10^{-02} (2.8×10^{-02} for filtered groundwater) and $1.9 \times 10^{+01}$ ($1.0 \times 10^{+01}$ for filtered groundwater), respectively. The total site ILCR and total site HI estimated for future adult residents obtaining groundwater from deep well location B-MW19B were 1.6×10^{-04} (1.3×10^{-04} for filtered groundwater) and $3.2 \times 10^{+00}$ (2.8 for filtered groundwater), respectively. The majority of the risks are associated with the shallow and deep groundwaters.

5.3.3.2 Future Child Residents

Future child residents living in the near vicinity of Area B Pond could potentially be exposed to COPCs detected in: shallow and deep groundwater (ingestion, dermal contact, and inhalation of VOCs); surface soils (ingestion, dermal contact, and inhalation of fugitive dusts); surface water (ingestion, dermal contact); shallow and deep sediments (ingestion, dermal contact); and outdoor air (inhalation). Tables 5-9 and 5-10 present ILCR and HI values associated with each of the affected media by exposure pathway.

Children exposed to COPCs through the future potential potable use of shallow and deep groundwaters in Area B Pond exhibit ILCR values of 1.4×10^{-02} and 3.8×10^{-05}, respectively. The ILCR value for the shallow zone exceeds the upper end of USEPA's target risk range of 1×10^{-04} because of the presence of vinyl chloride which accounted for over 95 percent of the overall risk in the shallow groundwater medium. The ICR value for deeper groundwater falls within USEPA's generally acceptable target risk range. This generally acceptable risk value is produced by the presence of arsenic which accounted for approximately 84 percent of the overall risk in the deep groundwater medium.

In addition to cancer risks, systemic health effects are also apparent when shallow and deep Area B Pond groundwaters are evaluated as potable water supplies. HI values of $3.0 \times 10^{+01}$ and 4.6 are derived because of the presence of 1,2-dichloroethene (28 percent) and manganese (28 percent) in shallow groundwater and the presence of manganese (83 percent) in the deep groundwater medium. The use of dissolved groundwater inorganic analytical results produces an HI decrease of approximately 43 percent in the shallow aquifer, but does not significantly effect the deep groundwater HI value.

Future potential child resident exposure to COPCs in surface soils by dermal contact, accidental ingestion and the inhalation of fugitive dusts produces an ILCR of 4.0×10^{-05} and an HI of 1.6. The ILCR value falls within USEPA's generally acceptable target risk range. The HI of 1.6 exceeds 1.0 suggesting the potential for systemic adverse health effects in children subsequent to surface soil exposure. The COPCs arsenic and cadmium constituted 73 percent of the HI value estimated for the surface soil medium via the ingestion pathway, and 84 percent estimated for the dermal pathway. Arsenic and cadmium target the skin and renal cortex, respectively.

The HI value (2.0) derived for future child residential exposure to shallow sediments also exceeds 1.0. The COPCs arsenic and cadmium are responsible for the majority (58 percent) of the HI for the shallow sediment medium and target the skin and renal cortex, respectively. The ILCR value associated with this medium, 7.1 x 10^{-05}, falls within the upper end of USEPA's generally acceptable risk range.

The HI values and ILCR values for surface water, deep sediments and outdoor air were all within USEPA acceptable criteria and constitute less than 5 percent of the total ILCR and the total HI values for this scenario. Therefore, the majority of the risks and associated hazards are with the shallow and deep groundwaters.

5.3.4 Future Adult and Child Residents, Area B School

Future adult and child (1-6 yrs.) residents at the Area B School, could potentially be exposed to COPCs in shallow and deep groundwater, surface soil, surface water and deep sediments by accidental ingestion and dermal contact. In addition, potential exposure via inhalation could occur from surface soil (fugitive dusts), groundwater (VOCs while showering), and outdoor air, if the area is developed for residential purposes in the future. Because of the availability of municipal water, potable use of groundwater is highly unlikely even in the event of residential development. Potable groundwater usage by future residents was, however, considered for the sake of conservatism, using both shallow and deep groundwater sample results. Table 5-11 presents the ILCR and HI values derived from the aforementioned scenarios.

5.3.4.1 Future Adult Residents

Adult residents potentially exposed to the shallow groundwater (Well B-MW11A), exhibited an ILCR of 2.9 x 10^{-02} and an HI of 1.9 x 10^{+01}. The ILCR exceeds USEPA's generally acceptable target risk range because of the presence of vinyl chloride contributing over 95 percent of the overall risk from groundwater. The HI value exceeded unity due to the presence of manganese (targeting the lung and central nervous system) and 1,2-dichloroethene (targeting the liver) which together accounted for 56 percent of the overall risk from groundwater. The ILCRs and HIs estimated for surface soil, surface water, deep sediment, and the inhalation of outdoor air are within USEPAs acceptable criteria. A sum of the ILCRs and HIs for the surface soil, surface water, deep sediments,

and outdoor air pathways produced a total ILCR of 6.9×10^{-05} and a total HI of 8.0×10^{-01}. However, the total site risk for the future adult on-site resident yielded ILCR and HI values of 2.9×10^{-02} and $2.0 \times 10^{+01}$, respectively. Therefore, it is obvious that the shallow groundwater medium is contributing essentially 100 percent of the total site risk and almost 94 percent of the total hazard index. A significant reduction in the HI value was apparent using dissolved (filtered) inorganic analytical results. Refer to Table 5-11.

Adult residents potentially exposed to deep groundwater (Well B-MW19B) exhibited an ILCR of 8.0×10^{-05} and an HI of 2.8. The ILCRs and HIs estimated for surface soil, surface water, deep sediment and the inhalation of outdoor air are within USEPAs acceptable criteria. A sum of the ILCRs and HIs for the surface soil, surface water, deep sediments, and outdoor air pathways produced a total ILCR of 6.9×10^{-05} and a total HI of 8.0×10^{-01}. The total site risk for the future adult on-site resident using the deep groundwater from well B-MW19B, exhibited an ILCR and an HI value of 1.5×10^{-04} and 3.6, respectively. The ILCR value exceeds USEPA's generally acceptable target risk range of 1×10^{-06} to 1×10^{-04} indicating that adverse carcinogenic effects may occur for individuals exposed to all media, as evaluated in this risk assessment. The HI value exceeds unity, indicating that noncarcinogenic health effects are likely to occur subsequent to future potable deep groundwater usage, since the deep groundwater is contributing approximately 73 percent of the total hazard index. This elevated value is due primarily to the presence of manganese (targeting the lung and central nervous system) which accounted for 83 percent of the overall risk from the deep groundwater. A reduction in human health effects was not apparent using dissolved inorganic analytical results obtained from the deep aquifer. Refer to Table 5-12.

5.3.4.2 Future Child Resident

Child residents potentially exposed to COPCs in shallow groundwater (Well B-MW11A), exhibited an ILCR of 1.4×10^{-02} and an HI of $3.0 \times 10^{+01}$. A sum of the ILCRs and HIs for the surface soil, surface water, deep sediment, and outdoor air pathways produced a total ILCR of 6.8×10^{-05} and a total HI of 4.6. The total site risk for the future child on-site resident exhibited an ILCR and an HI of 1.4×10^{-02} and $3.5 \times 10^{+01}$, respectively. The ILCR value exceeds the USEPA's generally acceptable target risk range, because of the presence of vinyl chloride. The HI value also exceeds 1.0, indicating that noncarcinogenic health effects are likely to occur subsequent to exposure. The HI is due primarily to the presence of 1,2-dichloroethene (targeting the liver) and manganese

(targeting the lung and central nervous system) in the shallow groundwater. An HI value of 4.5 was derived for future potential child residents contacting surface soils. The HI is a result of the presence of manganese in Area B surface soil samples. Refer to Table 5-11.

Child residents potentially exposed to deep groundwater (Well B-MW19B) exhibits an ILCR of 3.8×10^{-05} and an HI of 4.6. A sum of the ILCRs and HIs for the surface soil, surface water, deep sediment, and outdoor air pathways produced a total ILCR of 6.8×10^{-05} and a total HI of 4.6. The total site risk for the future child on-site resident using the deep groundwater from well B-MW19B, exhibited an ILCR and an HI of 1.1×10^{-04} and 9.2, respectively. The ILCR value exceeds USEPA's generally acceptable target risk range indicating that adverse carcinogenic effects may occur for individuals exposed to all media as evaluated in this risk assessment. The HI value exceeds 1.0, indicating that noncarcinogenic health effects are likely to occur subsequent to exposure. The HI is due to the presence of manganese (targeting the lung and central nervous system) in the deep groundwater. A reduction in either the HI or ILCR values was not apparent using dissolved inorganic analytical results. Refer to Table 5-12.

5.3.5 Future Construction Workers, Area B

Future construction workers could also be exposed to subsurface soil COPCs in Area B during excavations. The routes of exposure could include accidental ingestion, dermal contact and inhalation of fugitive dusts. The corresponding risk value and hazard indices are presented in Table 5-13.

Construction workers potentially exposed to COPCs in subsurface soils via ingestion, dermal contact and inhalation of fugitive dusts, produced a total ILCR value of 7.2×10^{-06} and a total HI value of 7.5×10^{-01}. The ILCR was within USEPA's target risk range and the HI value was below unity.

SECTION 5.0 TABLES

TABLE 5-1

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs) FOR CURRENT LOCAL ADULTS AND CHILDREN - RESIDENTIAL AREA AND AREA A CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE NORFOLK, VIRGINIA

	Receptors					
	Adults		Children (1-6 yrs.)		Children (7-15 yrs.)	
Pathway	HI	ILCR	HI	ILCR	HI	ILCR
Shallow Groundwater						
Ingestion	1.4×10^{-04}	2.4×10^{-07}	6.6×10^{-04}	2.3×10^{-07}	--	--
Dermal Contact	1.2×10^{-03}	4.0×10^{-07}	2.3×10^{-03}	1.5×10^{-07}	--	--
Subtotal	1.3×10^{-03}	6.4×10^{-07}	3.0×10^{-03}	3.8×10^{-07}	--	--
Surface Water						
Ingestion	8.2×10^{-03}	8.7×10^{-07}	--	--	2.8×10^{-02}	2.0×10^{-06}
Dermal Contact	2.3×10^{-03}	2.6×10^{-05}	--	--	1.2×10^{-02}	4.0×10^{-05}
Subtotal	1.1×10^{-02}	2.7×10^{-05}	--	--	4.0×10^{-02}	4.2×10^{-05}
Shallow Sediment						
Ingestion	1.8×10^{-02}	2.5×10^{-06}	--	--	2.7×10^{-01}	1.1×10^{-05}
Dermal Contact	2.1×10^{-02}	4.1×10^{-06}	--	--	1.1×10^{-01}	6.5×10^{-06}
Subtotal	3.9×10^{-02}	6.6×10^{-06}	--	--	3.8×10^{-01}	1.8×10^{-05}
Outdoor (Ambient) Air						
Inhalation	7.6×10^{-03}	9.1×10^{-08}	--	--	2.9×10^{-02}	1.0×10^{-07}
TOTAL	5.9×10^{-02}	3.4×10^{-05}	3.0×10^{-03}	3.8×10^{-07}	4.5×10^{-01}	6.0×10^{-05}

Notes: -- = Not applicable

TABLE 5-2

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs) FOR CURRENT BRIG PRISONERS AND BRIG EMPLOYEES - AREA A CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE NORFOLK, VIRGINIA

Pathway	Receptors			
	Brig Prisoners (Military)		Brig Employees (Civilian)	
	HI	ILCR	HI	ILCR
Surface Soil				
Ingestion	3.2×10^{-01}	2.2×10^{-06}	2.3×10^{-01}	2.0×10^{-05}
Dermal Contact	5.0×10^{-01}	5.6×10^{-06}	3.6×10^{-01}	5.0×10^{-05}
Inhalation[1]	7.4×10^{-05}	8.0×10^{-10}	2.6×10^{-04}	3.6×10^{-08}
Subtotal	8.2×10^{-01}	7.8×10^{-06}	5.9×10^{-01}	7.0×10^{-05}
Surface Water				
Ingestion	--	--	1.2×10^{-02}	1.2×10^{-06}
Dermal Contact	--	--	3.1×10^{-03}	2.8×10^{-05}
Subtotal	--	--	1.5×10^{-02}	2.9×10^{-05}
Shallow Sediment				
Ingestion	--	--	1.5×10^{-02}	1.8×10^{-06}
Dermal Contact	---	--	2.8×10^{-02}	4.6×10^{-06}
Subtotal	--	--	4.3×10^{-02}	6.4×10^{-06}
Indoor Air				
Inhalation	2.5×10^{-01}	3.1×10^{-07}	8.2×10^{-02}	1.3×10^{-06}
Outdoor (Ambient) Air				
Inhalation	3.6×10^{-02}	2.8×10^{-08}	6.0×10^{-01}	1.3×10^{-06}
TOTAL	$1.1 \times 10^{+00}$	8.1×10^{-06}	$1.3 \times 10^{+00}$	1.1×10^{-04}

Notes: [1] Fugitive dusts
-- = Not applicable

TABLE 5-3

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs) FOR FUTURE ON-SITE RESIDENTS - AREA A, SHALLOW WELL LOCATION B-20W CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE NORFOLK, VIRGINIA

	Receptors			
	Adults		Children (1-6 yrs.)	
Pathway	HI	ILCR	HI	ILCR
Shallow Groundwater (Well B20W)				
Ingestion	$1.9 \times 10^{+02}$ ($1.8 \times 10^{+02}$)	7.8×10^{-02} (7.8×10^{-02})	$2.9 \times 10^{+02}$ ($2.8 \times 10^{+02}$)	3.7×10^{-02} (3.6×10^{-02})
Dermal Contact	$1.8 \times 10^{+01}$ ($1.8 \times 10^{+01}$)	3.2×10^{-03} (3.2×10^{-03})	$3.4 \times 10^{+01}$ ($3.4 \times 10^{+01}$)	1.2×10^{-03} (1.2×10^{-03})
Inhalation(1)	$8.9 \times 10^{+01}$	1.9×10^{-01}	$3.0 \times 10^{+02}$	1.4×10^{-01}
Subtotal	$3.0 \times 10^{+02}$ ($2.9 \times 10^{+02}$)	2.7×10^{-01} (2.7×10^{-01})	$6.2 \times 10^{+02}$ ($6.1 \times 10^{+02}$)	1.8×10^{-01} (1.8×10^{-01})
Surface Soil				
Ingestion	6.3×10^{-01}	6.7×10^{-05}	$5.9 \times 10^{+00}$	1.2×10^{-04}
Dermal Contact	6.4×10^{-01}	1.1×10^{-04}	4.6×10^{-01}	1.6×10^{-05}
Inhalation(2)	3.7×10^{-05}	6.0×10^{-09}	2.5×10^{-04}	8.0×10^{-09}
Subtotal	$1.3 \times 10^{+00}$	1.8×10^{-04}	$6.4 \times 10^{+00}$	1.4×10^{-04}
Surface Water				
Ingestion	3.0×10^{-02}	4.2×10^{-06}	5.6×10^{-01}	1.6×10^{-05}
Dermal Contact	1.1×10^{-02}	1.2×10^{-04}	8.1×10^{-02}	1.8×10^{-04}
Subtotal	4.1×10^{-02}	1.2×10^{-04}	6.4×10^{-01}	2.0×10^{-04}
Shallow Sediment				
Ingestion	8.7×10^{-02}	1.2×10^{-05}	$3.3 \times 10^{+00}$	9.2×10^{-05}
Dermal Contact	1.0×10^{-01}	2.0×10^{-05}	7.2×10^{-01}	2.9×10^{-05}
Subtotal	1.9×10^{-01}	3.2×10^{-05}	$4.0 \times 10^{+00}$	1.2×10^{-04}
Deep Sediment				
Ingestion	1.1×10^{-01}	7.5×10^{-06}	$4.1 \times 10^{+00}$	5.7×10^{-05}
Dermal Contact	9.1×10^{-02}	1.3×10^{-05}	6.5×10^{-01}	1.8×10^{-05}
Subtotal	2.0×10^{-01}	2.1×10^{-05}	$4.8 \times 10^{+00}$	7.5×10^{-05}

TABLE 5-3 (Continued)

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs) FOR FUTURE ON-SITE RESIDENTS - AREA A, SHALLOW WELL LOCATION B-20W CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE NORFOLK, VIRGINIA

Pathway	Receptors			
	Adults		Children (1-6 yrs.)	
	HI	ILCR	HI	ILCR
Outdoor (Ambient) Air				
Inhalation	7.6×10^{-03}	9.1×10^{-08}	5.2×10^{-02}	1.2×10^{-07}
TOTAL	$3.0 \times 10^{+02}$ ($2.9 \times 10^{+02}$)	2.7×10^{-01} (2.7×10^{-01})	$6.4 \times 10^{+02}$ ($6.3 \times 10^{+02}$)	1.8×10^{-01} (1.8×10^{-01})

Notes: (1) Shower model; Andelman, 1985
(2) Fugitive dusts
( ) = Risk value derived using dissolved (filtered) inorganic concentrations.

TABLE 5-4

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs) FOR FUTURE ON-SITE RESIDENTS - AREA A, SHALLOW WELL LOCATION B-20WSS CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE NORFOLK, VIRGINIA

Pathway	Receptors			
	Adults		Children (1-6 yrs.)	
	HI	ILCR	HI	ILCR
Shallow Groundwater (Well B-20WSS)				
Ingestion	$4.1 \times 10^{+01}$ ($1.8 \times 10^{+01}$)	2.4×10^{-03} (8.6×10^{-04})	$6.4 \times 10^{+01}$ ($2.8 \times 10^{+01}$)	1.1×10^{-03} (4.0×10^{-04})
Dermal Contact	$2.7 \times 10^{+00}$ ($2.7 \times 10^{+00}$)	2.5×10^{-04} (2.5×10^{-04})	$5.1 \times 10^{+00}$ ($5.0 \times 10^{+00}$)	9.4×10^{-05} (9.3×10^{-05})
Inhalation(1)	$2.2 \times 10^{+01}$	2.7×10^{-03}	$7.3 \times 10^{+01}$	1.8×10^{-03}
Subtotal	$6.6 \times 10^{+01}$ ($4.3 \times 10^{+01}$)	5.4×10^{-03} (3.8×10^{-03})	$1.4 \times 10^{+02}$ ($1.1 \times 10^{+02}$)	3.0×10^{-03} (2.3×10^{-03})
Surface Soil				
Ingestion	6.3×10^{-01}	6.7×10^{-05}	$5.9 \times 10^{+00}$	1.2×10^{-04}
Dermal Contact	6.4×10^{-01}	1.1×10^{-04}	4.6×10^{-01}	1.6×10^{-05}
Inhalation(2)	3.7×10^{-05}	6.0×10^{-09}	2.5×10^{-04}	8.0×10^{-09}
Subtotal	$1.3 \times 10^{+00}$	1.8×10^{-04}	$6.4 \times 10^{+00}$	1.4×10^{-04}
Surface Water				
Ingestion	3.0×10^{-02}	4.2×10^{-06}	5.6×10^{-01}	1.6×10^{-05}
Dermal Contact	1.1×10^{-02}	1.2×10^{-04}	8.1×10^{-02}	1.8×10^{-04}
Subtotal	4.1×10^{-02}	1.2×10^{-04}	6.4×10^{-01}	2.0×10^{-04}
Shallow Sediment				
Ingestion	8.7×10^{-02}	1.2×10^{-05}	$3.3 \times 10^{+00}$	9.2×10^{-05}
Dermal Contact	1.0×10^{-01}	2.0×10^{-05}	7.2×10^{-01}	2.9×10^{-05}
Subtotal	1.9×10^{-01}	3.2×10^{-05}	$4.0 \times 10^{+00}$	1.2×10^{-04}
Deep Sediment				
Ingestion	1.1×10^{-01}	7.5×10^{-06}	$4.1 \times 10^{+00}$	5.7×10^{-05}
Dermal Contact	9.1×10^{-02}	1.3×10^{-05}	6.5×10^{-01}	1.8×10^{-05}
Subtotal	2.0×10^{-01}	2.1×10^{-05}	$4.8 \times 10^{+00}$	7.5×10^{-05}

TABLE 5-4 (Continued)

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs)
FOR FUTURE ON-SITE RESIDENTS - AREA A, SHALLOW WELL LOCATION B-20WSS
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Pathway	Receptors			
	Adults		Children (1-6 yrs.)	
	HI	ILCR	HI	ILCR
Outdoor (Ambient) Air				
Inhalation	7.6×10^{-03}	9.1×10^{-08}	5.2×10^{-02}	1.2×10^{-07}
TOTAL	$6.8 \times 10^{+01}$ ($4.5 \times 10^{+01}$)	5.8×10^{-03} (4.2×10^{-03})	$1.6 \times 10^{+02}$ ($1.3 \times 10^{+02}$)	3.5×10^{-03} (2.8×10^{-03})

Notes: (1) Shower model; Andelman, 1985
(2) Fugitive dust
( ) = Risk value derived using dissolved (filtered) inorganic concentrations.

TABLE 5-5

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs)
FOR FUTURE ON-SITE RESIDENTS - AREA A, DEEP WELL LOCATION A-MW1B
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Pathway	Receptors			
	Adults		Children (1-6 yrs.)	
	HI	ILCR	HI	ILCR
Deep Groundwater (Well A-MW1B)				
Ingestion	$7.2 \times 10^{+00}$ ($3.5 \times 10^{+00}$)	2.7×10^{-03} (2.3×10^{-03})	$1.1 \times 10^{+01}$ ($5.4 \times 10^{+00}$)	1.3×10^{-03} (1.1×10^{-03})
Dermal Contact	2.0×10^{-01} (1.9×10^{-01})	9.7×10^{-05} (9.6×10^{-05})	3.7×10^{-01} (3.5×10^{-01})	3.6×10^{-05} (3.6×10^{-05})
Inhalation(1)	7.6×10^{-02}	6.1×10^{-03}	2.5×10^{-01}	4.1×10^{-03}
Subtotal	$7.5 \times 10^{+00}$ ($3.8 \times 10^{+00}$)	8.9×10^{-03} (8.5×10^{-03})	$1.2 \times 10^{+01}$ ($6.0 \times 10^{+00}$)	5.4×10^{-03} (5.2×10^{-03})
Surface Soil				
Ingestion	6.3×10^{-01}	6.7×10^{-05}	$5.9 \times 10^{+00}$	1.2×10^{-04}
Dermal Contact	6.4×10^{-01}	1.1×10^{-04}	4.6×10^{-01}	1.6×10^{-05}
Inhalation(2)	3.7×10^{-05}	6.0×10^{-09}	2.5×10^{-04}	8.0×10^{-09}
Subtotal	$1.3 \times 10^{+00}$	1.8×10^{-04}	$6.4 \times 10^{+00}$	1.4×10^{-04}
Surface Water				
Ingestion	3.0×10^{-02}	4.2×10^{-06}	5.6×10^{-01}	1.6×10^{-05}
Dermal Contact	1.1×10^{-02}	1.2×10^{-04}	8.1×10^{-02}	1.8×10^{-04}
Subtotal	4.1×10^{-02}	1.2×10^{-04}	6.4×10^{-01}	2.0×10^{-04}
Shallow Sediment				
Ingestion	8.7×10^{-02}	1.2×10^{-05}	$3.3 \times 10^{+00}$	9.2×10^{-05}
Dermal Contact	1.0×10^{-01}	2.0×10^{-05}	7.2×10^{-01}	2.9×10^{-05}
Subtotal	1.9×10^{-01}	3.2×10^{-05}	$4.0 \times 10^{+00}$	1.2×10^{-04}
Deep Sediment				
Ingestion	1.1×10^{-01}	7.5×10^{-06}	$4.1 \times 10^{+00}$	5.7×10^{-05}
Dermal Contact	9.1×10^{-02}	1.3×10^{-05}	6.5×10^{-01}	1.8×10^{-05}
Subtotal	2.0×10^{-01}	2.1×10^{-05}	$4.8 \times 10^{+00}$	7.5×10^{-05}

TABLE 5-5 (Continued)

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs) FOR FUTURE ON-SITE RESIDENTS - AREA A, DEEP WELL LOCATION A-MW1B CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE NORFOLK, VIRGINIA

Pathway	Receptors			
	Adults		Children (1-6 yrs.)	
	HI	ILCR	HI	ILCR
Outdoor (Ambient) Air				
Inhalation	7.6×10^{-03}	9.1×10^{-08}	5.2×10^{-02}	1.2×10^{-07}
TOTAL	$9.2 \times 10^{+00}$ ($5.5 \times 10^{+00}$)	9.3×10^{-03} (8.9×10^{-03})	$2.8 \times 10^{+01}$ ($2.2 \times 10^{+01}$)	5.9×10^{-03} (5.7×10^{-03})

Notes: (1) Shower model; Andelman, 1985
(2) Fugitive dusts
( ) = Risk value derived using dissolved (filtered) inorganic concentrations.

TABLE 5-6

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs) FOR FUTURE CONSTRUCTION WORKERS - AREA A CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE NORFOLK, VIRGINIA

Pathway	Receptor	
	Construction Workers	
	HI	ILCR
Subsurface Soil		
Ingestion	2.7×10^{-02}	8.5×10^{-07}
Dermal Contact	5.3×10^{-02}	4.5×10^{-07}
Inhalation[1]	1.2×10^{-07}	1.5×10^{-13}
TOTAL	8.0×10^{-02}	1.3×10^{-06}

Notes: [1] Fugitive dusts

TABLE 5-7

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs)
FOR CURRENT ADULT WORKERS - AREA B POND AND LANDFILL
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Pathway	Receptor	
	Adult Workers	
	HI	ILCR
Surface Soil		
Ingestion	5.3×10^{-02}	4.6×10^{-06}
Dermal Contact	7.5×10^{-02}	1.4×10^{-05}
Inhalation[1]	1.3×10^{-04}	1.4×10^{-08}
Subtotal	1.3×10^{-01}	1.9×10^{-05}
Surface Water		
Ingestion	5.4×10^{-03}	1.2×10^{-06}
Dermal Contact	2.0×10^{-02}	9.0×10^{-07}
Subtotal	7.4×10^{-02}	2.1×10^{-06}
Shallow Sediments		
Ingestion	8.0×10^{-03}	9.8×10^{-07}
Dermal Contact	1.2×10^{-03}	3.4×10^{-06}
Subtotal	2.0×10^{-03}	4.4×10^{-06}
Outdoor (Ambient) Air		
Inhalation	1.3×10^{-01}	1.3×10^{-06}
TOTAL	2.9×10^{-01}	2.7×10^{-05}

Notes: [1] Fugitive dusts

TABLE 5-8

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs) FOR CURRENT EMPLOYEES AND CHILDREN - AREA B SCHOOL CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE NORFOLK, VIRGINIA

	Receptors			
	Adults		Children (6-12 yrs.)	
Pathway	HI	ILCR	HI	ILCR
Surface Soil				
Ingestion	1.5×10^{-01}	7.7×10^{-06}	4.9×10^{-01}	6.0×10^{-06}
Dermal Contact	1.9×10^{-01}	1.9×10^{-05}	2.4×10^{-01}	5.8×10^{-06}
Inhalation [1]	7.7×10^{-06}	2.3×10^{-08}	2.5×10^{-05}	1.8×10^{-08}
Subtotal	3.4×10^{-01}	2.7×10^{-05}	7.3×10^{-01}	1.2×10^{-05}
Surface Water				
Ingestion	9.5×10^{-03}	4.6×10^{-07}	2.9×10^{-03}	2.8×10^{-06}
Dermal Contact	7.8×10^{-04}	5.9×10^{-08}	1.6×10^{-02}	2.9×10^{-07}
Subtotal	1.0×10^{-02}	5.2×10^{-07}	1.9×10^{-02}	3.1×10^{-06}
Shallow Sediment				
Ingestion	--	--	--	--
Dermal Contact	--	--	--	--
Indoor Air				
Inhalation	7.4×10^{-02}	1.1×10^{-06}	9.4×10^{-02}	3.4×10^{-07}
Outdoor (Ambient) Air				
Inhalation	1.3×10^{-02}	1.3×10^{-07}	2.1×10^{-02}	4.9×10^{-08}
TOTAL	4.4×10^{-01}	2.9×10^{-05}	8.6×10^{-01}	1.5×10^{-05}

Notes: -- = No COPCs
[1] Fugitive dusts

TABLE 5-9

**INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs)
FOR FUTURE ADULT AND CHILD ON-SITE RESIDENTS
AREA B POND AND LANDFILL, SHALLOW WELL LOCATION B-MW11A
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE
NORFOLK, VIRGINIA**

Pathway	Receptors			
	Adults		Children (1-6 yrs.)	
	HI	ILCR	HI	ILCR
Shallow Groundwater (Well B-MW11A)				
Ingestion	$1.8 \times 10^{+01}$ ($9.0 \times 10^{+00}$)	2.2×10^{-02} (2.1×10^{-02})	$2.7 \times 10^{+01}$ ($1.4 \times 10^{+01}$)	1.0×10^{-02} (9.8×10^{-03})
Dermal Contact	4.1×10^{-01} (3.9×10^{-01})	8.9×10^{-04} (8.9×10^{-04})	7.6×10^{-01} (7.3×10^{-01})	3.3×10^{-04} (3.3×10^{-04})
Inhalation(1)	6.7×10^{-01}	5.8×10^{-03}	$2.2 \times 10^{+00}$	3.9×10^{-03}
Subtotal	1.9×10^{-01} (1.0×10^{-01})	2.9×10^{-02} (2.8×10^{-02})	$3.0 \times 10^{+01}$ ($1.7 \times 10^{+01}$)	1.4×10^{-02} (1.4×10^{-02})
Surface Soil				
Ingestion	1.5×10^{-01}	1.5×10^{-05}	$1.4 \times 10^{+00}$	2.9×10^{-05}
Dermal Contact	1.4×10^{-01}	3.0×10^{-05}	2.4×10^{-01}	1.1×10^{-05}
Inhalation (2)	7.5×10^{-06}	9.7×10^{-10}	5.1×10^{-05}	1.3×10^{-09}
Subtotal	2.9×10^{-01}	4.5×10^{-05}	$1.6 \times 10^{+00}$	4.0×10^{-05}
Surface Water				
Ingestion	1.5×10^{-02}	4.1×10^{-06}	2.9×10^{-01}	1.5×10^{-05}
Dermal Contact	7.2×10^{-03}	4.0×10^{-06}	5.1×10^{-02}	5.6×10^{-06}
Subtotal	2.2×10^{-02}	8.1×10^{-06}	3.4×10^{-06}	2.1×10^{-05}
Shallow Sediment				
Ingestion	4.5×10^{-02}	6.7×10^{-06}	$1.7 \times 10^{+00}$	5.0×10^{-05}
Dermal Contact	4.3×10^{-02}	1.5×10^{-05}	3.0×10^{-01}	2.1×10^{-05}
Subtotal	8.8×10^{-02}	2.2×10^{-05}	$2.0 \times 10^{+00}$	7.1×10^{-05}
Deep Sediment				
Ingestion	6.2×10^{-03}	2.2×10^{-07}	2.3×10^{-01}	1.7×10^{-06}
Dermal Contact	8.9×10^{-03}	5.5×10^{-07}	6.3×10^{-02}	7.8×10^{-07}
Subtotal	1.5×10^{-02}	7.7×10^{-07}	2.9×10^{-01}	2.5×10^{-06}

TABLE 5-9 (Continued)

**INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs)
FOR FUTURE ADULT AND CHILD ON-SITE RESIDENTS
AREA B POND AND LANDFILL, SHALLOW WELL LOCATION B-MW11A
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE
NORFOLK, VIRGINIA**

	Receptors			
	Adults		Children (1-6 yrs.)	
Pathway	HI	ILCR	HI	ILCR
Outdoor (Ambient) Air				
Inhalation	7.6×10^{-03}	9.1×10^{-08}	5.2×10^{-02}	1.2×10^{-07}
TOTAL	$1.9 \times 10^{+01}$ ($1.0 \times 10^{+01}$)	2.9×10^{-02} (2.8×10^{-02})	$3.4 \times 10^{+01}$ ($2.1 \times 10^{+01}$)	1.4×10^{-02} (1.4×10^{-02})

Notes: -- = No COPCs
(1) Shower model; Andelman, 1985
(2) Fugitive dusts
( ) = Unfiltered inorganics

TABLE 5-10

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs)
FOR FUTURE ADULT AND CHILD ON-SITE RESIDENTS
AREA B POND AND LANDFILL, DEEP WELL LOCATION B-MW19B
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Pathway	Receptors			
	Adults		Children (1-6 yrs.)	
	HI	ILCR	HI	ILCR
Deep Groundwater (Well B-MW19B)				
Ingestion	$2.5 \times 10^{+00}$ ($2.2 \times 10^{+00}$)	6.5×10^{-05} (3.6×10^{-05})	$3.8 \times 10^{+00}$ ($3.4 \times 10^{+00}$)	3.0×10^{-05} (1.7×10^{-05})
Dermal Contact	2.0×10^{-02} (2.0×10^{-02})	6.0×10^{-06} (5.9×10^{-06})	3.8×10^{-02} (3.7×10^{-02})	2.2×10^{-06} (2.2×10^{-06})
Inhalation(1)	2.3×10^{-01}	8.5×10^{-06}	7.9×10^{-01}	5.7×10^{-06}
Subtotal	$2.8 \times 10^{+00}$ ($2.5 \times 10^{+00}$)	8.0×10^{-05} (5.0×10^{-05})	$4.6 \times 10^{+00}$ ($4.2 \times 10^{+00}$)	3.8×10^{-05} (2.5×10^{-05})
Surface Soil				
Ingestion	1.5×10^{-01}	1.5×10^{-05}	$1.4 \times 10^{+00}$	2.9×10^{-05}
Dermal Contact	1.4×10^{-01}	3.0×10^{-05}	2.4×10^{-01}	1.1×10^{-05}
Inhalation (2)	7.5×10^{-06}	9.7×10^{-10}	5.1×10^{-01}	1.3×10^{-09}
Subtotal	2.9×10^{-01}	4.5×10^{-05}	$1.6 \times 10^{+00}$	4.0×10^{-05}
Surface Water				
Ingestion	1.5×10^{-02}	4.1×10^{-06}	2.9×10^{-01}	1.5×10^{-05}
Dermal Contact	7.2×10^{-03}	4.0×10^{-06}	5.1×10^{-02}	5.6×10^{-06}
Subtotal	2.2×10^{-02}	8.1×10^{-06}	3.4×10^{-01}	2.1×10^{-05}
Shallow Sediment				
Ingestion	4.5×10^{-02}	6.7×10^{-06}	$1.7 \times 10^{+00}$	5.0×10^{-05}
Dermal Contact	4.3×10^{-02}	1.5×10^{-05}	3.0×10^{-01}	2.1×10^{-05}
Subtotal	8.8×10^{-02}	2.2×10^{-05}	$2.0 \times 10^{+00}$	7.1×10^{-05}
Deep Sediment				
Ingestion	6.2×10^{-03}	2.2×10^{-07}	2.3×10^{-01}	1.7×10^{-06}
Dermal Contact	8.9×10^{-03}	5.5×10^{-07}	6.3×10^{-02}	7.8×10^{-07}
Subtotal	1.5×10^{-02}	7.7×10^{-07}	2.9×10^{-01}	2.5×10^{-06}

TABLE 5-10 (Continued)

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs)
FOR FUTURE ADULT AND CHILD ON-SITE RESIDENTS
AREA B POND AND LANDFILL, DEEP WELL LOCATION B-MW19B
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

	Receptors			
	Adults		Children (1-6 yrs.)	
Pathway	HI	ILCR	HI	ILCR
Outdoor (Ambient) Air				
Inhalation	7.6×10^{-03}	9.1×10^{-08}	5.2×10^{-02}	1.2×10^{-07}
TOTAL	$3.2 \times 10^{+00}$ ($2.8 \times 10^{+00}$)	1.6×10^{-04} (1.3×10^{-04})	$8.9 \times 10^{+00}$ ($8.5 \times 10^{+00}$)	1.7×10^{-04} (1.6×10^{-04})

Notes: -- = No COPCs
(1) Shower model; Andelman, 1985
(2) Fugitive dusts
( ) = Risk value derived using dissolved (filtered) inorganic concentrations.

TABLE 5-11

**INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs)
FOR FUTURE ADULT AND CHILD ON-SITE RESIDENTS
AREA B SCHOOL, SHALLOW WELL LOCATION B-MW11A
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE
NORFOLK, VIRGINIA**

	Receptors			
	Adults		Children (1-6 yrs.)	
Pathway	HI	ILCR	HI	ILCR
Shallow Groundwater (Well B-MW11A)				
Ingestion	$1.8 \times 10^{+01}$ ($9.0 \times 10^{+00}$)	2.2×10^{-02} (2.1×10^{-02})	$2.7 \times 10^{+01}$ ($1.4 \times 10^{+01}$)	1.0×10^{-02} (9.8×10^{-03})
Dermal Contact	4.1×10^{-01} (3.9×10^{-01})	8.9×10^{-04} (8.9×10^{-04})	7.6×10^{-01} (7.3×10^{-01})	3.3×10^{-04} (3.3×10^{-04})
Inhalation(1)	6.7×10^{-01}	5.8×10^{-03}	$2.2 \times 10^{+00}$	3.9×10^{-03}
Subtotal	$1.9 \times 10^{+01}$ ($1.0 \times 10^{+01}$)	2.9×10^{-02} (2.8×10^{-02})	$3.0 \times 10^{+01}$ ($1.7 \times 10^{+01}$)	1.4×10^{-02} (1.4×10^{-02})
Surface Soil				
Ingestion	4.2×10^{-01}	2.6×10^{-05}	$3.9 \times 10^{+00}$	4.8×10^{-05}
Dermal Contact	3.4×10^{-01}	4.1×10^{-05}	6.1×10^{-01}	1.4×10^{-05}
Inhalation (2)	4.6×10^{-06}	1.7×10^{-08}	3.1×10^{-05}	2.2×10^{-08}
Subtotal	7.6×10^{-01}	6.7×10^{-05}	$4.5 \times 10^{+00}$	6.2×10^{-05}
Surface Water				
Ingestion	2.7×10^{-02}	1.6×10^{-06}	8.2×10^{-03}	5.9×10^{-06}
Dermal Contact	2.9×10^{-03}	2.6×10^{-07}	2.0×10^{-02}	3.7×10^{-07}
Subtotal	3.0×10^{-02}	1.9×10^{-06}	2.8×10^{-02}	6.3×10^{-06}
Shallow Sediment				
Ingestion	--	--	--	--
Dermal Contact	--	--	--	--
Deep Sediment				
Ingestion	3.5×10^{-04}	--	1.3×10^{-02}	--
Dermal Contact	1.9×10^{-04}	--	1.3×10^{-03}	--
Subtotal	5.4×10^{-04}		1.4×10^{-02}	

TABLE 5-11 (Continued)

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs)
FOR FUTURE ADULT AND CHILD ON-SITE RESIDENTS
AREA B SCHOOL, SHALLOW WELL LOCATION B-MW11A
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Pathway	Receptors			
	Adults		Children (1-6 yrs.)	
	HI	ILCR	HI	ILCR
Outdoor (Ambient) Air				
Inhalation	7.6×10^{-03}	9.1×10^{-08}	5.2×10^{-02}	1.2×10^{-07}
TOTAL	$2.0 \times 10^{+01}$ ($1.1 \times 10^{+01}$)	2.9×10^{-02} (2.8×10^{-02})	$3.5 \times 10^{+01}$ ($2.2 \times 10^{+01}$)	1.4×10^{-02} (1.4×10^{-02})

Notes: -- = No COPCs
(1) Shower model; Andelman, 1985
(2) Fugitive dusts
( ) = Risk value derived using dissolved (filtered) inorganic concentrations.

TABLE 5-12

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs) FOR FUTURE ADULT AND CHILD ON-SITE RESIDENTS - AREA B SCHOOL, DEEP WELL LOCATION B-MW19B
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Pathway	Receptors			
	Adults		Children (1-6 yrs.)	
	HI	ILCR	HI	ILCR
Deep Groundwater (Well B-MW19B)				
Ingestion	$2.5 \times 10^{+00}$ ($2.2 \times 10^{+00}$)	6.5×10^{-05} (3.6×10^{-05})	$3.8 \times 10^{+00}$ ($3.4 \times 10^{+00}$)	3.0×10^{-05} (1.7×10^{-05})
Dermal Contact	2.0×10^{-02} (2.0×10^{-02})	6.0×10^{-06} (5.9×10^{-06})	3.8×10^{-02} (3.7×10^{-02})	2.2×10^{-06} (2.2×10^{-06})
Inhalation(1)	2.3×10^{-01}	8.5×10^{-06}	7.9×10^{-01}	5.7×10^{-06}
Subtotal	$2.8 \times 10^{+00}$ ($2.5 \times 10^{+00}$)	8.0×10^{-05} (5.0×10^{-05})	$4.6 \times 10^{+00}$ ($4.2 \times 10^{+00}$)	3.8×10^{-05} (2.5×10^{-05})
Surface Soil				
Ingestion	4.2×10^{-01}	2.6×10^{-05}	$3.9 \times 10^{+00}$	4.8×10^{-05}
Dermal Contact	3.4×10^{-01}	4.1×10^{-05}	6.1×10^{-01}	1.4×10^{-05}
Inhalation (2)	4.6×10^{-06}	1.7×10^{-08}	3.1×10^{-05}	2.2×10^{-08}
Subtotal	7.6×10^{-01}	6.7×10^{-05}	$4.5 \times 10^{+00}$	6.2×10^{-05}
Surface Water				
Ingestion	2.7×10^{-02}	1.6×10^{-06}	8.2×10^{-03}	5.9×10^{-06}
Dermal Contact	2.9×10^{-03}	2.6×10^{-07}	2.0×10^{-02}	3.7×10^{-07}
Subtotal	3.0×10^{-02}	1.9×10^{-06}	2.8×10^{-02}	6.3×10^{-06}
Shallow Sediment				
Ingestion	--	--	--	--
Dermal Contact	--	--	--	--
Deep Sediment				
Ingestion	3.5×10^{-04}	--	1.3×10^{-02}	--
Dermal Contact	1.9×10^{-04}	--	1.3×10^{-03}	--
Subtotal	5.4×10^{-04}		1.4×10^{-02}	

TABLE 5-12 (Continued)

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs)
FOR FUTURE ADULT AND CHILD ON-SITE RESIDENTS - AREA B SCHOOL,
DEEP WELL LOCATION B-MW19B
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Pathway	Receptors			
	Adults		Children (1-6 yrs.)	
	HI	ILCR	HI	ILCR
Outdoor (Ambient) Air				
Inhalation	7.6×10^{-03}	9.1×10^{-08}	5.2×10^{-02}	1.2×10^{-07}
TOTAL	$3.6 \times 10^{+00}$ ($3.3 \times 10^{+00}$)	1.5×10^{-04} (1.2×10^{-04})	$9.2 \times 10^{+00}$ ($8.8 \times 10^{+00}$)	1.1×10^{-04} (9.3×10^{-05})

Notes: -- = No COPCs
(1) Shower model; Andelman, 1985
(2) Fugitive dusts
( ) = Risk value derived using dissolved (filtered) inorganic concentrations.

TABLE 5-13

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICES (HIs)
FOR FUTURE CONSTRUCTION WORKERS - AREA B
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Pathway	Receptor	
	Construction Workers	
	HI	ILCR
Subsurface Soil		
Ingestion	6.0×10^{-01}	5.2×10^{-06}
Dermal Contact	1.5×10^{-01}	2.0×10^{-01}
Inhalation[1]	8.3×10^{-05}	6.9×10^{-11}
TOTAL	7.5×10^{-01}	7.2×10^{-06}

Notes: [1] Fugitive dusts

SECTION 6

6.0 ECOLOGICAL RISK ASSESSMENT

An ecological risk assessment (ERA) was performed for Camp Allen and surrounding areas to evaluate the impacts of the Camp Allen Landfill on terrestrial and aquatic habitats on, or adjacent to the site. The ERA assesses the results obtained from the field sampling conducted as part of the remedial investigation at the Camp Allen Landfill. The RI included sampling and chemical analysis of the air, surface water, sediments, soil, and groundwater. The supplementary ecological investigation was performed in June, 1993 and included benthic macroinvertebrate sampling in Area A drainage ditches and Area B pond. In addition, observations of aquatic and terrestrial flora and fauna were conducted and recorded. This assessment also evaluated the potential effects of contaminants from the site on sensitive environments, including wetlands, protected species, and valuable or critical habitats. The conclusions of the ERA will be used in conjunction with the human health risk assessment to determine the appropriate remedial action at this site for the overall protection of public health and the environment.

Three types of information are needed to evaluate potential links between the positively-detected chemical constituents and the ecological endpoints. First, chemical analyses of the appropriate media are necessary to establish the presence, concentrations, and variabilities of the detected chemical constituents. Second, ecological surveys are necessary to establish if adverse ecological effects have occurred. Finally, toxicological information is necessary to evaluate the potential effects of the detected chemical constituents on the ecological receptors. The combination of all three types of data allows the assessment of the relative contribution of other potential causes of the observed effects (as measured by the ecological endpoints), unrelated to the toxic effects of the contaminants of potential concern, such as habitat alterations and natural variability. Therefore, confidence in cleanup and monitoring decisions is greatly enhanced when based on a combination of chemical, ecological, and toxicological data.

Potential risks to aquatic receptors were quantitatively evaluated by comparing contaminant concentrations in the surface water to Virginia Water Quality Standards (WQS) and USEPA Ambient Water Quality Criteria (AWQC) and by comparing contaminant concentrations in the sediment to Sediment Screening Values (SSVs). This comparison also aided in the selection of COPCs. In addition, benthic macroinvertebrates were collected and analyzed for population

statistics; as per the Scope of Work (Baker, May 1993) and correspondence with the U.S. Fish and Wildlife Service, the benthic macroinvertebrates were identified to the family level.

Terrestrial receptors were qualitatively evaluated in this ERA by noting the presence and/or absence of species. In addition, information on the presence of wetlands, threatened or endangered species, and sensitive environments at the site was obtained and evaluated.

The risk assessment methodologies used in this evaluation are consistent with those outlined in the U.S. EPA Supplemental Risk Assessment Guidance for the Superfund, Volume II, Environmental Evaluation Manual (USEPA, 1989a). In addition, information found in the following documents were used to supplement the EPA guidance document:

- Framework for Ecological Risk Assessment (USEPA, 1992d)
- Ecological Assessment of Hazardous Waste Sites: A Field and Laboratory Reference (USEPA, 1989c)
- Rapid Bioassessment Protocols for Use in Streams and Rivers: Benthic Macroinvertebrates and Fish (USEPA, 1989d)

The following sections discuss the ecological risk assessment conducted at Camp Allen. Included in these sections are:

- Selection of target indicator species
- Selection of ecological endpoints
- Ecological exposure characterization
- Aquatic sampling methodologies
- Results of the field sampling
- Ecological effects characterization
- Risk characterization/integration

6.1 Selection of Target Indicator Species

The following sections discuss the target indicator species that were chosen for the ERA and the reason they were chosen. The five primary criteria used for the selection of the indicator species are listed below (USEPA, 1989b):

- Importance to ecological systems
- Sensitivity
- Relevance to human beneficial uses
- Availability of practical methods for prediction and measurement
- Trustee species or regulatory endpoints

The target indicator species included benthic macroinvertebrates, terrestrial flora and fauna, and aquatic flora and fauna, as discussed in the subsections below.

6.1.1 Benthic Macroinvertebrates

Benthic macroinvertebrates were selected as the primary target indicator species to assess the impacts to the aquatic life from the Camp Allen Landfill. The following section describes benthic macroinvertebrates and why they are good indicator species for assessing adverse impacts to the aquatic environment.

Benthic macroinvertebrates are defined as organisms that are large enough to be seen by the unaided eye, and are retained by a U.S. Standard No. 30 sieve (28 meshes per inch, 0.595 mm openings) (USEPA, 1990a). Benthic macroinvertebrates may include insect larvae, worms, mollusks, and crustaceans. They live at least part of their life cycles within or upon available substrates in a body of water or water transport systems (USEPA, 1990a).

Benthic macroinvertebrates are important organisms in the trophic food web. They are a primary food source for many fish species and some terrestrial and bird species. Therefore, a decrease in numbers of benthic macroinvertebrates could lead to decreased populations of fish and other species. Also, constituents that tend to bioaccumulate in organisms could be biomagnified in species that

ingest benthic macroinvertebrates. The advantages of using benthic macroinvertebrates as target indicator species are as follows (USEPA, 1989c):

- Benthic macroinvertebrates generally have limited migration patterns or they are sessile for periods in their life cycle. Therefore, they are good indicators for site-specific impacts (i.e., upstream-downstream).

- Benthic macroinvertebrates have a complex life cycle of approximately one year or more. Therefore, they are good indicators of short-term environmental variations because their sensitive life stages will respond quickly to various stresses, while the overall species community will respond more slowly.

- Benthic macroinvertebrates may be easily identified down to the family level by trained biologists. Degraded conditions often can be detected in the field in a very short time.

- Benthic macroinvertebrates are easily collected by one or two people using relatively inexpensive equipment. In addition, there is no detrimental effect on the resident biota from sampling efforts.

- Benthic macroinvertebrates are abundant in most water bodies.

6.1.2 Terrestrial Flora and Fauna

A variety of terrestrial organisms were present in the Camp Allen Landfill area. These organisms are discussed in greater detail in the Camp Allen Landfill Remedial Investigation Report. During ecological field investigations, the terrestrial studies focused on the areas near the benthic macroinvertebrate sampling locations.

In summary, terrestrial flora included deciduous and coniferous trees (sweet gum, willow oak, white oak, loblolly pine), saplings and shrubs (red mulberry, winged sumac, groundsel tree), vines, (poison ivy, Virginia creeper, Japanese honeysuckle, trumpet creeper, green briar), and herbs and grasses. Wetland areas included Spartina and Phragmites, as well as various sedges and rushes. The

presence, distribution, and abundance of actual species varied with each habitat and was a factor of soil composition, availability of water, habitat age, and human intervention.

Terrestrial fauna included small mammals, reptiles, amphibians, invertebrates, and birds. Raccoons, opossums, and muskrats were observed or detected, via sign, at the pond and at the drainage ways. Cottontail rabbits were observed in the field bordering the pond. Frogs were observed in both the pond and the drainageways and painted and snapping turtles were present in the Area A drainageways. Both painted and snapping turtles were laying eggs in the open ground along the drainageways. Several large colonies of fiddler crabs are present in the wetlands. A variety of birds was observed at Camp Allen including wading birds (egrets feeding at Area B pond), raptors (sparrow hawks and peregrine falcons), shorebirds (laughing gulls and killdeer), and perching birds (starlings, robins, barn swallows, mockingbirds, yellowthroats, red-winged blackbirds, grackles, and mourning doves).

6.1.3 Other Aquatic Fauna and Flora

Fish and other aquatic fauna (e.g., zooplankton, microorganisms) and aquatic flora (e.g., submerged and floating vegetation, algae, etc.) were present in the water bodies. Although these organisms are important in the ecological hierarchy, they were not collected or quantitatively evaluated in this assessment. However, they were accounted for in the application of the ecological endpoints (i.e., comparison of contaminant concentrations to water quality standards and sediment screening values).

6.2 Ecological Endpoints

There are two primary types of ecological endpoints: assessment endpoints and measurement endpoints. Assessment endpoints are environmental characteristics, which, if they were found to be significantly affected, would indicate a need for remediation (e.g., decrease in sport fisheries). Measurement endpoints are quantitative expressions of an observed or measured effect of the contamination of concern. Measurement endpoints may be identical to assessment endpoints (e.g., measurement of abundance of fish), or they may be used as surrogates for assessment endpoints (e.g., toxicity test endpoints). Both types of endpoints were used in this ERA.

The assessment endpoint should be well defined and operational with a subject (e.g., benthic macroinvertebrates) and a characteristic of the subject (e.g., decrease in numbers of benthic macroinvertebrates). An endpoint should be measurable (e.g., numbers of individuals) or predictable from measurements (e.g., toxicity tests), and the endpoint must be susceptible to the contaminant being assessed. Finally, the assessment endpoints should bear some logical relationship to the environmental decisions of concern.

The measurement endpoints must correspond to or be predictive of assessment endpoints. In addition, they must be readily measurable, preferably quickly and cheaply, using existing techniques. Measurement endpoints must take into consideration the magnitude of the contamination (e.g., it would be inappropriate to use abundance of a deer population to assess the effects of an one acre site) and the exposure pathway. The measurement endpoint should be able to document effects that are temporally distributed. Low natural variability in the endpoint is preferred to aid in attributing the variability in the endpoint to the contaminant. Measurement endpoints should be diagnostic of the pollutants of interest, as well as broadly applicable to allow comparison among sites and regions. Also, measurement endpoints should be standardized (e.g., standard procedures for toxicity tests). Finally, it is desirable to use endpoints that already are being measured (if they exist) to determine baseline conditions.

Endpoints are divided into four primary ecological groups: individual, population, community, and ecosystem endpoints. Individual endpoints (e.g., death, growth, tissue concentrations) are evaluated through toxicity tests, models, and other methods used to assess the effects on individual organisms. Population endpoints (e.g., occurrence, abundance, reproductive performance) are evaluated to determine presence and absence of species through field studies. Community endpoints (e.g., number of species, species diversity) are used to describe the complexity of the community. Finally, ecosystem endpoints (e.g., biomass, productivity, nutrient dynamics) are used to determine the effects between groups of organisms, and between organisms and the environment.

The primary goal in deciding upon which ecological endpoints to evaluate was to determine the current effects that the contamination is having on the environment. For this ERA, the assessment endpoint is the integrity of aquatic and/or terrestrial life. The measurement endpoints are the presence or absence of the indicator species (i.e. benthic macroinvertebrates), and the comparisons of contaminant concentrations in the environment to applicable water quality standards or sediment

screening values. Individual and population endpoints were evaluated in this ERA, and are discussed in the following sections. Community and ecosystem endpoint were not evaluated in the ERA as per the Scope of Work (Baker, 1993).

6.2.1 Individual Endpoints

This section discusses the individual endpoints that were used in the ecological investigation including water quality standards and sediment screening values.

6.2.1.1 Water Quality Standards

The Virginia State Water Control Board has promulgated Water Quality Standards (WQS) (VASWCB, 1992). These WQS meet the requirements of both Federal and State law; as regulatory values they are enforceable. WQS are used to evaluate the quality of waters in Virginia.

The Ambient Water Quality Criteria (AWQC) are values published and updated periodically by the United States Environmental Protection Agency (USEPA, 1987). They are not promulgated standards; therefore, they do not have a regulatory impact. The criteria were created to present scientific data and guidance on the environmental effects of pollutants and can be used to derive regulatory requirements based on considerations of water quality impacts (USEPA, 1987). The AWQC are grouped into six primary categories, with four categories for the protection of aquatic organisms and two for the protection of humans. The four categories established for the protection of aquatic organisms include freshwater acute, freshwater chronic, saltwater acute, and, saltwater chronic.

Contaminant concentrations detected in the surface water at Camp Allen were compared to both the Virginia WQS and USEPA AWQC to determine if any criteria were exceeded, based upon the published values. It should be noted that the Virginia WQS for metals are established as dissolved metals, while the AWQC for metals are established as total metals.

6.2.1.2 Sediment Screening Values

In addition to contaminants in surface water, aquatic organisms can be adversely affected by contaminants in sediments. Sediments tend to be a sink for certain types of toxic substances, with chemical concentrations in sediments often several orders of magnitude higher than their concentrations in the overlying water.

At present, promulgated sediment quality criteria do not exist. However, sediment screening values have been published (MacDonald, 1992 and Long and Morgan, 1991) for evaluating the potential for chemical constituents in sediments to cause adverse biological effects. The lower ten percentile [Effects Range-Low (ER-L)] and the median percentile [Effects Range-Median (ER-M)] of biological effects have been developed for several of the chemical constituents detected in sediment at Camp Allen. If sediment contaminant concentrations are above the ER-M, adverse effects on the biota are considered probable. If contaminant concentrations are between the ER-L and ER-M, adverse effects on the biota are considered possible, and USEPA recommends conducting toxicity tests as a follow-up. If contaminant concentrations are below the ER-L, adverse effects on the biota are considered unlikely (Long and Morgan, 1991). Positively-detected constituents detected in the sediments at Camp Allen were compared to the SSVs to determine if any criteria were exceeded.

6.2.2 Population Endpoints

The population endpoints that were evaluated in this ERA included taxon occurrence and abundance. The numbers and types of taxa collected from the sampling stations can qualitatively show trends of increasing or decreasing impacts. For example, for the aquatic environment, if few organisms are collected at downstream stations while there is an abundant population in the upstream stations, the downstream ecosystem may be stressed. Similarly, for the terrestrial environment, an area of stressed vegetation may be indicative of an adversely impacted area.

6.3 Ecological Exposure Characterization

This section of the ERA addresses each potential exposure pathway via surface water, sediment, soil, groundwater, and air. To determine if ecological exposure via these pathways may occur in the absence of remedial actions, an analysis was conducted including the identification and

characterization of the exposure pathways. The following four elements were examined to determine if a complete exposure pathway was present:

- A source and mechanism of chemical release
- An environmental transport medium
- A feasible receptor exposure route
- A receptor exposure point

6.3.1 Surface Water Exposure Pathway

Potential release sources to be considered in evaluating the surface water pathway are contaminated surface soils and groundwater. The release mechanisms to be considered are surface runoff and groundwater seepage. The potential routes to be considered for ecological exposure to the contaminated surface waters are ingestion and dermal contact. Potential exposure points for ecological receptors include species living in, or coming in contact with the surface water.

Positively-detected chemical constituents in the surface water, demonstrate a release from a source to the surface water transport medium. Potential receptors that may be exposed to contaminants in surface waters were observed in, or around surface water in the areas of detected chemical constituents. These potential receptors include fish, benthic macroinvertebrates, birds, reptiles, amphibians, and other aquatic and terrestrial life.

Fish are exposed to contaminants in the surface water by ingesting water while feeding, and by direct contact. Overall, fish have a high exposure to contaminants in the surface water. Potential adverse effects on fish from contaminants in the surface water were evaluated in this ERA by direct comparison of contaminant concentrations in the surface water to published water quality standards.

Benthic macroinvertebrates may swim through the water column and/or may attach themselves to substrates in the surface water. These species have a high potential exposure to contaminants in the surface water because they are in constant contact with the water and often they ingest water while feeding. Potential adverse effects on benthic macroinvertebrates from contaminants in the surface water were evaluated in this ERA by direct comparison of contaminant concentrations in the surface

water to published water quality standards and comparison of population statistics of collected individuals among stations.

Other aquatic organisms (e.g., zooplankton, macroinvertebrates) and aquatic plants are potentially exposed to contaminants in the surface water. These aquatic organisms are a primary food source for benthic macroinvertebrates and fish. In addition, aquatic flora add oxygen to the water through photosynthesis, add nutrients to the sediments when degraded, provide cover for small aquatic organisms, and are ingested by aquatic and terrestrial fauna. These potential receptors were evaluated in this ERA by direct comparisons of contaminant concentrations in the surface water to published water quality standards.

Finally, terrestrial faunal receptors are potentially exposed to contaminants in the surface water through ingestion and dermal contact. The magnitude of the exposure depends on their feeding habits and the amount of time they reside in the contaminated waters. In addition, terrestrial species may ingest organisms (e.g., fish, insects, plants) that have bioconcentrated contaminants from the surface water. Risk to terrestrial ecological receptors from contaminants in the surface water was evaluated qualitatively in this ERA.

6.3.2 Sediment Exposure Pathway

The potential release source to be considered in evaluating the sediment pathway is contaminated groundwater and surface soils. The release mechanisms to be considered are groundwater seepage and surface runoff. The potential routes to be considered for ecological exposure to the contaminated sediments are ingestion and dermal contact. Potential exposure points for ecological receptors include species living in, or coming in contact with the sediments.

Chemical constituents were detected in the sediment demonstrating a release from a source to the sediment transport medium. Potential receptors that may be exposed to contaminants in sediment were observed in, or around sediment in the areas of detected chemical constituents. These receptors include fish, benthic macroinvertebrates, birds, reptiles, amphibians, and other aquatic and terrestrial life.

Fish (especially bottom feeders) are exposed to contaminants in the sediments by ingesting sediments while feeding on benthic macroinvertebrates and/or organic detritus, or by direct contact with the sediments. In addition, fish may ingest benthic macroinvertebrates that have bioconcentrated chemicals from the sediments. Overall, fish can potentially contact sediments containing chemical constituents directly or indirectly. Potential adverse effects on fish from contaminants in the sediments were evaluated indirectly in this ERA by comparisons of contaminant concentrations in the sediments to SSVs.

Benthic macroinvertebrates reside on the bottom of the water bodies either on, or in the sediments. They are exposed to contaminants in sediments by ingesting sediments while feeding, burrowing, or direct contact, and therefore, have a high potential exposure to the contaminants. Potential adverse effects on benthic macroinvertebrates from contaminants in the sediments were evaluated in this ERA by direct comparisons of contaminant concentrations in the sediments to SSVs and comparison of population statistics of collected individuals among stations.

Other aquatic organisms (e.g., zooplankton, macroinvertebrates) and aquatic plants are potentially exposed to contaminants in the sediments. These aquatic organisms are a primary food source for benthic macroinvertebrates and fish. In addition, aquatic flora add oxygen to the water through photosynthesis, add nutrients to the sediments when degraded, provide cover for small aquatic organisms, and are ingested by aquatic and terrestrial fauna. These potential receptors were evaluated in this ERA by direct comparisons of contaminant concentrations in the sediments to SSVs.

Finally, terrestrial faunal receptors potentially are exposed to contaminants in the sediments through ingestion and dermal contact. The magnitude of the exposure depends on their feeding habits and the amount of time they reside in the contaminated sediments. In addition, terrestrial species may ingest organisms (e.g., fish, insects, plants) that have bioconcentrated contaminates from the sediments. Risk to terrestrial ecological receptors from contaminants in the sediments was qualitatively evaluated in this ERA.

6.3.3 Soil Exposure Pathway

Potential release sources to be considered in evaluating the soil pathway are surface or buried wastes and contaminated soil. The release mechanisms to be considered are fugitive dust, leaching, tracking, and surface runoff. The transport medium is the soil. The potential routes to be considered for ecological exposure to the contaminated soils are ingestion and dermal contact. Potential exposure points for ecological receptors include species living in, or coming in contact with the soils.

Chemical constituents were detected in the surface soil demonstrating a release from a source to the surface soil transport medium. Potential receptors that may be exposed to contaminants in surface soil were observed in, or around surface soil in the areas of positively-detected constituents. These receptors include mammals, reptiles and amphibians, burrowing insects, birds, plants, and other terrestrial life.

Terrestrial receptors potentially are exposed to contaminants in the soils through ingestion, dermal contact, and/or direct uptake (for flora). The magnitude of the exposure depends on feeding habits and the amount of time receptors spend in the contaminated soils. In addition, terrestrial species may ingest organisms (e.g., insects, plants) that have bioconcentrated contaminants from the soils. Risk to terrestrial ecological receptors from contaminants in the soils was evaluated qualitatively in this ERA.

6.3.4 Groundwater Exposure Pathway

The potential release source to be considered in evaluating the groundwater pathway is contaminated soils. The release mechanism to be considered is leaching. The routes to be considered for ecological exposure to the contaminated groundwater are ingestion and dermal contact. Groundwater discharge to area surface waters may represent a pathway for contaminant migration. Groundwater to surface water exposure will be evaluated in the surface water section of the ERA.

6.3.5 Air Exposure Pathway

There are two potential release mechanisms to be considered in evaluating the atmospheric pathway: release of contaminated particulates (fugitive dust emissions) and volatilization from surface soil, groundwater, and surface water. The potential exposure points for receptors are areas on, or adjacent to the site. Because the site is highly vegetated, the potential for fugitive dust emissions is limited. Furthermore, ambient air samples obtained during the RI did not contain chemical constituents associated with the landfill. Therefore, this pathway was not evaluated as part of the ERA.

6.4 Ecological Effects Characterization

The detailed biological sampling methodology including the sampling locations and the sampling procedures is discussed in Section 3.6 of the RI report. A summary of the sampling locations and procedures used is summarized below.

Benthic macroinvertebrates were collected at five stations at Camp Allen. Stations BC01 and BC02 were located in the Area B pond and Station BC03 was located in a drainage ditch within Area A, downstream of the pond. Station BC04 was located in the drainage ditch within Area A, on the western side of the landfill, and Station BC05 was located in a drainage ditch within Area A, upstream of Station BC04. The benthic macroinvertebrates were collected at the stations using a 5.5 inch long sediment core with a 3.9 inch diameter (sampling area of 0.0077 m^2).

The results of the ecological investigation, including the field water quality measurements, the abiotic characteristics of the waters, grain size analysis, the benthic macroinvertebrates that were collected at Camp Allen, and the terrestrial species that were observed at Camp Allen are discussed in Section 4.5 of the RI (Baker, 1994).

The results of the ecological effects characterization, including the comparison to available criteria and a discussion of the benthic macroinvertebrates and terrestrial flora and fauna observed at Camp Allen are presented below.

6.4.1 Water Quality Standards

Contaminant concentrations detected in the surface water, at locations close to the benthic macroinvertebrate sampling stations, were compared to both the Virginia WQS and USEPA AWQC to determine if any criteria exceeded the established values. Tables 6-1 through 6-4 include the RI station that was used for the surface water criteria comparison (See the RI for a complete discussion of the surface water sampling that was conducted at Camp Allen).

None of the positively-detected chemical parameters at Station BC04 had published criteria available for comparison, therefore, a comparison table has not been provided for this station. It is important to note that there were positively-detected chemicals at each of the sampling stations that did not have a published Virginia WQS or AWQC available for comparison, therefore, these positively-detected chemicals were not included in the tables. The paragraphs below present a summary of the surface water findings.

At station BC01, total zinc exceeded the acute and chronic AWQC. At Station BC02, total iron and lead exceeded the chronic AWQC. Refer to Tables 6-1 and 6-2.

At Station BC03, several pesticides and inorganics exceeded federal acute and/or chronic criteria for metals. The pesticides included gamma-chlordane, dieldrin, and endrin. The total inorganics included chromium, copper, silver, iron, mercury, zinc, and lead. Aroclor-1254 also exceeded the chronic AWQC. None of the dissolved metals concentrations exceeded Virginia WQS. Refer to Table 6-3.

At Station BC05, only total copper exceeded the acute AWQC. Refer to Table 6-4.

The WQS for dissolved oxygen for estuarine and non-tidal waters are 4.0 mg/L (minimum) and 5.0 mg/L (daily average) and the WQS for pH is 6.0 to 9.0 S.U. The maximum water temperature for non-tidal waters is 32°C. At Station BC03, the dissolved oxygen concentration (1.8 mg/L) was below the acceptable range of Virginia WQS values, while the water temperature (33°C) slightly exceeded the Virginia WQS at Station BC01. The pH at Station BC05 (9.03 S.U.) slightly exceeded the Virginia WQS.

6.4.2 Sediment Screening Values

Contaminant concentrations detected in the sediments, at locations close to the benthic macroinvertebrate sampling stations, were compared to published SSVs to determine if there were any constituents that exceeded the established values (refer to Tables 6-5 through 6-9). (See the RI for a complete discussion of the sediment sampling that was conducted at Camp Allen). Tables 6-5 through 6-9 also include the RI station that was used for the comparison. As with the surface water values, it is important to note that there were positively-detected chemicals at each of the sampling stations that did not have a published Sediment Screening Value available for comparison, and therefore these positively-detected chemicals were not included in the tables. The paragraphs below present a summary of the sediment findings.

At Station BC01, no chemical parameters exceeded ER-M values, however, the following constituents exceeded published ER-L values: 4,4'-DDD, 4,4'-DDE, dieldrin, endrin, and copper Refer to Table 6-5.

At Station BC02, the sediment sample from both Station SDB-06 and Station SWB/SDB-03, was used for comparison to SSVs. One polynuclear aromatic hydrocarbon (PAH), fluoranthene, slightly exceeded the ER-L. Four pesticides, gamma-chlordane, 4,4'-DDD, 4,4'-DDE, and dieldrin, exceeded the ER-L. Aroclor-1254 exceeded the ER-L. Several inorganics exceeded the ER-L, they included: arsenic, cadmium, copper, lead, chromium, nickel, zinc, mercury and silver. These constituents that exceeded the ER-L were primarily from the SWB/SDB-03 Station. Constituents with levels greater than the ER-M included: gamma-chlordane, 4,4'-DDD, 4,4'-DDE, dieldrin, Aroclor-1254, cadmium, copper, lead, silver, mercury, and zinc. Once again, the constituents that exceeded the ER-M values were primarily from the SWB/SDB-03 Station. Refer to Table 6-6.

At Station BC03, two sediment samples were used for comparison, SDA-26 for organics, and SWA/SDA-01 for inorganics. None of the PAHs exceed either the ER-L or ER-M, however, Aroclor-1260 exceeded both of these values. Four pesticides exceeded both the ER-L and ER-M, they were, gamma-chlordane, 4,4'-DDD, 4,4'-DDE, and 4,4'-DDT. In addition, all of the inorganics, arsenic, cadmium, chromium, lead, mercury and silver exceeded the ER-L values. All of the aforementioned inorganics, excluding chromium, exceeded the ER-M. Refer to Table 6-7.

At Station BC04, none of the inorganics with published SSVs exceeded the ER-M, however, five exceeded the ER-L. These included: arsenic, cadmium, chromium, lead and mercury. Refer to Table 6-8.

No chemical parameters at Station BC05 exceeded any of the published ER-L or ER-M values. Refer to Table 6-9.

6.4.3 Benthic Macroinvertebrates

Benthic macroinvertebrates were collected from freshwater and tidally influenced areas adjacent to Camp Allen Landfill. Therefore, the risk characterization will be divided into freshwater and tidally influenced stations.

The benthic macroinvertebrates were classified down to family level as per the scope of work. As a result, taxa richness, number of individuals, and density were calculated. Community statistics such as species diversity and biotic indices were not calculated. This approach was developed cooperatively by LANTDIV, the Navy, and natural resource trustee representatives during the initial site visit.

6.4.3.1 Freshwater Stations

Benthic macroinvertebrates were collected from three freshwater stations: BC01, BC02, and BC03. Station BC01 was sampled upstream of the Area B landfill seep that is discharging to the pond, however, other sources of contamination may be impacting this station. Station BC02 was situated slightly downstream of the Area B landfill seep in an area that has documented contamination.

Stations BC01 and BC02 were located in the Area B pond, while Station BC03 was located in a drainage ditch downstream of this pond. The water at the three stations had a negligible velocity, and the substrate was primarily sand covered with some silt and varying degrees of organic debris. This type of habitat usually contains some characteristic organisms including Tubificidae and Chironomidae (USEPA, 1983).

Benthic macroinvertebrates from eight, seven and six families were collected at Stations BC01, BC02, and BC03, respectively. The density was highest at Station BC02 (95,800/M^2) and lowest at Station BC01 (32,438/M^2). The density was 54,928/M^2 at Station BC03.

The Tubificidae family comprised 96 percent, 90.4 percent, and 91.5 percent of the individuals at Stations BC01, BC02, and BC03, respectively. These organisms are often present in organically polluted areas (Thorp, 1991). However, as discussed above, they also are characteristic species for the habitat type located at these three stations.

No other taxonomic family collected at Station BC01 exceeded more than 1.1 percent of the individuals at that station. At Station BC02, gastropods from the Planorbidae family comprised 5.1 percent of the individuals at this station, while flatworms from the Planariidae family comprised 2.1 percent of the individuals at this station. Finally, chironomids from the Tanypodinae family comprised 7.4 percent of the individuals at Station BC03.

The differences in the types and density of benthic macroinvertebrates collected at each of the stations may be related to the habitat and environmental conditions. Station BC01 was located approximately 30-40 feet upstream of Station BC02. The water was shallower at Station BC01 and the canopy cover was more open at this station than at BC02. Therefore, the water and sediment at Station BC01 would be more affected by the sun and might lead to slightly less favorable conditions for the benthic macroinvertebrates. This is evident in the higher water temperature at Station BC01 (33°C) compared to Station BC02 (28°C). Station BC03 was located in a drainage ditch. The habitat in this area was different than both Stations BC01 and BC02. This area was completely open and the drainage ditch only about ten feet wide. Therefore, different organisms would be expected to occur at this station.

Additionally, as discussed in Section 4.5 of the RI, the results of the grain size analysis indicates that the sediments at the three stations had a different composition. The most coarse sediment was collected from Station BC02, followed by Stations BC01 and BC03. The sediment structure would influence the types of benthic macroinvertebrates that inhabit the areas. Finally, as indicated by the variation between the replicates at the each station, there is an inherent natural spatial variability that would account for some of the differences between the stations.

Overall, three stations had a relatively high density. Station BC02 had the highest density and appeared to have the most diverse community (based on the number of individuals in the different taxa). Although the tubificids dominated all three samples, this is not surprising due to the substrate type. It is possible that these stations are being adversely impacted to some degree, especially due to the dominance of one group of organisms. However, these low level impacts cannot be evaluated without further data (i.e., results of species diversity and biotic indices that could not be calculated with identification of organisms to family level).

6.4.3.2 Tidally-Influenced Stations

Benthic macroinvertebrates were collected from two tidally-influenced stations: BC04 and BC05. Station BC05 was sampled as a background station for BC04, because chemical analytical results indicated that sediments at Station BC05 were less affected than those at Station BC04.

Both stations were located in drainage ditches. The sediments at Station BC04 were a black silt with some organic debris, while the sediments at Station BC05 were a black silt/sand with some organic debris. These types of habitats usually contain some characteristic organisms including Tubificidae and Chironomidae (USEPA, 1983).

Station BC04 had benthic macroinvertebrates representing six taxa while BC05 had benthic macroinvertebrates representing three taxa. Station BC04 had a density of 11,332 M^2, compared to a taxon density of 31,746 M^2 at Station BC05.

The Tubificidae family comprised 51.1 percent, and 87.9 percent, of the individuals at Stations BC04, and BC05, respectively. As discussed earlier, these organisms are often present in organically polluted areas (Thorp, 1991); however, they also are characteristic species for the habitat type located at the stations.

At Station BC04, polychaetes from the family Nereidae comprised 26.7 percent of the individuals at this station, while chironomids from the Chironominae family comprised 21.0 percent of the individuals at this station. At Station BC05, chironomids from the Chironominae family comprised 11.9 percent of the individuals at this station.

The differences in the types and densities of benthic macroinvertebrates collected at each of the stations may be related to the habitat and environmental conditions. Station BC04 is further downstream, and thus tidally influenced to a greater degree. This may account for the presence of polycheates at this station. The drainage ditch at Station BC04 was three to four times wider than the ditch at Station BC05, and the surrounding vegetation was more characteristic of a marsh. In addition, the area around Station BC04 was less disturbed.

The sediment structure also influences the types of benthic macroinvertebrates that inhabit the area. As discussed in the Section 4.5 of the RI, the results of the grain size analysis indicates that the sediments at the two stations had a different composition. The finest sediments were collected from Station BC05 followed by sediments collected at Station BC04. Finally, as discussed above, there is an inherent natural spatial variability that would account for some of the differences between the stations.

Overall, both stations had a relatively high density of individuals. Station BC04 had a lower density than Station BC05; however, it appeared to have the more diverse community (based on the number of individuals in the different taxa). While the tubificids dominated both samples, this is not surprising due to the substrate type, although tubificids may indicate that a habitat is affected by organic pollutants.

6.4.4 Terrestrial Flora and Fauna

Terrestrial organisms were evaluated qualitatively by field observation and identification. Attention was given to potential gross effects (i.e., vegetative stress, death) in addition to organism diversity and successful reproduction.

6.5 Risk Characterization/Integration

This section discusses the results of the ecological effects characterization of the surface water, sediment, and benthic macroinvertebrate analytical data and, the terrestrial field observations conducted during the RI and evaluates the potential impact on the biological integrity at the site from the contaminants.

6.5.1 Surface Water

The concentrations of contaminants in the surface water were compared to applicable state and federal water quality standards. Water quality standards are set to be protective of the aquatic environment; any constituents that exceed these standards, indicate a potentially hazardous environment for the aquatic organisms inhabiting the water body.

Based on this comparison, potential adverse impacts may occur at Station BC01 from zinc, Station BC02 from levels of iron and lead, Station BC03 from levels of gamma-chlordane, dieldrin, endrin, and Aroclor-1254, as well as the total metals levels for copper, mercury, iron, silver, zinc, and lead, and Station BC05 from levels of copper in the surface water, which exceeded the USEPA acute and/or chronic AWQC. In addition, the dissolved oxygen concentration was below the acceptable range for the Virginia WQS at Station BC03 which also may adversely impact aquatic life. The high iron and lead concentrations detected at Station BC02 may be related to leachate from the landfill or high background levels of these constituents. The high inorganic concentrations detected at Station BC03 are probably related to background levels and the refuse (e.g., tires, rims, etc.) disposed in the drainage ditch, the low concentration of pesticides are probably related to site background levels, originating from years of pesticide applications in this area. The low dissolved oxygen concentrations are probably caused by natural conditions (decaying organic matter).

The only other physical or chemical parameters that exceeded the applicable Virginia WQS or federal AWQC at any of the stations was temperature (a slight elevation) at Station BC01, and pH (a slight elevation) at Station BC05. The elevated temperature and pH at Stations BC01 and BC05, respectively, is probably due to factors unrelated to the landfill. The high temperature at Station BC01 is due to natural conditions, while the high pH at Station BC05 may be caused by the new construction upstream of the sampling location. Since the criteria are only minimally exceeded, the potential risk to aquatic life is expected to be low.

6.5.2 Sediment

The concentrations of contaminants in the sediments were compared to the lower 10 percentile (ER-L) and median percentile (ER-M) published sediment screening values (SSV). Because the

screening values are set to be protective of the aquatic environment, any concentrations above these values indicate a potentially toxic environment for the aquatic organisms inhabiting the water body.

At Station BC01, 4,4'-DDD, 4,4'-DDE, dieldrin, endrin and copper exceeded the ER-L values. At Station BC02, gamma-chlordane, 4,4'-DDD, 4,4'-DDE, dieldrin, Aroclor-1254, cadmium, copper, lead, mercury, silver, and zinc exceeded their ER-L and ER-M values, while fluoranthene (a PAH), arsenic, chromium, and nickel only exceeded their ER-L values. At Station BC03, gamma-chlordane, 4,4'-DDD, 4,4'-DDT, 4,4'-DDE, Aroclor-1260, arsenic, cadmium, lead, mercury and silver exceeded the ER-L and ER-M values. Chromium was the only constituent that exceeded just the ER-L value.

At Station BC04, concentrations of arsenic, cadmium, chromium, lead, and mercury exceeded their ER-L values. No chemical parameters at Station BC05 exceeded any of the ER-L or ER-M values.

Based on the comparison to SSVs, adverse impacts to aquatic life are possible at Stations BC01 and BC04 since some of the concentrations of COPCs were between the ER-L and ER-M values. Adverse impacts to aquatic life at Stations BC02 and BC03 from contaminants in the sediments are considered probable, but not necessarily certain, since some of the chemical concentrations at these two stations exceeded both the ER-L and ER-M values (Long and Morgan, 1991).

6.5.3 Benthic Macroinvertebrates

The benthic macroinvertebrate community at each of the stations displayed relatively dense populations of benthic macroinvertebrates typical for urban areas exhibiting some minor impacts. In heavily impacted areas, benthic macroinvertebrates would be absent, or nearly absent, or the substrate would be dominated by pollution resistant species. Lesser impacts are more difficult to establish due to natural spatial variability that occurs in the benthic community. Indices used to evaluate the health of the benthic community in greater detail were not used in the ecological evaluation.

6.5.4 Terrestrial Flora and Fauna

Terrestrial flora and fauna on and around the Camp Allen Landfill were evaluated qualitatively as part of this risk assessment. During the field study, gross impacts such as death, illness, or vegetative stress, if any, were evaluated. Overall diversity of species and evidence of reproduction was also used as a relative measure of environmental health.

From a qualitative standpoint, the flora at the site shows a diversity of vegetation both in general types (trees, shrubs, vines, grasses) and in number of species present. Diversity did vary from location to location based upon the habitat type and the amount of human activity. The woodland by the Area B pond was more diverse, for example, than the edge of the regularly mowed field. No vegetative stress was noted at any of the study locations including the seep at the Area B pond and the drainage ditch at Station BC03. In both these locations submerged aquatic vegetation was present and was growing vigorously. Duckweed (Lemna minor) which is sensitive to chromium (Eisler, 1986) was present in the Area B pond.

At all of the sampling locations, succession from one habitat type to another (i.e., from field to shrubby woods edge) was occurring. For succession to take place, seeds must be able to germinate and young plants must be able to successfully compete and thrive. Reproduction and growth can be a measure of the health of the environment.

The richness of the flora at the site offers cover and food for a variety of fauna. During the field investigation, a number of animals were observed including rabbits, muskrats, opossums, squirrels, turtles (two species), frogs, invertebrates, and birds. All of the animals that were actually observed appeared to be healthy.

In addition, it was clear that reproduction is taking place. Both species of turtles (Painted Turtle, Chrysemys picta marginata, and Snapping Turtle, Chelydra serpentina) were observed digging nest holes or laying eggs. The eggs of the snapping turtle were later dug up and eaten, probably by raccoons; the remaining egg shells appeared to be normal. Larval dragonflies, which appeared to be healthy, were observed in the sediment at Station BC03.

Birds also appear to be reproducing at the site. Several species were observed in marking activities, including red-winged blackbirds, a yellow-breasted chat, a yellowthroat, and mockingbirds. Starlings appeared to be nesting in some salvage yard buildings and the abandoned nest of a ground nesting bird was found along one of the drainage ditches.

The landfill area appeared to support a diverse population of wildlife for a relatively urban area. Many of the species observed are considered hardy and adaptable including raccoons and opossums, starlings, house sparrows, and snapping turtles. However, others like crested flycatchers, sparrow hawks, kingbirds, barn swallows, yellow-breasted chats, and painted turtles are usually found in relatively undeveloped suburban and rural areas.

Little specific information is available outlining effects of contaminants on terrestrial receptors. In addition, actual environmental effects are dependent upon contaminant characteristics, environmental characteristics, and the habits and territories of specific terrestrial species. Therefore, general endpoints were used to assess the health of the terrestrial environment.

6.5.5 Threatened and Endangered Species

One endangered bird, the peregrine falcon (Falco peregrinus), had been observed at the Camp Allen Landfill during the RI field sampling effort. The falcon does not nest in the area and has been seen infrequently. Local ecologists believe that is was attracted to the site to feed on flocks of starlings and pigeons at the salvage yard.

Incomplete information is available on the levels of environmental contamination at the site (i.e., contaminant levels in site plants and animals), on bioaccumulation and bioavailability of contaminants, and on specific contaminant effects to peregrine falcons. Therefore, it is not possible to definitively assess risk to the falcons. However, the falcons are not present at the site regularly and the birds on which they do feed appear to be healthy.

6.5.6 Wetlands

Several wetland areas have been identified at the site. Based upon the results of benthic macroinvertebrate survey and the terrestrial study, these wetlands do not appear to be impacted by the landfill.

6.6 Summary of Risk Characterization/Integration

The following paragraphs discuss the overall results of the risk characterization/integration for the areas at the Camp Allen Landfill.

6.6.1 Station BC01

Based upon the available water quality and sediment criteria, there do not appear to be any potential adverse impacts to the aquatic life at this station due to chemical contaminants in the surface water. Potential adverse effects on aquatic life from contaminants in the sediments are considered possible but not certain. The potential risk to aquatic life from the elevated temperatures is expected to be low, and due primarily to natural conditions. Based upon the macroinvertebrate results, the benthic community at this station does not appear to be significantly impacted.

6.6.2 Station BC02

Based upon the available water quality and sediment criteria, there do not appear to be any potential adverse impacts to the aquatic life at this station due to chemical contaminants in the surface water. However, adverse impacts to the aquatic life due to chemical contaminants in the sediments are considered possible but not certain. Based upon the macroinvertebrate field study, the benthic community at this station does not appear to be significantly impacted.

6.6.3 Station BC03

Based upon the available water quality and sediment criteria, adverse impacts to the aquatic life at this station due to chemical contaminants in the surface water and sediments are considered possible but not certain. Adverse impacts to the aquatic life may occur due to the low dissolved oxygen

concentration; however, low levels of dissolved oxygen are probably due to natural conditions. Based upon the macroinvertebrate study, the benthic community at this station does not appear to be significantly impacted.

6.6.4 Station BC04

Based upon the available water quality and sediment criteria, there do not appear to be any potential adverse impacts to the aquatic life at this station due to chemical contaminants in the surface water; however, adverse effects from contaminants in the sediments are considered possible but not certain. Based upon the macroinvertebrate study, the benthic community at this station does not appear to be significantly impacted.

6.6.5 Station BC05

Based upon the available water quality and sediment criteria, there do not appear to be any potential adverse impacts to the aquatic life at this station due to chemical contaminants in the surface water or sediments. The potential risk to aquatic life from the high pH at this station is expected to be low. The elevated pH levels are probably due to the new construction upstream of the site. Based upon the macroinvertebrate study, the benthic community at this station does not appear to be significantly impacted.

6.6.6 Terrestrial Flora and Fauna

Insufficient information is available to evaluate specific effects from the landfill contaminants on the terrestrial environment. However, based upon the qualitative evaluation, flora appears to be diverse and healthy. The fauna observed were numerous, diverse, and healthy. Normal reproduction was apparent. No gross effects from contaminants were evident.

6.6.7 Threatened and Endangered Species

There does not appear to be any effect upon the visiting peregrine falcon from COPCs at the Camp Allen Landfill.

6.6.8 Wetlands

Wetlands surrounding the Camp Allen Landfill do not appear to be affected by COPCs based on the results of the terrestrial field study and the results of the benthic macroinvertebrate study.

6.7 Ecological Significance

The results of the ecological risk assessment is used in conjunction with the human health risk assessment to support the selection of remedial action(s) for the Camp Allen Landfill that are protective of public health and the environment. The assessment endpoint used to assess the aquatic and terrestrial environment is decreased integrity of the aquatic and terrestrial community. Exceedances of surface water and sediment quality measurement endpoints indicate a low to moderate potential for risk to aquatic life. The benthic community is characteristic of an aquatic ecosystem that has potential impacts from both contaminant exposure and natural conditions. In addition, this benthic community exhibited spatial variations within the range of natural population variation in similar environments. The terrestrial qualitative evaluation did not produce any significant indicators of risk to terrestrial receptors based on observations of diversity and productivity of the fauna and flora. Based on this finite ecological risk assessment, the aquatic community may be impacted by releases from the Camp Allen Landfill. However, remedial measures are being implemented that provide both source removal and source containment, as well as treatment to control further COPC migration into the drainage ditches. Therefore, post-remediation studies are warranted to evaluate the reduction of risks to the aquatic community as a result of site remediation activities.

SECTION 6.0 TABLES

TABLE 6-1

SURFACE WATER DATA AT STATION BC01 COMPARED TO PUBLISHED VIRGINIA WATER QUALITY STANDARDS AND FEDERAL AMBIENT WATER QUALITY CRITERIA FOR THE PROTECTION OF AQUATIC LIFE
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	Station BC01[1] Contaminant Concentration (µg/L)		State of Virginia WQS for the Protection of Aquatic Life[2]				Federal AWQC for the Protection of Aquatic Life[3]			
			Freshwater (µg/L)		Exceeded Criteria for Dissolved Metals		Freshwater (µg/L)		Exceeded Criteria for Total Metals	
	Total	Dissolved	Acute	Chronic	Acute	Chronic	Acute	Chronic	Acute	Chronic
VOCs: Chloroform	24	NA	--	--	NA	NA	28,900*	1,240*	No	No
Inorganics: Iron	539	ND	--	--	NA	NA	--	1,000	NA	No
Lead	1.4J	ND	*	*	NA	NA	82*	3.2*	No	No
Zinc	146J	111	*	*	NA	NA	120*	110*	Yes	Yes

Notes: * Hardness dependent. Federal values use a hardness of 100 mg/L as $CaCO_3$.
(1) Sample concentrations from RI Station SWB/SDB-02.
(2) The WQS are established for dissolved metals.
(3) The AWQC are established for total metals.
ND = Not Detected
NA = Not Applicable
J = Estimated value

TABLE 6-2

SURFACE WATER DATA AT STATION BC02 COMPARED TO PUBLISHED VIRGINIA WATER QUALITY STANDARDS AND FEDERAL AMBIENT WATER QUALITY CRITERIA FOR THE PROTECTION OF AQUATIC LIFE
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	Station BC02[1] Contaminant Concentration (µg/L)		State of Virginia WQS for the Protection of Aquatic Life[2]				Federal AWQC for the Protection of Aquatic Life[3]			
			Freshwater (µg/L)		Exceeded Criteria for Dissolved Metals		Freshwater (µg/L)		Exceeded Criteria for Total Metals	
	Total	Dissolved	Acute	Chronic	Acute	Chronic	Acute	Chronic	Acute	Chronic
VOCs:										
Benzene	6J	NA	--	--	NA	NA	5,300*	--	No	NA
Chloroform	3J	NA	--	--	NA	NA	28,900*	1,240*	No	No
1,2-dichloroethane	3J	NA	--	--	NA	NA	118,000*	20,000*	No	No
Trichloroethene	45	NA	--	--	NA	NA	45,000*	21,900*	No	No
Pesticides:										
4,4'-DDD	0.007J	ND	--	--	NA	NA	0.6*	--	No	NA
Inorganics:										
Iron	6,440	386	--	--	NA	NA	--	1,000	NA	Yes
Lead	5.9J	ND	*	*	NA	NA	83*	3.2*	No	Yes
Zinc	60.8J	57.8	*	*	NA	NA	120*	110*	No	No

Notes: * Hardness dependent. Federal values use a hardness of 100 mg/L as $CaCO_3$.
(1) Sample concentrations from RI Station SWB/SDB-03.
(2) The WQS are established for dissolved metals.
(3) The AWQC are established for total metals.
ND = Not Detected
NA = Not Applicable
J = Estimated value

TABLE 6-3

SURFACE WATER DATA AT STATION BC03 COMPARED TO PUBLISHED VIRGINIA WATER QUALITY STANDARDS AND FEDERAL AMBIENT WATER QUALITY CRITERIA FOR THE PROTECTION OF AQUATIC LIFE
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	Station BC03[1] Contaminant Concentration (µg/L)		State of Virginia WQS for the Protection of Aquatic Life[2]				Federal AWQC for the Protection of Aquatic Life[3]			
			Freshwater (µg/L)		Exceeded Criteria for Dissolved Metals		Freshwater (µg/L)		Exceeded Criteria for Total Metals	
	Total	Dissolved	Acute	Chronic	Acute	Chronic	Acute	Chronic	Acute	Chronic
VOCs: Trichloroethene	3J	NA	--	--	NA	NA	45,000*	21,900*	No	No
PCBs/Pesticides: Aroclor-1254	0.44J	NA	--	0.014	NA	No	0.014	--	NA	Yes
gamma-chlordane	0.024J	ND	2.4	0.0043	No	No	2.4	0.0043	No	Yes
4,4'-DDD	0.26L	ND	--	--	NA	NA	0.6*	--	No	NA
4,4'-DDE	0.069J	ND	--	--	NA	NA	1,050*	--	No	NA
Dieldrin	0.027J	ND	2.5	0.0019	No	No	2.5	0.0019	No	Yes
Endrin	0.07J	ND	0.18	0.0023	No	No	0.18	0.0023	No	Yes
Inorganics: Chromium	104	ND	16	11	No	No	16	11	Yes	Yes
Copper	446	ND	*	*	NA	NA	18*	12*	Yes	Yes
Iron	78,300	817	--	--	NA	NA	--	1,000	NA	Yes
Lead	800	2.7	*	*	NA	NA	82*	3.2*	Yes	Yes
Mercury	3.9	ND	2.4	0.012	No	No	2.4	0.012	Yes	Yes
Nickel	57	ND	*	*	NA	NA	1,400*	160*	No	No
Silver	12	ND	*	--	NA	NA	4.1*	--	Yes	NA
Zinc	1,860J	61J	*	*	NA	NA	120*	110*	Yes	Yes

Notes: * Hardness dependent. Federal values use a hardness of 100 mg/L as $CaCO_3$.
(1) Sample concentrations from RI Station SWA/SDA-01.
(2) The WQS are established for dissolved metals.
(3) The AWQC are established for total metals.
ND = Not Detected NA = Not Applicable J = Estimated value L = Estimated value, biased low

TABLE 6-4

SURFACE WATER DATA AT STATION BC05 COMPARED TO PUBLISHED VIRGINIA WATER QUALITY STANDARDS AND FEDERAL AMBIENT WATER QUALITY CRITERIA FOR THE PROTECTION OF AQUATIC LIFE CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	Station BC05[1] Contaminant Concentration (µg/L)		State of Virginia WQS for the Protection of Aquatic Life[2]				Federal AWQC for the Protection of Aquatic Life[3]			
			Freshwater (µg/L)		Exceeded Criteria for Dissolved Metals		Freshwater (µg/L)		Exceeded Criteria for Total Metals	
	Total	Dissolved	Acute	Chronic	Acute	Chronic	Acute	Chronic	Acute	Chronic
Pesticides:										
4,4'-DDD	0.008J	ND	--	--	NA	NA	3.6**	--	No	NA
Inorganics:										
Copper	5.1	ND	2.9	2.9	No	No	2.9	2.9	Yes	Yes

Notes:
- * Hardness dependent. Federal values use a hardness of 100 mg/L as $CaCO_3$.
- ** No criteria available. Lowest Observed Effect Level (LOEL) is presented.
- (1) Sample concentrations from RI Station SWA/SDA-11.
- (2) The WQS are established for dissolved metals.
- (3) The AWQC are established for total metals.
- ND = Not Detected
- NA = Not Applicable
- J = Estimated value

TABLE 6-5

SEDIMENT DATA AT STATION BC01 COMPARED TO PUBLISHED SEDIMENT SCREENING VALUES CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	Station BC01[1] Contaminant Concentration (mg/kg)	SSV[2] (mg/kg)			
		ER-L	Exceeded Criteria[3]	ER-M	Exceeded Criteria[3]
PAHs:					
Benzo(a)anthracene	0.050J	0.261	No	1.60	No
Benzo(a)pyrene	0.048J	0.430	No	1.60	No
Chrysene	0.050J	0.384	No	2.80	No
Fluoranthene	0.130J	0.600	No	5.10	No
Phenanthrene	0.086J	0.240	No	1.50	No
Pyrene	0.075J	0.665	No	2.60	No
Pesticides:					
4,4'-DDD	0.015	0.002	Yes	0.02	No
4,4'-DDE	0.0049	0.002	Yes	0.027	No
Dieldrin	0.0036J	0.00002	Yes	0.008	No
Endrin	0.00087J	0.00002	Yes	0.045	No
Inorganics:					
Chromium	3	81	No	370	No
Copper	41.4	34	Yes	270	No
Lead	26.9J	46	No	218	No
Zinc	80.4	150	No	410	No

Notes:
(1) Sample concentration from RI Station SWB/SDB-02.
(2) Sediment Screening Values (MacDonald, 1992 or Long and Morgan, 1991).
(3) If sediment concentrations are above the ER-M, adverse health effects on biota are considered probable. If sediment concentrations are between ER-L and ER-M, adverse health effects on biota are possible. If contaminant concentrations are below the ER-L, adverse health effects are considered unlikely.

ER-L - Effective Range-Low ER-M - Effective Range-Median
ND - Not Detected J - Estimated value

TABLE 6-6

SEDIMENT DATA AT STATION BC02 COMPARED TO PUBLISHED SEDIMENT SCREENING VALUES
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	Station BC02 Contaminant Concentration (mg/kg)		SSV[1] (mg/kg)					
				Exceeded Criteria[2]			Exceeded Criteria[2]	
	RI Station SBD-06	RI Station SWB/SDB-03	ER-L	RI Station SDB-06	RI Station SWB/SDB-03	ER-M	RI Station SDB-06	RI Station SWB/SDB-03
PAHs: Benzo(a)anthracene	ND	0.19J	0.261	No	No	1.60	No	No
Benzo(a)pyrene	ND	0.21J	0.430	No	No	1.60	No	No
Chrysene	ND	0.35J	0.384	No	No	2.80	No	No
Fluoranthene	ND	0.63J	0.60	No	Yes	5.10	No	No
2-methylnaphthalene	0.030J	ND	0.070	No	No	0.67	No	No
Naphthalene	0.046J	ND	0.160	No	No	2.10	No	No
Phenanthrene	ND	0.20J	0.24	No	No	1.50	No	No
Pyrene	ND	0.35J	0.665	No	No	2.60	No	No
Pesticides/PCBs: gamma-Chlordane	0.0024J	0.056K	0.0005	Yes	Yes	0.006	No	Yes
4,4'-DDD	0.100K	4.2	0.002	Yes	Yes	0.02	Yes	Yes
4,4'-DDE	0.036K	0.850	0.002	Yes	Yes	0.027	Yes	Yes
Dieldrin	0.0031J	0.086K	0.00002	Yes	Yes	0.008	No	Yes
Aroclor-1254	0.200	7.6	0.022	Yes	Yes	0.180	Yes	Yes

Notes: (1) Sediment Screening Values (MacDonald, 1992 or Long and Morgan, 1991).
(2) If sediment concentrations are above the ER-M, adverse health effects on biota are considered probable. If sediment concentrations are between ER-L and ER-M, adverse health effects on biota are possible. If contaminant concentrations are below the ER-L, adverse health effects are considered unlikely.

ER-L - Effective Range-Low
ER-M - Effective Range-Median
ND - Not Detected
J = Estimated value
K = Estimated value, biased high
L = Estimated value, biased low

TABLE 6-6 (Continued)

SEDIMENT DATA AT STATION BC02 COMPARED TO PUBLISHED SEDIMENT SCREENING VALUES
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	Station BC02 Contaminant Concentration (mg/kg)		SSV[1] (mg/kg)					
				Exceeded Criteria[2]			Exceeded Criteria[2]	
	RI Station SBD-06	RI Station SWB/SDB-03	ER-L	RI Station SDB-06	RI Station SWB/SDB-03	ER-M	RI Station SDB-06	RI Station SWB/SDB-03
Inorganics:								
Arsenic	6.3L	25.2	8	No	Yes	70	No	No
Cadmium	ND	41.9	1.2	No	Yes	9.6	No	Yes
Chromium	6.1	225	81	No	Yes	370	No	No
Copper	12.8	298	34	No	Yes	270	No	Yes
Lead	23.1J	497J	46	No	Yes	218	No	Yes
Nickel	ND	42.3	21	No	Yes	223	No	No
Mercury	0.26K	1.4K	0.15	Yes	Yes	0.71	No	Yes
Silver	ND	10	1.0	No	Yes	3.7	No	Yes
Zinc	43.2	1,020	150	No	Yes	410	No	Yes

Notes: (1) Sediment Screening Values (MacDonald, 1992 or Long and Morgan, 1991).
(2) If sediment concentrations are above the ER-M, adverse health effects on biota are considered probable. If sediment concentrations are between ER-L and ER-M, adverse health effects on biota are possible. If contaminant concentrations are below the ER-L, adverse health effects are considered unlikely.

ER-L - Effective Range-Low
ER-M - Effective Range-Median
ND - Not Detected
J = Estimated value
K = Estimated value, biased high
L = Estimated value, biased low

TABLE 6-7

SEDIMENT DATA AT STATION BC03 COMPARED TO PUBLISHED SEDIMENT SCREENING VALUES
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	Station BC03[1] Contaminant Concentration (mg/kg)	SSV[2] (mg/kg)			
		ER-L	Exceeded Criteria[3]	ER-M	Exceeded Criteria[3]
Pesticides/PCBs: gamma-chlordane	0.069L	0.0005	Yes	0.006	Yes
4,4'-DDD	0.120L	0.002	Yes	0.02	Yes
4,4'-DDT	0.073L	0.001	Yes	0.0461	Yes
4,4'-DDE	0.095L	0.002	Yes	0.027	Yes
Aroclor-1260	0.350L	0.022	Yes	0.180	Yes
PAHs: Benzo(a)anthracene	0.042J	0.261	No	1.6	No
Benzo(a)pyrene	0.036J	0.43	No	1.6	No
Chrysene	0.038J	0.384	No	2.8	No
Fluoranthene	0.064J	0.60	No	5.1	No
Phenanthrene	0.027J	0.24	No	1.5	No
Pyrene	0.663J	0.665	No	2.6	No
Inorganics: Arsenic	100	8	Yes	70	Yes
Cadmium	72	1.2	Yes	9.6	Yes
Chromium	200	81	Yes	370	No
Lead	1,000	46	Yes	218	Yes
Mercury	3	0.15	Yes	0.71	Yes
Silver	13	1.0	Yes	3.7	Yes

Notes:
(1) Sample concentration from RI Station SDA-26 for organics and SWA/SDA-01 for inorganics.
(2) Sediment Screening Values (MacDonald, 1992 or Long and Morgan, 1991).
(3) If sediment concentrations are above the ER-M, adverse health effects on biota are considered probable. If sediment concentrations are between ER-L and ER-M, adverse health effects on biota are possible. If contaminant concentrations are below the ER-L, adverse health effects are considered unlikely.

ER-L - Effective Range-Low
ER-M - Effective Range-Median
ND - Not Detected
K = Estimated value, biased high
L = Estimated value, biased low
J = Estimated value

TABLE 6-8

SEDIMENT DATA AT STATION BC04 COMPARED TO PUBLISHED SEDIMENT SCREENING VALUES CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	Station BC04[1] Contaminant Concentration (mg/kg)	SSV[2] (mg/kg)			
		ER-L	Exceeded Criteria[3]	ER-M	Exceeded Criteria[3]
Inorganics: Arsenic	53	8	Yes	70	No
Cadmium	8.1	1.2	Yes	9.6	No
Chromium	160	81	Yes	370	No
Lead	130	46	Yes	218	No
Mercury	0.5	0.15	Yes	0.71	No

Notes: (1) Sample concentration from RI Station SWA/SDA-05, sampled for inorganics only.
(2) Sediment Screening Values (MacDonald, 1992 or Long and Morgan, 1991).
(3) If sediment concentrations are above the ER-M, adverse health effects on biota are considered probable. If sediment concentrations are between ER-L and ER-M, adverse health effects on biota are possible. If contaminant concentrations are below the ER-L, adverse health effects are considered unlikely.

ER-L - Effective Range-Low
ER-M - Effective Range-Median
ND - Not Detected
NA - Not Applicable

TABLE 6-9

SEDIMENT DATA AT STATION BC05 COMPARED TO PUBLISHED SEDIMENT SCREENING VALUES
CAMP ALLEN LANDFILL, NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Chemical	Station BC05[1] Contaminant Concentration (mg/kg)	SSV[2] (mg/kg)			
		ER-L	Exceeded Criteria[3]	ER-M	Exceeded Criteria[3]
Inorganics: Lead	23	46	No	218	No

Notes: [1] Sample concentration from RI Station SWA/SDA-11, sampled for inorganics only.
[2] Sediment Screening Values (MacDonald 1992 or Long and Morgan, 1991).
[3] If sediment concentrations are above the ER-M, adverse health effects on biota are considered probable. If sediment concentrations are between ER-L and ER-M, adverse health effects on biota are possible. If contaminant concentrations are below the ER-L, adverse health effects are considered unlikely.
ER-L - Effective Range-Low
ER-M - Effective Range-Median
ND - Not Detected
NA - Not Applicable

SECTION 7

7.0 SOURCES OF UNCERTAINTY

Uncertainties are encountered throughout the process of performing a risk assessment. This section discusses the sources of uncertainty inherent in the following elements of the public health and environmental evaluation performed for the Camp Allen Landfill site:

- Sampling and Analysis
- Selection of COPCs
- Exposure assessment
- Toxicity assessment
- Risk characterization
- Ecological risk assessment
- Chemicals not quantitatively evaluated

Uncertainties associated with this risk assessment are discussed in the following paragraphs. Table 7-1 summarizes the potential effects of certain uncertainties on the estimation of human health risks and ecological effects.

7.1 Sampling and Analysis

The development of a risk assessment depends on the reliability of, and uncertainties with, the analytical data available to the risk assessor. These, in turn, are dependent on the operating procedures and techniques applied to the collection of environmental samples in the field and their subsequent analyses in the laboratory. To minimize the uncertainties associated with sampling and analysis at the Camp Allen Landfill, USEPA approved sampling and analytical methods were employed. Data was generated in most cases using USEPA's Statement of Work for CLP. Samples were analyzed for target compound list (TCL) organics, target analyte list (TAL) inorganics and cyanide. Samples were taken from locations specified in the approved Work Plan along with the necessary QA/QC samples.

Analytical data are limited by the precision and accuracy of the methods of analysis. For example, contract laboratory program (CLP) methods of analysis have, in general, a precision of approximately plus or minus 50 percent depending upon the sample media and the presence of interfering compounds. A value of 100 μg/kg could be as high as 150 μg/kg or as low as 50 μg/kg. In addition, the statistical methods used to compile and analyze the data (mean concentrations,

detection frequencies) are subject to the overall uncertainty in data measurement. Furthermore, chemical concentrations in environmental media fluctuate over time and with respect to sampling location. Analytical data must be sufficient to consider the temporal and spatial characteristics of contamination at the site with respect to exposure.

7.2 Selection of COPCs

The selection of COPCs is performed in a risk assessment following the evaluation of data. Analytical data must also be comprehensive in order to address the COPCs associated with the site. Types of COPCs encountered at the Camp Allen site include volatile organics, semivolatile organics, pesticides, PCBs, and inorganic constituents.

Soil, groundwater and air COPCs for the Camp Allen site were selected based on comparisons of maximum detected concentrations with Region III COPC screening concentrations as the primary criterion, in conjunction with prevalence. Region III COPC screening concentrations were used as secondary criteria in the selection of surface water COPCs, since AWQCs were used as the primary criteria. For the selection of sediment COPCs, the NOAA SSVs (ER-Ms) were applied, with no application of Region III COPC screening concentrations.

Region III COPC screening concentrations are based on exposure assumptions and equations that are intended to introduce conservatism in the risk assessment process by changing the COPC screening method from a relative toxicity screen of RAGS to an absolute comparison of risk. However, the use of the Region III COPC screening concentrations results in the application of a set of non-site-specific assumptions in the determination of COPCs at the Camp Allen Landfill site. Currently, no base closures are planned for the Camp Allen Landfill site and future residential development is not considered an expected land use for the area. The application of the residential COPC screening concentrations to soil and groundwater COPC selections would, therefore, tend to result in a list of COPCs that could be considered overly conservative for a military base. Likewise, the application of acute and chronic freshwater and marine AWQCs (where human health AWQCs are unavailable) in the selection of surface water COPCs, and the application of NOAA SSVs in the selection of sediment COPCs introduces additional conservatism into the baseline human health RA. The use of conservative COPC selections in the baseline RA ensures the protection of public health

in that the results of the baseline RA are incorporated into the determination of remedial alternatives and remedial action objectives in the Feasibility Study.

7.3 Exposure Assessment

In performing exposure assessments, uncertainties arise from two main sources. First, uncertainties arise in estimating the fate of a compound in the environment, including estimating release and transport in a particular environmental medium. Second, uncertainties arise in the estimation of chemical intakes resulting from contact by a receptor with a particular medium.

To estimate an intake, certain assumptions must be made about exposure events, exposure durations, and the corresponding assimilation of constituents by the receptor. Exposure factors have been generated by the scientific community and have undergone review by the USEPA. The USEPA has published an Exposure Factors Handbook which contains the best and latest values. Regardless of the validity of these exposure factors, they have been derived from a range of values generated by studies of limited numbers of individuals. In all instances, values used in this risk assessment, scientific judgments, and conservative assumptions agree with those of the USEPA.

For potential dermal exposures to COPCs, a non-steady state approximation for dermal absorption was used. The non-steady state approximation assumes that the skin absorbs COPCs and slowly releases the dose over time. This approximation is a factor of about one order-of-magnitude greater than the steady-state absorption daily intake commonly used in the risk assessment process. Because the non-steady state approximation of absorbed dose is based on the K_{ow} of organic chemicals and default inputs to the absorption algorithm, the potential for overestimating absorbed dose is likely high. Skin exhibits bi-phasic characteristics, and transport of chemicals across this barrier is governed by many factors which cannot be accounted for in the risk assessment process.

The use of a Reasonable Maximum Exposure (RME) approach, designed as not to underestimate daily intakes, was employed throughout this risk assessment. The use of 95th percent upper confidence estimates of the arithmetic mean or maximum values as the concentration term in estimating the CDI reduces the potential for underestimating exposure at the Camp Allen Landfill site. Recent research using Monte-Carlo estimation techniques indicate that USEPA's RME represents the 98 to 99.99 percent upper limit of the estimated risk distribution.

Uncertainty was also introduced into the Exposure Assessment by the methodology employed for determination of groundwater exposure concentrations used for the estimations of chronic daily intakes. Hypothetical exposure point locations were selected at wells in which the most elevated concentrations of COPCs were detected. The most recent data from these wells were used as exposure concentrations. This resulted in some COPCs not being quantitatively evaluated in the risk assessment since not all COPCs were detected in samples collected from the selected well locations. In the shallow aquifer beneath Area A, 1,2-dichloroethane, bis(2-ethylhexyl)phthalate, aldrin, and antimony were not evaluated; in the shallow aquifer in Area B, 1,2-dishloroethene, chlorobenzene, PCE, bis(2-chloroethyl)ether, bis(2-ethylhexyl)phthalate, dieldrin, gamma-BHC, heptachlor epoxide, antimony, cadmium, mercury, dissolved antimony, dissolved arsenic, and dissolved chromium were not evaluated; in the deep aquifer beneath Area B, vinyl chloride, dieldrin, heptachlor epoxide, aluminum, antimony, beryllium, cadmium, chromium, nickel, and vanadium were not quantitatively evaluated. However, it should be noted that even without these COPCs, quantitative evaluations of future residential exposures to both aquifers in Areas A and B resulted in risk levels exceeding acceptable USEPA criteria. Therefore, exclusion of these COPCs resulted in an insignificant underestimation of chronic daily intake.

The use of total inorganic analytical results in groundwater to represent conditions "at the tap", result in an overestimation of potential risks for these COPCs. This is particularly true for Area B deep groundwaters. The presence of fine particulates in unfiltered groundwater samples contribute greatly to the concentration of inorganic constituents such as lead, beryllium, antimony, and chromium. The presence of fine particulates in groundwater samples can be attributed to the design of monitoring wells which is different than potable well design.

Potential human receptors evaluated for groundwater exposures included current local adult and young child (ages 1-6 years) residents living in the Glenwood area, and future on-site adult and young child residents living in Areas A and B. Residents in the Glenwood area were evaluated for potential exposures resulting from non-potable use of the shallow groundwater (i.e., car washing, lawn/garden watering, etc.). Future on-site residents were evaluated for the more conservative potable use scenarios (i.e., drinking water and bathing). Older children (ages 7-15 years) were not evaluated under the current and future exposure scenarios for non-potable and potable uses, respectively. This may seem to introduce some uncertainty since older children are more likely than young children to engage in non-potable use activities, and just as likely to engage in potable use

activities. However, it should be noted that evaluation of exposures to adults and young children represent minimum and maximum potential CDIs under both the current and future scenarios. If CDIs were estimated for the older child, they would be expected to fall within the CDI range defined by those estimated for the adult and young child. Therefore, CDIs estimated for adults and young children, and consequently, the risk levels derived from those CDIs, are inclusive of those that would be estimated for the older children.

7.4 Toxicological Assessment

In making quantitative estimates of the toxicity of varying dosages of compounds to human receptors, uncertainties arise from two sources. First, data on human exposure and the subsequent effects are usually insufficient, if they are at all available. Human exposure data usually lack adequate concentration estimations and suffer from inherent temporal variability. Therefore, animal studies are often used and new uncertainties arise from the process of extrapolating animal results to humans. Second, to obtain observable effects with a manageable number of experimental subjects, high doses of a compound are often used. In this situation, a high dose means that high exposures are used in the experiment with respect to most environmental exposures. Therefore, when applying the results of the animal experiment to the human condition, the effects at the high doses must be extrapolated to approximate effects at lower doses.

In extrapolating effects from high doses in animals to low doses in people, scientific judgment and conservative assumptions are employed. In selecting animal studies for use in dose-response calculations, the following factors are considered:

- Studies are preferred where the animal closely mimics human pharmacokinetics.
- Studies are preferred where dose intake most closely mimics the intake route and duration for humans.
- Studies are preferred which demonstrate the most sensitive response to the compound in question.

For compounds believed to cause threshold effects (i.e., noncarcinogens) safety factors are employed in the extrapolation of effects from animals to humans and from high doses to low doses. In deriving carcinogenic potency factors, the 95th percent upper confidence value is promulgated by the agency to prevent underestimation of potential risk.

The use of conservative assumptions, results in quantitative indices of toxicity that are not expected to underestimate potential toxic effects, but may overestimate these effects by an order of magnitude or more.

7.5 Human Risk Characterization

The risk characterization bridges the gap between risk assessment and risk management, ultimately providing impetus for the remediation of the site.

Other uncertainties associated with risk characterization include the assumption of chemical additivity and the inability to predict synergistic or antagonistic interactions between COPCs. These uncertainties are inherent in any inferential risk assessment. USEPA promulgated inputs to the quantitative risk assessment and toxicological indices are calculated to be protective of the human receptor and to err conservatively, so as to not underestimate the potential human health risks.

7.6 Ecological Risk Assessment

The procedures used to assess risks to ecological receptors, are also subject to uncertainties. Uncertainty exists in the use of toxicological data in ecological risk assessments. The values used in the ecological endpoint comparison (WQSs and the NOAA SSVs) are set to be protective of a majority of the potential receptors. There are some species, however, that are not protected by the values because of their sensitivity to the chemicals. Furthermore, the toxicity of chemical mixtures is not well understood. All the toxicity information used in the ERA for evaluating risk to the ecological receptors is for individual chemicals. Chemical mixtures can affect the organisms very differently than the individual chemicals. In addition, there were several constituents that did not have WQSs or SSVs. Therefore, potential effects to ecological receptors from these chemicals can not be determined.

7.7 Compounds Not Quantitatively Evaluated

The inorganic COPCs, aluminum and lead, were not quantitatively evaluated in the baseline RA. Lead is currently considered a B2 - probable human carcinogen, as well as a developmental toxin in young children. The lack of promulgated toxicological indices for aluminum and lead does not have significant effects on the underestimation of risk due to the presence of other COPCs such as vinyl chloride, 1,1,1-trichloroethane, Aroclor-1254, and Aroclor-1260 in environmental media at relatively high levels.

Although these constituents were not quantitatively evaluated, this risk assessment has been performed using conservative concentration estimates (RMEs), exposure scenarios (use of the shallow aquifer as a drinking water source), and available toxicological information.

SECTION 7.0 TABLES

TABLE 7-1

SUMMARY OF UNCERTAINTIES IN THE RESULTS OF THE HUMAN HEALTH AND ECOLOGICAL ASSESSMENT CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

	Potential Magnitude for Over-Estimation of Risks	Potential Magnitude for Under-Estimation of Risks	Potential Magnitude for Over or Under-Estimation of Risks
Environmental Sampling and Analysis Sufficient samples may not have been taken to characterize the media being evaluated.			Low
Systematic or random errors in the chemical analysis may yield erroneous data.			Low
Groundwater samples taken from residential wells in Glenwood Park were not analyzed for semivolatile organics, pesticides, PCBs or inorganic constituents.		Low	
Selection of COPCs The use of USEPA Region III COPC screening concentrations in selecting COPCs in soil, groundwater, air, and sediments.			Low
The use of AWQCs and NOAA SSVs in selecting COPCs for human health evaluation.	Low		
Exposure Assessment The standard assumptions regarding body weight, exposure period, life expectancy, population characteristics, and lifestyle may not be representative of the actual exposure situations.			Moderate
The use of the 95th percentile upper confidence level data in the estimation of the RME.	Moderate		
Use of non-steady state dermal absorption model for organics versus use of the steady-state model.	Moderate		
Eliminating elevated detection limits or use of CRQLs as surrogate concentrations for elevated detection limits in the derivation of the 95th UCL.		Low	
Assessing future residential property use when the likelihood of residential development is low.	High		
The use of total inorganic results for groundwater to evaluate potential chronic daily intakes associated with potable use.	High		
The amount of media intake is assumed to be constant and representative of any actual exposure.			Low
Exclusion of some COPCs, not detected at well locations selected as groundwater exposure point locations, from quantitative determination of CDIs.		Low	

TABLE 7-1 (Continued)

SUMMARY OF UNCERTAINTIES IN THE RESULTS OF THE HUMAN HEALTH AND ECOLOGICAL ASSESSMENT CAMP ALLEN LANDFILL NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

	Potential Magnitude for Over-Estimation of Risks	Potential Magnitude for Under-Estimation of Risks	Potential Magnitude for Over or Under-Estimation of Risks
Toxicological Assessment Toxicological indices derived from high dose animal studies, extrapolated to low dose human exposure.	Moderate		
Lack of promulgated toxicological indices for inhalation pathway.		Low	
Risk Characterization Assumption of additivity in the quantitation of cancer risks without consideration of synergism, antagonism, promotion and initiation.			Moderate
Assumption of additivity in the estimation of systemic health effects without consideration of synergism, antagonism, etc.			Moderate
Additivity of risks by individual exposure pathways (dermal and ingestion and inhalation)			Low
Compounds not quantitatively evaluated.		Low	

Notes:

Low - Assumptions categorized as "low" may effect risk estimates by less than one order of magnitude.

Moderate - Assumptions categorized as "moderate" may effect estimates of risk by between one and two orders of magnitude.

High - Assumptions categorized as "high" may effect estimates of risk by more than two orders of magnitude.

Source: Risk Assessment Guidance for Superfund, Volume 1, Part A: Human Health Evaluation Manual. USEPA, 1989a.

SECTION 8

8.0 SUMMARY OF RISK ASSESSMENT RESULTS

This section summarizes the results of the baseline RA and identifies environmental media and COPCs which could potentially pose human health risks and/or ecological effects.

8.1 Total Site Risk

Risk results from each logical exposure pathway were summed for each receptor to determine the total site risk posed by the Camp Allen Landfill. The following paragraphs present the potential current and future exposure pathways and the subsequent potential total site risk to humans.

8.1.1 Current Potential Receptors and Exposure Pathways

Potential current receptors to COPCs detected in environmental media at the Camp Allen Landfill include:

- Local Adults
- Local Children (ages 7 - 15 years)
- Local Children (ages 1 - 6 years)
- Brig Employees
- Brig Prisoners
- Elementary School Children (ages 6 - 12 years)
- Elementary School Employees

Local adults and older children could potentially access Area A and Area B on a limited basis and contact COPCs in ditch surface waters and sediments. Local adults and younger children to the west of Area A could also be potentially exposed to COPCs in shallow groundwater during non-potable use of groundwaters that are drawn from residential area wells. Inhalation of volatilized COPCs in outdoor ambient air is another pathway of potential concern for these receptors.

Civilian brig employees and brig prisoners could also be potentially exposed to COPCs in environmental media in Area A. Brig employees could potentially be exposed to COPCs in surface soils, surface waters and sediments. Prisoners could potentially be exposed, via inhalation, to

COPCs in surface soils. Both employees and prisoners are also potentially exposed to volatilized organic COPCs in indoor and outdoor air, as well as COPCs adsorbed onto windblown fugitive dust particulates.

Potential current total site risks to these receptors are presented in Table 8-1. All ILCR values are within USEPA's target risk range of 10^{-6} to 10^{-4}, except for that value which was estimated for Brig employees in Area A (1.1×10^{-04}). The target risk range represents the range of potential risks that USEPA generally believes to be acceptable. The exceedance was due to the presence of arsenic in surface soils, and arsenic and Aroclor-1254 in surface water. HI values presented in Table 8-1 for current Brig prisoners and employees exceed 1.0, indicating that noncarcinogenic adverse human health risks may occur. The HI values estimated for the Brig prisoners and employees are 1.3 and 1.1, respectively.

Two district areas of potential exposure exist in Area B. These are the Pond Area (Area B Pond) and the area surrounding the Camp Allen Elementary School (Area B School). Area B Pond is inaccessible and only authorized military personnel enter the area. Adult workers (Brig Facility employees and prisoners) currently maintain Area B Pond and could contact soils, surface waters, and sediments. Area B School is accessible to both children and adults. Local adults working at the school could potentially contact COPCs in soils, surface waters and sediments through maintenance activities. Children attending the school could also contact these media.

Table 8-2 presents the current potential total site risk values for human receptors in Area B. The total site ILCR values fall within USEPA's target risk range for each Area B receptor. HI values are less than 1.0 suggesting that current potential noncarcinogenic adverse health effects will not occur subsequent to Area B exposure.

8.1.2 Future Potential Receptors and Exposure Pathways

Property use at the Camp Allen Landfill will remain the same in the foreseeable future. Future residential development of the Camp Allen Landfill is highly unlikely. However for the sake of conservatism, future residential development and associated potential risks were evaluated. The potential human receptors evaluated for under the future scenarios were:

- Future resident adults
- Future resident children (ages 1-6 years)
- Future construction workers.

Tables 8-3 and 8-4 present the total ILCR and HI values for the future potential residential development of Area A and Area B, respectively. Future potential adults and children residents could be exposed to COPCs in soils, surface waters and sediments. Future development of Area A and Area B shallow or deep groundwaters for potable purposes is unlikely even in the event of future residential development because of the availability of municipal water. However, potential potable exposure to COPCs in groundwater was evaluated for the sake of conservatism. In general, ILCR values for future resident children and adults exceed USEPA's target risk range because of the presence of vinyl chloride, 1,2-dichloroethane, and total arsenic. HI values for both adults and children were greater than 1.0, suggesting that noncarcinogenic adverse health effects could occur. 1,2-Dichloroethene, trichloroethene, 4-methylphenol, arsenic, and manganese were the main contributors to the total HIs in groundwater.

8.2 Ecological Evaluation

The ecological evaluation focused upon three measures of environmental impact from the Camp Allen Landfill: exceedances of state and Federal criteria for surface waters and sediments, the presence and distribution of benthic macroinvertebrates, and a qualitative assessment of terrestrial flora and fauna.

COPCs exceeded Federal criteria and/or State of Virginia Standards in surface waters at sampled locations throughout Areas A and B. NOAA sediment criteria were also exceeded at various locations. These exceedances represent the potential for environmental impacts.

The benthic community analysis indicated potential impacts from both contaminant exposure and natural conditions. High populations of organisms were present in the sediment sampled and a variety of taxa were represented.

In addition, results of the ERA suggest that significant potential effects on terrestrial receptors resulting from Area A or B were not observed at any location. For an urban area, the terrestrial habitats appear to be diverse and productive.

8.3 Conclusions

A quantitative evaluation of current potential human exposure to COPCs detected in environmental media investigated at the Camp Allen Landfill resulted in total site ILCR value in excess of USEPA's target risk range (10^{-6} to 10^{-4}) for Brig employees in Area A who contact Aroclor-1254 in ditch surface waters, and adult workers in Area B. Total risks associated with individual media in Area B did not, however, exceed USEPA's target risk range. In general, the target risk range represents the range of risks that USEPA usually considers to be acceptable. Current potential human exposure did not result in HI value equal to, or exceeding 1.0, indicating that noncarcinogenic adverse human health effects will not occur.

Current property usage at the Camp Allen Landfill will remain unchanged in the foreseeable future. Future potential development of the property in the unlikely event of a base closure would probably be commercial/industrial or recreational because of deed restriction concerning former landfills. However, future residential property development and potential residential exposure were evaluated for the sake of conservatism in evaluating future potential land use. Total ILCR values for future resident adults and children would exceed the target risk range, particularly if on-site shallow and deep groundwaters were developed for potable purposes. Vinyl chloride was responsible for more than 80 percent of the total site ILCR values generated for both shallow and deep groundwaters in Area A, and over 95 percent of the total site ILCR values to the shallow groundwater in Area B. ILCRs estimated for the deep aquifer were within USEPA's acceptable target range.

Total HI values exceeding 1.0 were also derived. The COPCs 4-methylphenol, 1,2-dichloroethene and arsenic contributed approximately 90 percent of the total HI derived for potential potable use of Area A shallow groundwater. Trichloroethene and 1,2-dichloroethene accounted for approximately 70 percent of the total HI for Area A deep groundwater, via the dermal pathway; while unfiltered thallium contributed 99 percent of the total HI via the ingestion pathway.

Total HI values exceeding 1.0 were derived for the future potential residential exposure of children and adults in Area B. The presence of 1,2-dichloroethene, arsenic and manganese in shallow Area B groundwaters accounted for over 70 percent of the total HI for ingestion. The presence of 1,2-dichloroethene and trichloroethene in shallow Area B groundwaters accounted for over 85 percent of the total HI for the dermal pathway. Dissolved arsenic was not detected in the shallow aquifer, however, dissolved manganese was detected at a concentration contributing 43 percent of the total HI for ingestion. 1,2-Dichloroethene contributed approximately 54 percent of the total HI in filtered groundwater. Total HIs of 4.6 and 4.2 were calculated for future residential children and adults potentially exposed to COPCs in Area B deep groundwater. For the ingestion pathway, trichloroethene and 1,2-dichloroethene accounted for only 5 percent of the total HI values. For the dermal pathway, these VOCs accounted for over 75 percent of the total HI values. The presence of arsenic and manganese accounted for more than 90 percent of the total HI for both children and adults via the ingestion pathway. Arsenic and manganese were detected at concentrations accounting for about 5 and 90 percent, respectively, of the total HI estimated for ingestion of Area B filtered deep groundwater, suggesting that the presence of arsenic in deep groundwater is related to the suspended solids in the sample.

Future potential exposure by child residents to soils in Area A accounted for an ILCR value of 1.4×10^{-4}, which exceeds USEPA's target risk range, which is generally considered to be acceptable, due to the presence of arsenic and Aroclor-1260. The HI value for future potential child exposures to this medium was 6.4, indicating that the potential for adverse noncarcinogenic health effects may exist. The presence of arsenic and cadmium in Area A soils were responsible for the elevated HI value.

This risk assessment has been performed in accordance with USEPA guidelines presented in the Risk Assessment Guidance for Superfund (USEPA, 1989a). Risk estimates were derived using conservative exposure inputs, including the reasonable maximum exposure chemical concentrations and upper bound estimates of daily intake. The risks presented herein reflect those associated with Areas A and B at the Camp Allen Landfill.

As a result of USEPA's reasonable maximum exposure to potential current and future potential receptors, the following media and associated chemicals should be evaluated in screening of

remedial alternatives in the Feasibility Study. These chemicals and environmental media were responsible for future potential human health risks. They include:

AREA A

Shallow Groundwater

1,2-Dichloroethene
Vinyl chloride
Trichloroethene

Deep Groundwater

Vinyl chloride
Trichloroethene
1,2-Dichloroethene

Soil

Arsenic
Cadmium

Sediment

Arsenic
Aroclor-1254
Aroclor-1260

AREA B

Shallow Groundwater

1,2-Dichloroethene
Vinyl chloride
Trichloroethene

Deep Groundwater

1,2-Dichloroethene
Arsenic
Benzene

Risk estimates for these chemicals and environmental media may be revisited to support remedial alternatives and strategies in the Feasibility Study. Preliminary cleanup goals and residual risk estimates for these chemicals and media will also be derived to help select the best, cost effective remediation approach.

SECTION 8.0 TABLES

TABLE 8-1

TOTAL SITE ICR AND HI VALUES FOR CURRENT POTENTIAL HUMAN RECEPTORS, AREA A NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Total HI	Total ILCR
Local Adults(1)	5.9×10^{-02}	3.4×10^{-05}
Local Children(2)*	4.5×10^{-01}	6.0×10^{-05}
Brig Employees(3)	$1.3 \times 10^{+00}$	1.1×10^{-04}
Brig Prisoners(4)	$1.1 \times 10^{+00}$	8.1×10^{-06}

Notes: (1) Local adults could potentially be exposed to COPCs by dermal contact and accidental ingestion of shallow groundwater, surface waters, and sediments, as well as inhalation of VOCs in outdoor air.

(2) Local children could potentially be exposed to surface waters, sediments, and shallow groundwaters, as well as inhalation of VOCs in outdoor air. Total site risk values represent potential exposure to surface waters and sediments by older children and total site risk values for younger children potentially exposed to COPCs in residential area shallow groundwater.

(3) Brig employees (civilian) could potentially be exposed to COPCs by dermal contact and accidental ingestion of soils, surface waters, and sediments, as well as the inhalation of VOCs detected in indoor and outdoor air and fugitive dusts.

(4) Brig prisoners could potentially be exposed to COPCs through dermal contact and accidental ingestion of soils, as well as inhalation of VOCs detected in indoor and outdoor air. Prisoners do not generally gain access to the ditches.

* Total HI and ICR values derived by summing the HI and ICR values for younger children (ages 1 to 6 years) and older children (ages 7 to 15 years) potentially exposed to Area A ditch surface waters and sediments.

TABLE 8-2

TOTAL SITE ICR AND HI VALUES FOR CURRENT POTENTIAL HUMAN RECEPTORS, AREA B NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Total HI	Total ILCR
Adult Workers(1)	2.9×10^{-01}	2.7×10^{-05}
Elementary School Children(2)	8.6×10^{-01}	1.5×10^{-05}
Elementary School Workers(3)	4.4×10^{-01}	2.9×10^{-05}

Notes: (1) Adult workers (employees and prisoners) could potentially be exposed to COPCs by dermal contact and accidental ingestion of soils, surface waters, and sediments, as well as inhalation of fugitive dusts and VOCs in outdoor air, in Area B Pond during maintenance activities.

(2) Elementary school children (6 to 12) could potentially be exposed to COPCs by dermal contact and accidental ingestion of soils, surface water, and sediments, as well as the inhalation of fugitive dusts and VOCs in outdoor air, in Area B School.

(3) Elementary school workers could potentially be exposed to COPCs by dermal contact and accidental ingestion of soils, surface waters, and sediments, as well as the inhalation of fugitive dusts and VOCs in outdoor air, in Area B School.

TABLE 8-3

TOTAL SITE ICR AND HI VALUES FOR FUTURE POTENTIAL HUMAN RECEPTORS, AREA A, SHALLOW WELL LOCATION B-20W*
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Total HI	Total ILCR
Resident Adults(1)	$3.0 \times 10^{+02}$ ($2.9 \times 10^{+02}$)	2.7×10^{-01} (2.7×10^{-01})
Resident Children(2)	$6.4 \times 10^{+02}$ ($6.3 \times 10^{+02}$)	1.8×10^{-01} (1.8×10^{-01})
Construction Workers(3)	8.0×10^{-02}	1.3×10^{-06}

Notes: Values in parentheses represent risk values derived using dissolved inorganic constituent results for groundwaters.

(1) Future resident adults could potentially be exposed to COPCs by dermal contact and accidental ingestion of soils, surface waters, and sediments, as well as inhalation of VOCs detected in outdoor air. Potable use of shallow and deep groundwaters were also evaluated. Potential exposure pathways included ingestion, whole body dermal contact, and inhalation of VOCs while showering.

(2) Future resident children could potentially be exposed to COPCs by dermal contact and accidental ingestion of soils, surface waters, and sediments, as well as inhalation of VOCs detected in outdoor air, and by the potable use of shallow and deep groundwaters.

(3) Construction workers could potentially be exposed to COPCs by dermal contact and accidental ingestion of subsurface soils, and the inhalation of fugitive dusts emanating from excavated subsurface soils.

* Total site ICR and HI values presented using shallow well location B-20W since this location was associated with the most elevated risks in Area A.

TABLE 8-4

TOTAL SITE ICR AND HI VALUES FOR FUTURE POTENTIAL HUMAN RECEPTORS, AREA B, SHALLOW WELL LOCATION B-MW11A*
NORFOLK NAVAL BASE, NORFOLK, VIRGINIA

Receptors	Total HI	Total ILCR
Resident Adults(1)	$2.0 \times 10^{+01}$ ($1.1 \times 10^{+01}$)	2.9×10^{-02} (2.8×10^{-02})
Resident Children(2)	$3.5 \times 10^{+01}$ ($2.2 \times 10^{+01}$)	1.4×10^{-02} (1.4×10^{-02})
Construction Workers(3)	7.5×10^{-01}	7.2×10^{-06}

Notes: Values in parentheses represent risk values derived using dissolved inorganic constituent results for groundwaters.

(1) Future resident adults could potentially be exposed to COPCs by dermal contact and accidental ingestion of soils, surface waters, and sediments, as well as inhalation of fugitive dusts and VOCs detected in outdoor air. Potable use of shallow and deep groundwaters were also evaluated. Potential exposure pathways included ingestion, whole body dermal contact, and inhalation of VOCs while showering.

(2) Future resident children could potentially be exposed to COPCs by dermal contact and accidental ingestion of soils, surface waters, and sediments, as well as inhalation of fugitive dusts and VOCs detected in outdoor air, and by the potable use of shallow and deep groundwaters.

(3) Construction workers could potentially be exposed to COPCs by dermal contact and accidental ingestion of subsurface soils, and the inhalation of fugitive dusts emanating from excavated subsurface soils.

* Total site ICR and HI values presented using shallow well location B-MW11A since this location was associated with the most elevated risks in Area B.

SECTION 9

9.0 REFERENCES

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APPENDIX A - COPC SELECTION AND EXPOSURE CONCENTRATION SUMMARY TABLES

APPENDIX A
SELECTION OF COPCs AND DETERMINATION OF EXPOSURE CONCENTRATIONS

TABLE A-1
AREA A SURFACE SOILS
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	Region III Residential Soil COPC Screening Level	Retained as COPC?	Exposure Point Conc. (mg/kg)
SEMIVOLATILES (ug/kg)								
Benzo(a)anthracene	4/5	69 J	77.4	73.59891	168.7732	880	No	NA
Benzo(a)pyrene	4/5	48 J	68.6	77.07983	164.2948	88	No	NA
Benzo(b)fluoranthene	4/5	110 J	97.2	68.11901	181.7699	880	No	NA
Benzo(k)fluoranthene	2/5	51 J	140.6	89.44719	251.6489	8800	No	NA
Chrysene	4/5	76 J	83.6	71.43739	172.2897	88000	No	NA
Fluoranthene	5/5	96 J	54.6	29.72036	91.49791	310000	No	NA
Indeno(1,2,3-cd)pyrene	1/5	25 J	171	82.94577	273.9774	880	No	NA
Phenanthrene	2/5	36 J	133.2	94.4521	250.4625	310000 [3]	No	NA
Pyrene	5/5	89 J	58.6	28.79757	94.35226	230000	No	NA
PESTICIDES/PCBs (ug/kg)								
4,4'-DDD	3/5	6.2 L	3.46	1.749071	5.631477	2700	No	NA
4,4'-DDE	5/5	14	5.16	5.453714	11.9308	1900	No	NA
4,4'-DDT	2/5	3.9 J	2.42	0.882185	3.515235	1900	No	NA
Aldrin	1/5	9.1	2.68	3.591065	7.138317	38	No	NA
alpha-Chlordane	5/5	3.1 K	1.374	1.208131	2.873898	490 [4]	No	NA
Aroclor-1260	4/5	420 L	143.3	168.5258	352.5253	83	Yes	0.353
Dieldrin	2/5	27 K	6.87	11.26203	20.85184	40	No	NA
Endosulfan I	1/5	0.61 J	1.002	0.253515	1.31674	47000 [5]	No	NA
gamma-Chlordane	2/5	3.8 K	1.66	1.210062	3.162295	490 [4]	No	NA
Heptachlor epoxide	2/5	2.4 J	1.248	0.669156	2.078759	70	No	NA
METALS (mg/kg)								
Aluminum	5/5	9880	6936	2279.798	9766.376	7800	Yes	9766
Arsenic	5/5	70	28.82	28.00762	63.59154	0.37 [6]	Yes	63.6
Barium	5/5	1050 J	366.12	414.1284	880.2615	550	Yes	880
Cadmium	3/5	88.9	29.35	36.37815	74.51357	3.9	Yes	74.5
Calcium	5/5	20200	9173.2	9373.811	20810.81	NA	No	NA
Chromium	5/5	121	44.12	45.72578	100.8887	39 [7]	Yes	101
Cobalt	2/5	18.3	5.97	7.433001	15.19809	470	No	NA
Copper	3/5	477	150.4	196.0913	393.8479	290	Yes	394
Iron	5/5	20800	12500	6669.573	20780.29	NA	No	NA
Lead	5/5	683	239.16	272.1819	577.0745	400 [8]	Yes [10]	577
Manganese	5/5	128	84.6	32.08216	124.4301	39	Yes	124
Mercury	3/5	0.77	0.324	0.297035	0.69277	2.3	No	NA
Nickel	4/5	84.1	27.98	33.78975	69.93006	160	No	NA
Thallium	3/5	0.92	0.498	0.379368	0.968986	0..63 [9]	Yes	0.920
Vanadium	5/5	78.7	41.3	26.29905	73.95034	55	Yes	74.0
Zinc	3/5	916	337.8	395.3068	829.8159	2300	No	NA

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] Naphthalene COPC screening level used as a surrogate.
[4] Chlordane COPC screening level used as a surrogate.
[5] Endosulfan COPC screening level used as a surrogate.
[6] COPC screening level is for carcinogenic arsenic.
[7] COPC screening level is for chromium VI.
[8] Soil screening level for residential land use (USEPA, 1994b).
[9] COPC screening level is for thallium carbonate, thallium chloride and thallium sulfate.
[10] Lead retained for qualitative evaluation since no toxicity criteria are established.
NA - Not available.

TABLE A-2
AREA B LANDFILL AND POND SURFACE SOILS
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1,2]	Region III Residential Soil COPC Screening Level	Retained as COPCs?	Exposure Point Conc. (mg/kg)
VOLATILES (ug/kg)								
Methylene chloride	1/4	54	18.75	23.53	55.55	85000	No	NA
2-butanone	1/4	61	19.75	27.51	62.77	4700000	No	NA
SEMIVOLATILES (ug/kg)								
Naphthalene	1/4	29 J	186	117.38	369.58	310000	No	NA
Phenanthrene	1/4	70 J	168.75	70.40	278.86	310000[8]	No	NA
Fluoranthene	3/4	150 J	115.25	96.13	265.60	310000	No	NA
Pyrene	3/4	130 J	110.25	94.22	257.62	230000	No	NA
Butylbenzylphthalate	1/4	17 J	183	122.76	374.99	1600000	No	NA
Benzo(a)anthracene	3/4	75 J	96.75	94.50	244.55	880	No	NA
Chrysene	3/4	73 J	101.25	91.46	244.29	88000	No	NA
Benzo(k)fluoranthene	3/4	38 J	82.75	101.80	241.96	8800	No	NA
Benzo(a)pyrene	3/4	68 J	96.75	93.49	242.97	88	No	NA
Indeno(1,2,3-cd)pyrene	3/4	45 J	85.5	100.05	241.98	880	No	NA
Benzo(g,h,i)perylene	3/4	40 J	83.25	101.63	242.20	310000[8]	No	NA
PESTICIDES/PCBs (ug/kg)								
Aldrin	2/4	5.8 L	2.19	2.41	5.96	38	No	NA
Endosulfan I	4/4	8.4 L	2.91	3.69	8.68	47000[4]	No	NA
4,4'-DDE	3/4	13 L	8.49	4.19	15.03	1900	No	NA
4,4'-DDD	1/4	6.8 L	3.39	2.35	7.06	2700	No	NA
4,4'-DDT	1/4	16 L	5.79	6.83	16.47	1900	No	NA
Alpha-Chlordane	4/4	22 L	7.93	9.77	23.21	490[3]	No	NA
Gamma-Chlordane	2/4	0.78	0.93	0.45	1.63	490[3]	No	NA
Aroclor-1260	4/4	780 L	350.00	297.77	815.71	83	Yes	0.780
METALS (mg/kg)								
Aluminum	4/4	4870	3215.00	1304.31	5254.95	7800	No	NA
Arsenic	4/4	11.6	5.63	4.27	12.30	0.37[5]	Yes	11.6
Barium	2/4	44.4 J	19.59	22.65	55.01	550	No	NA
Cadmium	4/4	20.5	9.33	9.03	23.45	3.9	Yes	20.5
Calcium	4/4	31400 J	10377.50	14197.78	32582.84	NA	No	NA
Chromium	4/4	44.3	23.35	15.08	46.93	39[6]	Yes	44.3
Cobalt	1/4	6.7	2.74	2.65	6.89	470	No	NA
Copper	4/4	87.8	42.63	31.58	92.02	290	No	NA
Iron	4/4	13600	8800.00	4013.61	15077.29	NA	No	NA
Lead	4/4	251	132.80	88.77	271.64	400[7]	No	NA
Manganese	4/4	102	63.45	34.09	116.77	39	Yes	102
Mercury	4/4	0.77	0.36	0.29	0.81	2.3	No	NA
Nickel	3/4	25.3	12.39	10.39	28.64	160[9]	No	NA
Thallium	1/4	0.23	0.16	0.05	0.24	0.63[10]	No	NA
Vanadium	4/4	19.8	15.85	4.02	22.14	55	No	NA
Zinc	1/4	405	103.13	201.25	417.88	2300	No	NA

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] Chlordane RBC used as a surrogate.
[4] Endosulfan RBC used as a surrogate.
[5] COPC screening level is for carcinogenic arsenic.
[6] COPC screening level is for chromium VI.
[7] Soil screening level for residential land use (USEPA, 1994b).
[8] COPC screening level is for naphthalene used.
[9] COPC screening level for soluable salts used.
[10] COPC screening level is for thallium carbonate, thallium chloride and thallium sulfate.
NA Not Applicable

TABLE A-3
AREA B SURFACE SOILS IN THE ELEMENTARY SCHOOL AREA (INCLUDES LOCATION SSB-09)
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1,2]	Region III Residential Soil COPC Screening Level	Retained as COPCs?	Exposure Point Conc. (mg/kg)
PESTICIDES/PCBs (ug/kg)								
Heptachlor epoxide	1/1	1.5 J	1.5	NA	NA	70	No	NA
Dieldrin	1/1	5.2	5.2	NA	NA	40	No	NA
4,4'-DDE	1/1	6.1	6.1	NA	NA	1900	No	NA
4,4'-DDD	1/1	1.6	1.6	NA	NA	2700	No	NA
4,4'-DDT	1/1	1.4 J	1.4	NA	NA	1900	No	NA
Alpha-Chlordane	1/1	2.8	2.8	NA	NA	490[3]	No	NA
Gamma-Chlordane	1/1	1.9	1.9	NA	NA	490[3]	No	NA
Aroclor-1260	1/1	26 J	26	NA	NA	83	No	NA
METALS (mg/kg)								
Aluminum	4/4	9120	6730.00	1951.60	9834.99	NA	No	7.8
Antimony	3/4	7.8 L	5.13	2.99	9.89	3.1	Yes	NA
Arsenic	4/4	25.1 L	9.98	10.36	26.46	0.37[4]	Yes	25.1
Barium	3/4	410 J	161.33	185.02	455.69	550	No	NA
Cadmium	1/4	31.3	8.18	15.42	32.70	3.9	No	NA
Calcium	4/4	19900 J	7982.50	7983.32	20683.96	NA	No	NA
Chromium	4/4	869	225.50	429.01	908.05	39[5]	Yes	869
Cobalt	3/4	7.5	4.25	2.65	8.46	470	No	NA
Copper	3/4	28.3	13.90	11.18	31.69	290	No	NA
Iron	4/4	8700	7197.50	1868.30	10169.97	NA	No	NA
Lead	4/4	213 J	78.15	90.99	222.91	400[6]	No	NA
Magnesium	3/4	1010	624.25	443.43	1329.74	NA	No	NA
Manganese	4/4	61.2	47.65	12.07	66.86	39	Yes	61.2
Mercury	3/4	0.24 K	0.15	0.08	0.27	2.3	No	NA
Nickel	3/4	18.7	10.11	7.11	21.42	160[8]	No	NA
Potassium	3/4	1710	766.00	678.72	1845.85	NA	No	NA
Selenium	2/4	1.2	0.61	0.47	1.36	39	No	NA
Vanadium	4/4	128	50.00	53.79	135.58	55	Yes	128
Zinc	2/4	2570	673.75	1265.38	2686.97	2300	No	NA

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] Chlordane COPC screening level used as a surrogate.
[4] COPC screening level is for carcinogenic arsenic.
[5] COPC screening level is for chromium VI.
[6] Soil screening level for residential land use (USEPA, 1994b).
[7] COPC screening level for soluble salts.
NA Not Applicable

TABLE A-4
AREA A SOIL BORINGS
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	Region III Residential Soil COPC Screening Level	Retained as COPC?	Exposure Point Conc. (mg/kg)
VOLATILES (ug/kg)								
1,1,1-Trichloroethane	1/10	63	11394.95	26401.13	30281.5282	700000	No	NA
1,2-Dichloroethene	1/10	17	11391.6	26402.73	30279.3223	70000 [3]	No	NA
2-Butanone	8/10	91000	12324.75	28311.81	32578.1677	4700000	No	NA
4-Methyl-2-pentanone	1/10	13 B	11391.25	26402.9	30279.0921	630000	No	NA
Acetone	8/10	58000 B	8655.4	18125.61	21621.9175	780000	No	NA
Benzene	1/10	12 J	11391.1	26402.97	30278.9935	22000	No	NA
Carbon disulfide	3/10	22	11391.15	26402.95	30279.0265	780000	No	NA
Ethylbenzene	3/10	45000 J	7455.75	14219.97	17628.2922	780000	No	NA
Methylene chloride	1/10	4 J	11390.3	26403.35	30278.4677	85000	No	NA
Toluene	5/10	3000000	306756.4	946383.4	983770.653	1600000	Yes	984
Xylenes(total)	5/10	340000	41168.85	105765.7	116830.489	16000000	No	NA
SEMIVOLATILES (ug/kg)								
2-Methylnaphthalene	8/10	21000	2873	6407.916	7457.03082	NA	No	NA
2-Methylphenol	2/10	6400 J	1855	2249.317	3464.09407	390000	No	NA
2,4-Dimethylphenol	4/10	41000	5534.5	12623.44	14564.9297	160000	No	NA
4-Methylphenol	1/10	5500 J	1555	1855.468	2882.3461	39000	No	NA
Acenaphthene	5/10	5600	1298.3	1875.578	2640.03215	470000	No	NA
Benzo(a)anthracene	2/10	70 J	1316.5	1443.312	2349.00232	880	No	NA
Benzo(a)pyrene	1/10	70 J	1333.5	1427.587	2354.75324	88	No	NA
Benzo(b)fluoranthene	2/10	85 J	1317.6	1442.273	2349.35875	880	No	NA
Benzo(g,h,i)perylene	1/10	39 J	1330.4	1430.666	2353.85577	310000 [4]	No	NA
Benzo(k)fluoranthene	1/10	51 J	1331.6	1429.467	2354.19804	8800	No	NA
Bis(2-ethylhexyl)phthalate	5/10	17000	3746.6	6103.642	8112.96216	46000	No	NA
Butylbenzylphthalate	1/10	25 J	1334.5	1427.421	2355.63408	1600000	No	NA
Chrysene	3/10	72 J	1297.1	1460.755	2342.08002	88000	No	NA
Dibenzofuran	3/10	1700	1140.6	1264.444	2045.14548	31000	No	NA
Diethylphthalate	3/10	130 J	1283.3	1467.391	2333.02788	6300000	No	NA
Di-n-butylphthalate	9/10	5800 B	1067.7	1775.178	2337.6092	780000	No	NA
Fluoranthene	3/10	150 J	1309.7	1449.115	2346.35358	310000	No	NA
Fluorene	4/10	1300 J	1057.1	1281.447	1973.80874	310000	No	NA
Indeno(1,2,3-cd)pyrene	1/10	42 J	1330.7	1430.366	2353.94073	880	No	NA
Isophorone	1/10	680 K	1371.5	1398.195	2371.72704	670000	No	NA
Naphthalene	7/10	32000	6669.4	10861.27	14439.2246	310000	No	NA
Phenanthrene	5/10	370 J	1034.3	1402.411	2037.5428	310000 [4]	No	NA
Pyrene	3/10	180 J	1310.2	1448.847	2346.66151	230000	No	NA
PESTICIDES/PCBs (ug/kg)								
4,4'-DDD	8/10	20 K	10.998	7.282585	16.2077428	2700	No	NA
4,4'-DDE	6/10	16 J	9.255	8.830265	15.571907	1900	No	NA
4,4'-DDT	1/10	11 K	11.185	9.531412	18.003486	1900	No	NA
alpha-Chlordane	2/10	2.6 L	5.205	5.031757	8.80456844	490 [5]	No	NA
Aroclor-1254	2/10	1600	259.4	480.8674	603.398104	83	Yes	0.603
Aroclor-1260	5/10	1800	475.05	660.5477	947.586012	83	Yes	0.948
delta-BHC	1/10	1.4 K	5.145	5.070363	8.77218584	350	No	NA
Dieldrin	7/10	89 K	23.773	31.39954	46.2352916	40	Yes	0.046
Endosulfan I	4/10	15 K	5.464	5.47162	9.37823298	47000 [6]	No	NA
Endosulfan II	1/10	3.1 K	9.76	10.16928	17.0348008	47000 [6]	No	NA
Endrin	1/10	2.7 J	10.19	10.02848	17.3640751	2300	No	NA
Endrin aldehyde	4/10	34 K	11.525	11.24241	19.5674838	NA	No	NA
Heptachlor epoxide	1/10	2.7 K	5.275	4.979528	8.83720471	70	No	NA

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] RBC is for mixture of cis- and trans- isomers.
[4] Naphthalene RBC used as a surrogate
[5] Chlordane RBC used as a surrogate.
[6] Endosulfan RBC used as a surrogate.
NA - Not available.

TABLE A-5
AREA B SOIL BORINGS
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	Region III Residential Soil COPC Screening Level	Retained as COPC?	Exposure Point Conc. (mg/kg)
VOLATILES (ug/kg)								
1,1-Dichloroethane	1/10	12	832	2354.453	2516.157	780000	No	NA
1,1,1-Trichloroethane	1/10	8 J	831.1	2354.8	2515.506	700000	No	NA
1,2-Dichloroethane	2/10	25 J	831.9	2354.495	2516.087	7000	No	NA
1,2-Dichloroethene	4/10	4300	1233.5	2575.691	3075.91	70000 [3]	No	NA
2-Butanone	7/10	10000 J	1234.65	3155.29	3491.652	4700000	No	NA
4-methyl-2-pentanone	4/10	2200	1016.3	2376.79	2716.435	630000	No	NA
Acetone	9/10	6000 J	834.1	1906.028	2197.496	780000	No	NA
Benzene	2/10	250	856.4	2346.196	2534.651	22000	No	NA
Ethylbenzene	1/10	30000	3081.4	9461.107	9848.998	780000	No	NA
Methylene chloride	1/10	200	850.8	2347.92	2530.284	85000	No	NA
Toluene	4/10	16000	1694.9	5031.544	5294	1600000	No	NA
Trichloroethene	2/10	3100	1067.1	2460.217	2826.911	58000	Yes	2.83
Vinyl chloride	1/10	16	832.4	2354.299	2516.447	340	Yes	0.016
Xylenes(total)	3/10	200000	20081.1	63217.49	65301.02	16000000	No	NA
SEMIVOLATILES (ug/kg)								
1,2-Dichlorobenzene	1/10	6500	915.5	1978.189	2330.513	700000	No	NA
2-Methylnaphthalene	1/10	3300 J	595.5	982.8684	1298.553	NA	No	NA
2-Methylphenol	2/10	180 J	363.6	560.4974	764.5279	390000	No	NA
4-Methylphenol	1/10	650 J	425.5	554.2735	821.9759	39000	No	NA
Bis(2-ethylhexyl)phthalate	1/10	22 J	443.7	591.4823	866.7916	46000	No	NA
Diethylphthalate	6/10	95 J	368.1	631.0255	819.4771	6300000	No	NA
Di-n-butylphthalate	10/10	830 J	380.9	216.7264	535.9259	780000	No	NA
Naphthalene	1/10	14000	1665.5	4341.167	4770.768	310000	No	NA
Phenanthrene	1/10	230 J	288.5	251.8934	468.6812	310000 [4]	No	NA
Phenol	3/10	13000	1626.3	4036.333	4513.518	4700000	No	NA
PESTICIDES/PCBs (ug/kg)								
4,4'-DDD	1/10	3800	381.855	1201.014	1240.949	2700	Yes	1.24
4,4'-DDE	1/10	14 J	3.255	3.787292	5.964078	1900	No	NA
Aroclor-1254	1/10	9500	968.55	2997.647	3112.789	83	Yes	3.11
Dieldrin	1/10	1500	151.855	473.69	490.6888	40	Yes	0.491
Endosulfan I	1/10	78	8.73	24.33945	26.14019	47000 [5]	No	NA
Endosulfan II	1/10	17 J	3.555	4.733594	6.940974	47000 [5]	No	NA
Endrin aldehyde	1/10	12 J	3.055	3.157218	5.313381	NA	No	NA
METALS (mg/kg)								
Aluminum	10/10	15500	8320	4237.502	11351.12	7800	Yes	11351
Antimony	8/10	8 L	3.762	2.082039	5.251297	3.1	Yes	5.25
Arsenic	10/10	60.5 J	7.682	18.61861	21.00002	0.37	Yes	21.0
Barium	10/10	1480	167.465	461.3165	497.448	550	Yes	497
Beryllium	8/10	5.6	0.937	1.641448	2.111139	0.15	Yes	2.11
Cadmium	8/10	1.3	0.69725	0.260145	0.883334	3.9	No	NA
Calcium	10/10	9290	1419.8	2779.874	3408.264	NA	No	NA
Chromium	10/10	24.9	9.21	6.854755	14.11326	39 [6]	No	NA
Cobalt	8/10	16.2	3.1375	4.607832	6.433516	470	No	NA
Copper	10/10	63.6	8.82	19.29938	22.62499	290	No	NA
Iron	10/10	22700	7645.5	6376.647	12205.76	NA	No	NA
Lead	10/10	19.8 J	6.245	5.172494	9.944922	400 [7]	No	NA
Magnesium	10/10	2180	660.05	561.567	1061.743	NA	No	NA
Manganese	10/10	63.5	21.225	18.6641	34.57556	39	Yes	34.6

TABLE A-5
AREA B SOIL BORINGS
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA
(CONTINUED)

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	Region III Residential Soil COPC Screening Level	Retained as COPC?	Exposure Point Conc. (mg/kg)
METALS (mg/kg)								
Mercury	8/10	0.68	0.15875	0.18487	0.290988	2.3	No	NA
Nickel	10/10	38.7	7.3975	11.03296	15.28945	160	No	NA
Potassium	10/10	2230	577.1	591.9768	1000.545	NA	No	NA
Selenium	6/10	5.7 J	0.90125	1.690297	2.110332	39	No	NA
Silver	7/10	0.49	0.4135	0.101244	0.48592	39	No	NA
Sodium	10/10	1250	443.925	331.4174	680.9902	NA	No	NA
Thallium	10/10	2	0.686	0.466814	1.019916	0.63 [8]	Yes	1.02
Vanadium	10/10	149	23.8	44.52637	55.65003	55	Yes	55.7
Zinc	10/10	47.9 J	16.265	12.94244	25.52282	2300	No	NA

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] COPC screening level is for mixture of cis- and trans- isomers.
[4] Naphthalene COPC screening level used as a surrogate.
[5] Endosulfan COPC screening level used as a surrogate.
[6] COPC screening level is for chromium VI.
[7] Soil screening level for residential land use (USEPA, 1994b).
[8] COPC screeing level is for thallium carbonate, thallium chloride and thallium sulfate.
NA - Not available.

TABLE A-6
AREA A - SHALLOW AQUIFER GROUNDWATER
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Region III Tapwater COPC Screening Level [1]	MCLs State of Virginia	MCLs Federal SDWA	Retained as COPC?	Exposure Point Conc. (mg/L) B-20W [2]	Exposure Point Conc. (mg/L) B-20WSS [2]
VOLATILES (ug/L)								
1,2-Dichloroethane	1/18	3 J	0.12	NA	5	Yes	ND	ND
1,2-Dichloroethene	4/18	6100	5.5 [3]	NA	70	Yes	6.1	1.6
2-Butanone	1/18	4300	190	NA	NA	Yes	4.3	ND
4-methyl-2-pentanone	2/18	16000	290	NA	NA	Yes	16	0.22
Acetone	2/18	2600	370	NA	NA	No	NA	NA
Benzene	1/18	310 J	0.36	NA	5	Yes	0.31	ND
Methylene chloride	2/18	57 J	4.1	NA	5	Yes	ND	0.057
Tetrachloroethene	2/18	620	1.1	NA	5	Yes	ND	0.62
Toluene	2/18	5400	75	NA	1000	Yes	5.4	1.2
Trichloroethene	2/18	1800	1.6	NA	5	Yes	ND	1.8
Vinyl chloride	3/18	3300	0.019	NA	2	Yes	3.3	ND
Xylenes(total)	2/18	250 J	1200	NA	10000	No	NA	NA
SEMIVOLATILES (ug/L)								
2-Methylphenol	2/17	1800 J	180	NA	NA	Yes	1.8	0.28
2,4-Dimethylphenol	2/17	1400 J	73	NA	NA	Yes	1.4	0.025
4-Methylphenol	2/17	21000	18	NA	NA	Yes	21	0.1
Acenaphthene	1/17	1 J	220	NA	NA	No	NA	NA
Bis(2-ethylhexyl)phthalate	6/17	13	4.8	NA	6	Yes	ND	ND
Diethylphthalate	6/17	6 J	2900	NA	NA	No	NA	NA
Di-n-butylphthalate	3/17	0.9 J	370	NA	NA	No	NA	NA
Naphthalene	1/17	4 J	150	NA	NA	No	NA	NA
Phenol	1/17	1800 J	2200	NA	NA	No	NA	NA
PESTICIDES/PCBs (ug/L)								
4,4'-DDD	1/17	0.11 L	0.28	NA	NA	No	NA	NA
Aldrin	1/17	0.026 J	0.004	0.003	NA	Yes	ND	ND
gamma-Chlordane	1/17	0.007 J	0.052 [4]	0.01	2	No	NA	NA
Heptachlor epoxide	3/17	0.14 L	0.0012	0.001	0.2	Yes	0.00014	ND
UNFILTERED METALS (ug/L								
Aluminum	17/17	132000	3700	NA	NA	Yes	80.6	132
Antimony	1/17	31	1.5	NA	6	Yes	ND	ND
Arsenic	16/17	309	0.038 [5]	50	50	Yes	0.2	0.0681
Barium	17/17	7270	260	1000	2000	Yes	7.27	0.57
Beryllium	3/17	10.6	0.016	NA	4	Yes	0.0106	0.0067
Cadmium	3/17	45.9	1.8	10	5	Yes	0.0459	ND
Calcium	17/17	337000	NA	NA	NA	No	NA	NA
Chromium	14/17	353	18 [6]	50	100	Yes	0.187	0.353
Cobalt	4/17	84.2	220	NA	NA	No	NA	NA
Copper	15/17	356	140	1000	1300	Yes	0.279	0.183
Iron	17/17	226000	NA	NA	NA	No	NA	NA
Lead	13/17	381 L	NA	50	15	Yes [7]	0.298	0.381
Magnesium	17/17	260000	NA	NA	NA	No	NA	NA
Manganese	17/17	2060 J	18	NA	NA	Yes	2.76	2.06
Mercury	5/17	0.52	1.1	0.05	2	No	NA	NA
Nickel	10/17	352	73	NA	100	Yes	0.114	0.352
Potassium	17/17	120000	NA	NA	NA	No	NA	NA
Silver	1/17	5	18	NA	NA	No	NA	NA
Sodium	17/17	2140000	NA	NA	NA	No	NA	NA
Vanadium	16/17	396	26	NA	NA	Yes	0.396	0.344
Zinc	16/17	1090 J	1100	50 [8]	NA	No	NA	NA

TABLE A-6
AREA A - SHALLOW AQUIFER GROUNDWATER
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA
(CONTINUED)

Parameter	Frequency of Detection	Maximum Detected Conc.	Region III Tapwater COPC Screening Level [1]	MCLs State of Virginia	MCLs Federal SDWA	Retained as COPC?	Exposure Point Conc. (mg/L) B-20W [2]	Exposure Point Conc. (mg/L) B-20WSS [2]
FILTERED METALS (ug/L)								
Dissolved Arsenic	11/17	200 L	0.038 [5]	50	50	Yes	0.2	0.0118
Dissolved Barium	15/17	6060	260	1000	2000	Yes	6.06	ND
Dissolved Calcium	17/17	353000	NA	NA	NA	No	NA	NA
Dissolved Copper	5/17	19.2	140	1000	1300	No	NA	NA
Dissolved Iron	14/17	55400	NA	NA	NA	No	NA	NA
Dissolved Lead	1/17	1.6	NA	50	15	Yes [7]	ND	ND
Dissolved Magnesium	16/17	242000	NA	NA	NA	No	NA	NA
Dissolved Manganese	16/17	2630	18	NA	NA	Yes	2.63	0.198
Dissolved Nickel	1/17	63.8	73	NA	100	No	NA	NA
Dissolved Potassium	17/17	109000	NA	NA	NA	No	NA	NA
Dissolved Sodium	17/17	2360000	NA	NA	NA	No	NA	NA
Dissolved Vanadium	2/17	5.3	26	NA	NA	No	NA	NA
Dissolved Zinc	7/17	61.3	1100	50 [8]	NA	No	NA	NA

Notes:
[1] Derived based on age-adjusted ingestion and inhalation exposures to contaminants in tap water.
[2] Maximum values from Rounds 2 and 3 were selected as exposure point concentrations.
[3] COPC screening level is for mixture of cis- and trans- isomers.
[4] Chlordane COPC screening level used as a surrogate.
[5] COPC screening level is for carcinogenic arsenic.
[6] COPC screening level is for chromium VI.
[7] Lead retained for qualitative evaluation since no toxicity criteria are established.
[8] Virginia groundwater standard.
NA - Not applicable.
ND - Not detected.

TABLE A-7
AREA B - SHALLOW AQUIFER GROUNDWATER
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Region III Tapwater COPC Screening Level [1]	MCLs State of Virginia	MCLs Federal SDWA	Retained as COPC?	Exposure Point Conc. (mg/L) B-MW11A [2]
VOLATILES (ug/L)							
1,1-Dichloroethene	3/26	51	0.044	NA	7	Yes	ND
1,1,1-Trichloroethane	1/26	30 J	130	NA	200	No	NA
1,2-Dichloroethane	6/26	180	0.12	NA	5	Yes	0.058
1,2-Dichloroethene	5/26	1600	5.5 [3]	NA	70	Yes	1.6
4-Methyl-2-pentanone	1/26	525 J	290	NA	NA	No	NA
Acetone	2/26	1250	370	NA	NA	No	NA
Benzene	4/26	410	0.36	NA	5	Yes	0.029
Chlorobenzene	1/26	48	3.9	NA	NA	Yes	ND
Ethylbenzene	1/26	18	130	NA	700	No	NA
Tetrachloroethene	3/26	10 J	1.1	NA	5	Yes	ND
Trichloroethene	5/26	520	1.6	NA	5	Yes	0.044
Vinyl chloride	5/26	940 J	0.019	NA	2	Yes	0.94
Xylenes(total)	2/26	140	1200	NA	10000	No	NA
SEMIVOLATILES (ug/L)							
1,2-Dichlorobenzene	3/19	16	37	NA	600	No	NA
1,4-Dichlorobenzene	2/19	3 J	0.44	NA	75	Yes	0.003
2-Methylnaphthalene	3/19	8 J	NA	NA	NA	No	NA
2,2'-Oxybis(1-chloropropane)	1/19	4 J	NA	NA	NA	No	NA
2,4-Dimethylphenol	1/19	0.6 J	73	NA	NA	No	NA
4-Methylphenol	1/19	13	18	NA	NA	No	NA
Acenaphthene	2/19	76	220	NA	NA	No	NA
Bis(2-chloroethyl)ether	1/19	8 J	0.0092	NA	NA	Yes	ND
Bis(2-ethylhexyl)phthalate	5/19	5 J	4.8	NA	6	Yes	ND
Dibenzofuran	1/19	7 J	15	NA	NA	No	NA
Diethylphthalate	7/19	2 J	2900	NA	NA	No	NA
Fluorene	1/19	0.5 J	150	NA	NA	No	NA
Naphthalene	4/19	4 J	150	NA	NA	No	NA
N-Nitrosodiphenylamine	1/19	1 J	14	NA	NA	No	NA
Phenanthrene	1/19	0.9 J	150 [4]	NA	NA	No	NA
Phenol	7/19	14	2200	NA	NA	No	NA
Pyrene	1/19	0.8 J	110	NA	NA	No	NA
PESTICIDES/PCBs (ug/L)							
4,4'-DDD	1/19	0.14	0.28	NA	NA	No	NA
4,4'-DDE	2/19	0.047 J	0.2	NA	NA	No	NA
4,4'-DDT	1/18	0.015 J	0.2	0.001	NA	No	NA
alpha-BHC	1/19	0.005 J	0.011	NA	NA	No	NA
delta-BHC	1/19	0.014 J	0.037	NA	NA	No	NA
Dieldrin	5/19	0.043 J	0.0042	0.003	NA	Yes	ND
Endrin	1/19	0.031 J	1.1	0.004	2	No	NA
Endrin aldehyde	1/19	0.009 J	NA	NA	NA	No	NA
gamma-BHC	1/19	0.15	0.052	0.01	0.2	Yes	ND
Heptachlor epoxide	2/19	0.006 J	0.0012	0.001	0.2	Yes	ND
UNFILTERED METALS (ug/L)							
Aluminum	19/19	192000	3700	NA	NA	Yes	88.8
Antimony	2/19	28.7	1.5	NA	6	Yes	ND
Arsenic	15/19	93.6	0.038 [5]	50	50	Yes	0.032
Barium	19/19	1740	260	1000	2000	Yes	0.210
Beryllium	7/19	18.5	0.016	NA	4	Yes	0.0041

TABLE A-7
AREA B - SHALLOW AQUIFER GROUNDWATER
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA
(CONTINUED)

Parameter	Frequency of Detection	Maximum Detected Conc.	Region III Tapwater COPC Screening Level [1]	MCLs State of Virginia	MCLs Federal SDWA	Retained as COPC?	Exposure Point Conc. (mg/L) B-MW11A [2]
UNFILTERED METALS (ug/L)							
Cadmium	4/19	17.8	1.8	10	5	Yes	ND
Calcium	19/19	192000	NA	NA	NA	No	NA
Chromium	13/19	774.5	18 [6]	50	100	Yes	0.141
Cobalt	8/19	202.5	220	NA	NA	No	NA
Copper	18/19	380	140	1000	1300	Yes	0.0592
Iron	19/19	734500	NA	NA	NA	No	NA
Lead	17/19	1020	NA	50	15	Yes [7]	0.0448
Magnesium	19/19	126500	NA	NA	NA	No	NA
Manganese	19/19	4880	18	NA	NA	Yes	0.906
Mercury	6/19	3	1.1	0.05	2	Yes	ND
Nickel	11/19	433	73	NA	100	Yes	0.0276
Potassium	19/19	45900 J	NA	NA	NA	No	NA
Silver	3/19	2.4	18	NA	NA	No	NA
Sodium	19/19	222000	NA	NA	NA	No	NA
Vanadium	15/19	1610	26	NA	NA	Yes	0.297
Zinc	14/19	1550 J	1100	50 [8]	NA	Yes	0.231
FILTERED METALS (ug/L)							
Dissolved Aluminum	1/19	201	3700	NA	NA	No	NA
Dissolved Antimony	1/19	32.9	1.5	NA	6	Yes	ND
Dissolved Arsenic	6/19	16.4	0.038 [5]	50	50	Yes	ND
Dissolved Barium	16/19	96.5	260	1000	2000	No	NA
Dissolved Calcium	19/19	146000	NA	NA	NA	No	NA
Dissolved Chromium	2/19	22.2	18 [6]	50	100	Yes	ND
Dissolved Cobalt	3/19	13.55	220	NA	NA	No	NA
Dissolved Copper	5/19	10.5	140	1000	1300	No	NA
Dissolved Iron	19/19	64600	NA	NA	NA	No	NA
Dissolved Magnesium	19/19	40150	NA	NA	NA	No	NA
Dissolved Manganese	19/19	1385	18	NA	NA	Yes	0.715
Dissolved Potassium	19/19	20500	NA	NA	NA	No	NA
Dissolved Sodium	19/19	227000	NA	NA	NA	No	NA
Dissolved Vanadium	8/19	29.9	26	NA	NA	Yes	0.004
Dissolved Zinc	2/19	22	1100	50 [8]	NA	No	NA

Notes:
[1] Derived based on age-adjusted ingestion and inhalation exposures to contaminants in tap water.
[2] Maximum values from Rounds 2 and 3 were selected as exposure point concentrations.
[3] COPC screening level is for mixture of cis- and trans- isomers.
[4] Naphthalene COPC screening level used as a surrogate.
[5] COPC screening level is for carcinogenic arsenic.
[6] COPC screening level is for chromium VI.
[7] Lead retained for qualitative evaluation since no toxicity criteria are established.
[8] Virginia groundwater standard.
NA - Not applicable.
ND - Not detected.

TABLE A-8
AREA A - YORKTOWN AQUIFER (DEEP) GROUNDWATER
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Region III Tapwater COPC Screening Level [1]	MCLs State of Virginia	MCLs Federal SDWA	Retained as COPC?	Exposure Point Conc. (mg/L) A-MW1B [2]
VOLATILES (ug/L)							
1,2-Dichloroethane	4/32	38 J	0.12	NA	5	Yes	0.038
1,2-Dichloroethene	18/32	540	5.5 [3]	NA	70	Yes	0.54
2-Butanone	1/32	2 J	2200	NA	NA	No	NA
Benzene	2/32	3 J	0.36	NA	5	Yes	ND
Chloroform	2/32	8	0.15	NA	100	Yes	ND
Tetrachloroethene	1/32	4	1.1	NA	5	Yes	ND
Toluene	1/32	1 J	75	NA	1000	No	NA
Trichloroethene	10/32	100	1.6	NA	5	Yes	0.1
Vinyl chloride	10/32	100	0.019	NA	2	Yes	0.1
Xylenes(total)	1/32	1 J	1200	NA	10000	No	NA
SEMIVOLATILES (ug/L)							
1,2,4-Trichlorobenzene	1/17	0.5 J	19	NA	70	No	NA
2,2'-Oxybis(1-chloropropane)	1/17	4.5 J	NA	NA	NA	No	NA
Bis(2-chloroethyl)ether	1/17	2 J	0.0092	NA	NA	Yes	ND
Bis(2-ethylhexyl)phthalate	7/17	3.5 J	4.8	NA	6	No	NA
Diethylphthalate	2/17	5.3 J	2900	NA	NA	No	NA
Di-n-butylphthalate	4/17	2 J	370	NA	NA	No	NA
Naphthalene	2/17	1 J	150	NA	NA	No	NA
Phenol	3/16	7.5 J	2200	NA	NA	No	NA
PESTICIDES/PCBs (ug/L)							
4,4'-DDT	1/17	0.016 J	0.2	0.001	NA	No	NA
Heptachlor epoxide	2/17	0.0065 J	0.0012	0.001	0.2	Yes	ND
UNFILTERED METALS (ug/L)							
Aluminum	14/17	49600	3700	NA	NA	Yes	8.56
Arsenic	15/17	64.35 L	0.038 [4]	50	50	Yes	0.0193
Barium	17/17	243.5 J	260	1000	2000	No	NA
Cadmium	1/17	6.5	1.8	10	5	Yes	ND
Calcium	17/17	461000	NA	NA	NA	No	NA
Chromium	9/17	165.5	18 [5]	50	100	Yes	0.0259
Copper	7/17	56.7	140	1000	1300	No	NA
Iron	16/17	248500	NA	NA	NA	No	NA
Lead	12/17	44.2	NA	50	15	Yes [7]	0.0098
Magnesium	16/17	40200	NA	NA	NA	No	NA
Manganese	15/17	2170	18	NA	NA	Yes	0.49
Mercury	2/17	0.34	1.1	0.05	2	No	NA
Nickel	3/17	50.65	73	NA	100	No	NA
Potassium	17/17	25400	NA	NA	NA	No	NA
Sodium	17/17	582000	NA	NA	NA	No	NA
Thallium	1/17	6 L	0.29 [6]	NA	2	Yes	ND
Vanadium	8/17	355.5	26	NA	NA	Yes	0.064
Zinc	14/17	367	1100	50 [8]	NA	No	NA
FILTERED METALS (ug/L)							
Dissolved Aluminum	1/17	693	3700	NA	NA	No	NA
Dissolved Arsenic	6/17	4.9	0.038 [4]	50	50	Yes	ND
Dissolved Barium	16/17	155	260	1000	2000	No	NA
Dissolved Calcium	17/17	336000	NA	NA	NA	No	NA
Dissolved Copper	3/17	5.1	140	1000	1300	No	NA

TABLE A-8
AREA A - YORKTOWN AQUIFER (DEEP) GROUNDWATER
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA
(CONTINUED)

Parameter	Frequency of Detection	Maximum Detected Conc.	Region III Tapwater COPC Screening Level [1]	MCLs State of Virginia	MCLs Federal SDWA	Retained as COPC?	Exposure Conc. (mg/L) A-MW1B [2]
FILTERED METALS (ug/L)							
Dissolved Iron	6/17	2720	NA	NA	NA	No	NA
Dissolved Magnesium	15/17	37700	NA	NA	NA	No	NA
Dissolved Manganese	13/17	284	18	NA	NA	Yes	0.244
Dissolved Potassium	17/17	25700	NA	NA	NA	No	NA
Dissolved Sodium	17/17	513500	NA	NA	NA	No	NA
Dissolved Zinc	8/17	34	1100	50 [8]	NA	No	NA

Notes:
[1] Derived based on age-adjusted ingestion and inhalation exposures to contaminants in tap water.
[2] Maximum values from Rounds 2 and 3 were selected as exposure point concentrations.
[3] COPC screening level is for mixture of cis- and trans- isomers.
[4] COPC screening level is for carcinogenic arsenic.
[5] COPC screening level is for chromium VI.
[6] COPC screening level is for thallium carbonate, thallium chloride and thallium sulfate.
[7] Lead retained for qualitative evaluation since no toxicity criteria are established.
[8] Virginia groundwater standard.
NA - Not applicable.
ND - Not detected.

TABLE A-9
AREA B - YORKTOWN AQUIFER (DEEP) GROUNDWATER
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Region III Tapwater COPC Screening Level [1]	MCLs State of Virginia	MCLs Federal SDWA	Retained as COPC?	Exposure Point Conc. (mg/L) B-MW19B [2]
VOLATILES (ug/L)							
1,2-Dichloroethane	6/13	450	0.12	NA	5	Yes	0.003
1,2-Dichloroethene	7/13	16	5.5 [3]	NA	70	Yes	0.016
Benzene	1/13	12	0.36	NA	5	Yes	0.012
Chloroform	1/13	1 J	0.15	NA	100	Yes	0.001
Toluene	1/13	1 J	75	NA	1000	No	NA
Trichloroethene	6/13	35	1.6	NA	5	Yes	0.018
Vinyl chloride	2/13	3	0.019	NA	2	Yes	ND
Xylenes(total)	1/13	1 J	1200	NA	10000	No	NA
SEMIVOLATILES (ug/L)							
Diethylphthalate	3/7	1.9 J	2900	NA	NA	No	NA
Phenol	3/7	6 J	2200	NA	NA	No	NA
PESTICIDES/PCBs (ug/L)							
4,4'-DDD	1/7	0.018 J	0.28	NA	NA	No	NA
Dieldrin	1/7	0.009 J	0.0042	0.003	NA	Yes	ND
Heptachlor epoxide	1/7	0.0105 J	0.0012	0.001	0.2	Yes	ND
UNFILTERED METALS (ug/L)							
Aluminum	6/7	146000	3700	NA	NA	Yes	ND
Antimony	3/7	25.2 L	1.5	NA	6	Yes	ND
Arsenic	5/7	194 L	0.038 [4]	50	50	Yes	0.0027
Barium	7/7	569	260	1000	2000	Yes	0.0986
Beryllium	2/7	11.2	0.016	NA	4	Yes	ND
Cadmium	2/7	30.8	1.8	10	5	Yes	ND
Calcium	7/7	705000	NA	NA	NA	No	NA
Chromium	3/7	542 K	18 [5]	50	100	Yes	ND
Cobalt	2/7	181	220	NA	NA	No	NA
Copper	3/7	225	140	1000	1300	Yes	ND
Iron	7/7	428000	NA	NA	NA	No	NA
Lead	3/7	183	NA	50	15	Yes [6]	ND
Magnesium	7/7	41800	NA	NA	NA	No	NA
Manganese	7/7	4740 K	18	NA	NA	Yes	0.373
Nickel	3/7	203	73	NA	100	Yes	ND
Potassium	7/7	15700	NA	NA	NA	No	NA
Silver	2/7	3.2	18	NA	NA	No	NA
Sodium	7/7	149000	NA	NA	NA	No	NA
Vanadium	5/7	769 K	26	NA	NA	Yes	ND
Zinc	5/7	985 K	1100	50 [7]	NA	No	NA
FILTERED METALS (ug/L)							
Dissolved Arsenic	1/7	1.3	0.038 [4]	50	50	Yes	0.0013
Dissolved Barium	7/7	94.1	260	1000	2000	No	NA
Dissolved Calcium	7/7	197000	NA	NA	NA	No	NA
Dissolved Iron	4/7	6450	NA	NA	NA	No	NA
Dissolved Magnesium	7/7	13200 J	NA	NA	NA	No	NA
Dissolved Manganese	7/7	356	18	NA	NA	Yes	0.356
Dissolved Potassium	7/7	6080	NA	NA	NA	No	NA
Dissolved Sodium	7/7	155000	NA	NA	NA	No	NA
Dissolved Vanadium	5/7	4	26	NA	NA	No	NA

Notes:
[1] Derived based on age-adjusted ingestion and inhalation exposures to contaminants in tap water.
[2] Maximum values from Rounds 2 and 3 were selected as exposure point concentrations.
[3] COPC screening level is for mixture of cis- and trans- isomers.
[4] COPC screening level is for carcinogenic arsenic.
[5] COPC screening level is for chromium VI.
[6] Lead retained for qualitative evaluation since no toxicity criteria are established.
[7] Virginia groundwater standard.
NA - Not applicable.

TABLE A-10
AREA A - SURFACE WATER
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	Federal Ambient Water Quality Criteria: Water and Organisms [3]	Federal Ambient Water Quality Criteria: Organisms Only [3]	Virginia Water Quality Standards: Public Water Supplies [5]	Virginia Water Quality Standards: Other Surface Waters [6]
VOLATILES (ug/L)									
1,2-Dichloroethene	2/10	20	6.2	4.894441	9.701029	700	140000	NA	NA
Acetone	1/10	9 B	5.4	1.264911	6.3048	NA	NA	NA	NA
Tetrachloroethene	1/10	6 J	5.1	0.316228	5.3262	0.8	8.85	318	3519
Trichloroethene	3/10	20	6	5.033223	9.600301	3.1	92	27	807
Vinyl chloride	1/10	6 J	5.1	0.316228	5.3262	2	525	20	5250
Xylenes(total)	1/10	3 J	4.8	0.632456	5.2524	NA	NA	NA	NA
SEMIVOLATILES (ug/L)									
Acenaphthene	1/10	0.8 J	4.57	1.359779	5.54266	1200	2700	NA	NA
Bis(2-ethylhexyl)phthalate	5/10	3 J	3.58	1.615068	4.73527	1.8	5.9	18	59
Di-n-butylphthalate	3/10	1 J	3.47	2.043445	4.931691	2700	12000	NA	NA
PESTICIDES/PCBs (ug/L)									
4,4'-DDD	6/10	0.26 L	0.0689	0.08381	0.12885	NA	NA	NA	NA
4,4'-DDE	1/10	0.069 J	0.0519	0.006008	0.056198	0.00059	0.00059	NA	NA
alpha-BHC	1/10	0.006 J	0.0231	0.006008	0.027398	0.0039	0.013	7	25
alpha-Chlordane	1/10	0.015 J	0.024	0.003162	0.026262	0.00058	0.00059	0.0058	0.0059
Aroclor-1254	1/10	0.44 J	0.494	0.018974	0.507572	0.000044	0.000045	0.00044	0.00045
delta-BHC	1/10	0.025 J	0.025	0	0.025	0.0123 [P]	0.0414 [P]	7	25
Dieldrin	1/10	0.027 J	0.0477	0.007273	0.052903	0.00014	0.00014	0.0014	0.0014
Endrin	1/10	0.07 J	0.052	0.006325	0.056524	0.46	0.49	0.76	0.91
Endrin aldehyde	1/10	0.047 J	0.0497	0.000949	0.050379	0.46	0.46	0.76	0.91
gamma-BHC	1/10	0.013 J	0.0238	0.003795	0.026514	0.019	0.063	7	25
gamma-Chlordane	1/10	0.024 J	0.0249	0.000316	0.025126	0.00058	0.00059	0.0058	0.0059
Heptachlor epoxide	2/10	0.006 J	0.0209	0.008672	0.027103	0.0002	0.0002	0.0021	0.0021
UNFILTERED METALS (ug/L)									
Aluminum	5/10	20300 J	2326.6	6326.907	6852.282	87 [F]	87 [F]	NA	NA
Arsenic	3/10	64.2	9.9	19.905	24.13819	0.018	0.14	50	NA
Barium	8/10	409	79.23	120.1235	165.1552	1000 [P]	NA	2000	NA
Calcium	10/10	119000	94040	32497.08	117285.4	NA	NA	NA	NA
Chromium	2/10	104	14.8	31.44236	37.29095	160	3200	170	3400
Cobalt	1/10	13.2	4.92	2.909295	7.00104	NA	NA	NA	NA
Copper	3/10	446	46.45	140.4018	146.8804	1300	NA	1300	NA
Iron	10/10	78300	11106	23695.33	28055.44	300 [P]	NA	NA	NA
Lead	4/10	800	81.05	252.6152	261.7474	50 [P]	NA	15	NA
Magnesium	10/10	375000	130550	114747	212629.3	NA	NA	NA	NA
Manganese	10/10	697	253.96	163.0506	370.5912	50 [P]	100 [P]	50	NA
Mercury	2/10	3.9	0.492	1.198831	1.349532	0.14	0.15	0.144	0.146
Nickel	1/10	57	10.65	16.28573	22.2993	510	3800	607	4583
Potassium	10/10	144000	47715	41701.95	77544.7	NA	NA	NA	NA
Silver	1/10	12	2.1	3.478505	4.5882	0.92	NA	NA	NA
Sodium	10/10	3880000 J	1167250	1209956	2032740	NA	NA	NA	NA
Vanadium	1/10	103	12.1	31.939	34.9462	NA	NA	NA	NA
Zinc	5/10	1860 J	203.28	582.5081	619.9523	110 [F]	110 [F]	5000	NA

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] Recalculated values from IRIS, based on a risk level of 1E-06, are presented. If none are available, then published or freshwater/marine chronic criteria are presented, if available. Published criteria are flagged [P]; freshwater criteria are flagged [F]; and marine criteria are flagged [M]. Freshwater criteria were given preference over marine criteria, if both are available.
[4] Lead retained for qualitative evaluation since no toxicity criteria are established.
[5] Protective of human health from toxic effects resulting from drinking water and fish consumption.
[6] Protective of human health from toxic effects resulting from fish consumption.
NA - Ambient water quality criteria: "None Available"; exposure point concentrations:"Not Applicable".

TABLE A-11
AREA A - SHALLOW SEDIMENTS (0-6")
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	NOAA Screening Guidelines ER-M Conc.	Region III Residential Soil COPC Screening Level	Retained as COPC?	Exposure Point Conc. (mg/kg)
VOLATILES (ug/kg)									
1,2-Dichloroethene	1/6	11 J	12.20833	3.951002	16.35533	NA	70000 [3]	No	NA
2-Butanone	4/6	42.5	31.08333	28.12546	60.604	NA	4700000	No	NA
Carbon disulfide	1/6	6 J	10.70833	5.201963	16.16835	NA	780000	No	NA
Chlorobenzene	1/6	6 J	10.04167	4.945747	15.23275	NA	160000	No	NA
Trichloroethene	1/6	3 J	10.875	5.490332	16.63769	NA	58000	No	NA
SEMIVOLATILES (ug/kg)									
Acenaphthene	2/6	430 J	360.8333	337.0967	714.6521	500	470000	No	NA
Anthracene	1/6	71 J	359.3333	331.2985	707.0663	1100	2300000	No	NA
Benzo(a)anthracene	4/6	500 J	235.8333	182.931	427.8389	1600	880	No	NA
Benzo(a)pyrene	4/6	320 J	194.1667	145.5767	346.9649	1600	88	No	NA
Benzo(b)fluoranthene	4/6	670 J	268.8333	233.5084	513.9252	NA	880	No	NA
Benzo(g,h,i)perylene	2/6	170 J	218.1667	113.7636	337.5737	NA	310000[4]	No	NA
Benzo(k)fluoranthene	3/6	220 J	205.3333	128.7659	340.4869	NA	8800	No	NA
Bis(2-ethylhexyl)phthalate	2/6	4100	938.3333	1552.046	2567.371	NA	46000	No	NA
Butylbenzylphthalate	1/6	170 J	284.1667	114.3423	404.1811	NA	1600000	No	NA
Carbazole	1/6	46 J	355.1667	335.777	707.6003	NA	32000	No	NA
Chrysene	4/6	570 J	245.3333	203.6464	459.0819	2800	88000	No	NA
Fluoranthene	5/6	1100 J	330.1667	406.5378	756.8713	5100	310000	No	NA
Indeno(1,2,3-cd)pyrene	2/6	190 J	224.8333	106.5372	336.6554	NA	880	No	NA
Phenanthrene	4/6	300 J	204.5	149.4373	361.3503	1500	310000 [4]	No	NA
Pyrene	5/6	850 J	260.6667	307.3439	583.2568	2600	230000	No	NA
PESTICIDES/PCBs (ug/kg)									
4,4'-DDD	6/6	310	106.3583	109.1232	220.8947	NA	2700	No	NA
4,4'-DDE	6/6	110	59.3	42.10439	103.493	27	1900	Yes	0.103
4,4'-DDT	5/6	73 L	22.14167	25.32766	48.72574	46.1	1900	Yes	0.049
alpha-BHC	2/6	62	15.42083	23.75544	40.3547	NA	100	No	NA
alpha-Chlordane	6/6	64 L	16.31583	24.37209	41.89694	NA	490 [5]	No	NA
Aroclor-1248	1/6	21 J	21	NA	NA	180 [6]	83	No	NA
Aroclor-1254	1/6	170	170	NA	NA	180 [6]	83	No	NA
Aroclor-1260	5/6	1500	351.5	574.6337	954.6392	180 [6]	83	Yes	0.955
beta-BHC	2/6	55	13.92083	20.9508	35.91093	NA	350	No	NA
delta-BHC	2/6	180	33.5875	71.79791	108.947	NA	350	No	NA
Dieldrin	2/6	14 L	7.020833	4.933366	12.19893	NA	40	No	NA
Endosulfan I	4/6	15	4.554167	5.214125	10.02695	NA	47000 [7]	No	NA
Endrin aldehyde	1/6	32	9.704167	11.37021	21.63841	NA	NA	No	NA
gamma-BHC	4/6	130	30.9825	51.22886	84.75264	NA	490	No	NA
gamma-Chlordane	6/6	69 L	15.74167	26.42178	43.47413	NA	490 [5]	No	NA
Heptachlor epoxide	1/6	1.2 J	1.2	NA	NA	NA	70	No	NA
METALS (mg/kg)									
Aluminum	1/1	1470	1470	NA	NA	NA	7800	No	NA
Arsenic	20/23	590	64.66957	122.711	117.737	70	0.37 [8]	Yes	118
Barium	1/1	22.1	22.1	NA	NA	NA	550	No	NA
Cadmium	13/23	160	24.69565	55.18058	48.55898	9.6	3.9	Yes	48.6
Calcium	1/1	509	509	NA	NA	NA	NA	No	NA
Chromium	20/23	3000	293.8348	681.0253	588.3502	370	39 [9]	Yes	588
Cobalt	1/1	3.3	3.3	NA	NA	NA	470	No	NA
Copper	1/1	553 J	553	NA	NA	270	290	Yes	553
Iron	1/1	5880	5880	NA	NA	NA	NA	No	NA

TABLE A-11
AREA A - SHALLOW SEDIMENTS (0-6")
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA
(CONTINUED)

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	NOAA Screening Guidelines ER-M Conc.	Region III Residential Soil COPC Screening Level	Retained as COPC?	Exposure Point Conc. (mg/kg)
METALS (mg/kg) - CON'T.									
Lead	21/23	1000	188.413	256.8237	299.4787	218	400 [10]	Yes [11]	299
Magnesium	1/1	367	367	NA	NA	NA	NA	No	NA
Manganese	1/1	51.2	51.2	NA	NA	NA	39	Yes	51.2
Mercury	20/23	3	0.495652	0.634055	0.769855	0.71	2.3	Yes	0.770
Nickel	1/1	25.6	25.6	NA	NA	223	160	No	NA
Potassium	1/1	274	274	NA	NA	NA	NA	No	NA
Silver	7/23	110	8.847826	24.5513	19.46526	3.7	39	Yes	19.5
Vanadium	15/23	180	53.9913	53.34657	77.0615	NA	55	Yes	77.1
Zinc	1/1	244 K	244	NA	NA	410	2300	No	NA

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] COPC screening level is for mixture of cis- and trans- isomers.
[4] Naphthalene COPC screening level used as a surrogate.
[5] Chlordane COPC screening level used as a surrogate.
[6] NOAA ER-M screening value for total PCBs.
[7] Endosulfan COPC screening level used as a surrogate.
[8] COPC screening level is for carcinogenic arsenic.
[9] COPC screening level is for chromium VI.
[10] Soil screening level for residential land use (USEPA, 1994b).
[11] Lead retained for qualitative evaluation since no toxicity criteria are established.
NA - Not applicable.

TABLE A-12
AREA A - DEEP SEDIMENTS
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	NOAA Screening Guidelines ER-M Conc.	Region III Residential Soil COPC Screening Level	Retained as COPC?	Exposure Point Conc. (mg/kg)
VOLATILES (ug/kg)									
1,2-Dichloroethene	1/1	26 J	26	NA	NA	NA	70000 [3]	No	NA
2-Butanone	1/1	120	120	NA	NA	NA	4700000	No	NA
Acetone	1/1	520	520	NA	NA	NA	780000	No	NA
Carbon disulfide	1/1	29 J	29	NA	NA	NA	780000	No	NA
Chlorobenzene	1/1	27 J	27	NA	NA	NA	160000	No	NA
Trichloroethene	1/1	12 J	12	NA	NA	NA	58000	No	NA
SEMIVOLATILES (ug/kg)									
Acenaphthene	1/1	4100	4100	NA	NA	500	470000	Yes	4.1
Benzo(a)anthracene	1/1	250 J	250	NA	NA	1600	880	No	NA
Benzo(b)fluoranthene	1/1	330 J	330	NA	NA	NA	880	No	NA
Bis(2-ethylhexyl)phthalate	1/1	1900 J	1900	NA	NA	NA	46000	No	NA
Chrysene	1/1	460 J	460	NA	NA	2800	88000	No	NA
Fluoranthene	1/1	1000 J	1000	NA	NA	5100	310000	No	NA
Naphthalene	1/1	200 J	200	NA	NA	2100	310000	No	NA
Pyrene	1/1	800 J	800	NA	NA	2600	230000	No	NA
PESTICIDES/PCBs (ug/kg)									
4,4'-DDD	1/1	380	380	NA	NA	NA	2700	No	NA
4,4'-DDE	1/1	85	85	NA	NA	27	1900	Yes	0.085
4,4'-DDT	1/1	1.1 J	1.1	NA	NA	46.1	1900	No	NA
Aldrin	1/1	14	14	NA	NA	NA	38	No	NA
Aroclor-1248	1/1	68 J	68	NA	NA	180 [4]	83	No	NA
Aroclor-1254	1/1	980	980	NA	NA	180 [4]	83	Yes	0.980
Dieldrin	1/1	62	62	NA	NA	NA	40	Yes	0.062
Endrin	1/1	11	11	NA	NA	NA	23000	No	NA
Endrin ketone	1/1	1.1 J	1.1	NA	NA	NA	NA	No	NA
gamma-BHC	1/1	1.2 J	1.2	NA	NA	NA	490	No	NA
gamma-Chlordane	1/1	16	16	NA	NA	NA	490 [5]	No	NA
METALS (mg/kg)									
Aluminum	1/1	2090	2090	NA	NA	NA	7800	No	NA
Arsenic	4/5	71	36.56	27.27761	70.4242	70	0.37 [6]	Yes	70.4
Barium	1/1	36.7	36.7	NA	NA	NA	550	No	NA
Cadmium	3/5	180	42.26	77.30128	138.2268	9.6	3.9	Yes	138
Calcium	1/1	2000	2000	NA	NA	NA	NA	No	NA
Chromium	4/5	1700	423.28	721.5914	1319.11	370	39 [7]	Yes	1319
Cobalt	1/1	1.9	1.9	NA	NA	NA	470	No	NA
Copper	1/1	216 J	216	NA	NA	270	290	No	NA
Iron	1/1	9330	9330	NA	NA	NA	NA	No	NA
Lead	5/5	540	156.4	223.9337	434.4058	218	400 [8]	Yes [9]	434
Magnesium	1/1	512	512	NA	NA	NA	NA	No	NA
Manganese	1/1	50.7	50.7	NA	NA	NA	39	Yes	50.7
Mercury	4/5	1.1	0.652	0.447795	1.207921	0.71	2.3	Yes	1.1
Nickel	1/1	11.1	11.1	NA	NA	223	160	No	NA
Potassium	1/1	341	341	NA	NA	NA	NA	No	NA
Silver	2/5	49	14.82	21.1611	41.09076	3.7	39	Yes	41.1
Vanadium	3/5	74	32.12	38.43139	79.83123	NA	55	Yes	74.0
Zinc	1/1	542 K	542	NA	NA	410	2300	Yes	542

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] COPC screening level is for mixture of cis- and trans- isomers.
[4] NOAA ER-M screening value for total PCBs.
[5] Chlordane COPC screening level used as a surrogate.
[6] COPC screening level is for carcinogenic arsenic.
[7] COPC screening level is for chromium VI.
[8] Soil screening level for residential land use (USEPA, 1994b).
[9] Lead retained for qualitative evaluation since no toxicity criteria are established.
NA - Not applicable.

TABLE A-13
AREA B - POND SURFACE WATER
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95%UCL for Arithmetic Mean [1][2]	Federal Ambient Water Quality Criteria		Virginia Water Quality Standards	
						Water and Organisms [3]	Organisms Only [3]	Public Water Supplies [5]	Other Surface Waters [6]
VOLATILES (ug/L)									
1,2-Dichloroethane	2/4	8 J	5.25	2.0615528	8.5299305	0.38	99	3.8	990
1,2-Dichloroethene	3/4	46	23.5	17.710637	51.677624	700	140000	NA	NA
2-Butanone	1/4	4 J	4.75	0.5	5.5455	NA	NA	NA	NA
Benzene	3/4	12	6.5	3.8729833	12.661917	1.2	71	12	710
Bromodichloromethane	1/4	6 J	5.25	0.5	6.0455	NA	NA	3	220
Carbon disulfide	1/4	3 J	4.5	1	6.091	NA	NA	NA	NA
Chloroform	2/4	24	9.25	9.8784277	24.966578	5.7	470	57	4700
Methylene chloride	1/4	3 B	4.5	1	6.091	4.7	1600	47	16000
Trichloroethene	3/4	45	25.25	16.740669	51.884404	3.1	92	27	807
Vinyl chloride	3/4	22	11	7.788881	23.39211	2	525	20	5250
SEMIVOLATILES (ug/L)									
2,4-Dimethylphenol	1/4	1 J	4	2	7.182	NA	NA	NA	NA
4-Methylphenol	1/4	3 J	4.5	1	6.091	NA	NA	NA	NA
Bis(2-ethylhexyl)phthalate	1/4	9 J	6	2	9.182	1.8	5.9	18	59
Di-n-Butylphthalate	1/4	1 J	4	2	7.182	2700	12000	NA	NA
Phenol	1/4	0.9 J	3.975	2.05	7.23655	21	4600	21000	4600000
PESTICIDES/PCBs (ug/L)									
4,4'-DDD	1/4	0.007 J	0.05175	0.0380208	0.1122411	NA	NA	NA	NA
gamma-BHC	1/4	0.007 J	0.02675	0.0176706	0.0548639	0.019	0.063	7	25
UNFILTERED METALS (ug/L)									
Aluminum	1/4	690	179.25	340.5	720.9855	87 [F]	87 [F]	NA	NA
Arsenic	3/4	6.7	4.175	2.3627315	7.9341058	0.018	0.14	50	NA
Barium	4/4	197	87.025	74.314753	205.25977	1000 [P]	NA	2000	NA
Calcium	4/4	106000	48825	38804.328	110562.69	NA	NA	NA	NA
Iron	4/4	8780	5984.75	3763.6857	11972.774	300 [P]	NA	NA	NA
Lead	4/4	15.8 J	6.875	6.2371869	16.798364	50 [P]	NA	15	NA
Magnesium	4/4	11400	7647.5	3765.2922	13638.08	NA	NA	NA	NA
Manganese	4/4	272	164.9	105.89762	333.38312	50 [P]	100 [P]	50	NA
Nickel	1/4	15	7.875	4.75	15.43225	510	3800	607	4583
Potassium	4/4	13600	5765	5234.5041	14093.096	NA	NA	NA	NA
Sodium	4/4	31400	25950	4992.3274	33892.793	NA	NA	NA	NA
Vanadium	2/4	5.4	3.375	1.6740669	6.0384404	NA	NA	NA	NA
Zinc	4/4	202	129.45	59.633408	224.32675	110[F]	110[F]	5000	NA

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] Recalculated values from IRIS, based on a risk level of 1E-06, are presented. If none are available, then published or freshwater/marine chronic criteria are presented, if available. Published criteria are flagged [P]; freshwater criteria are flagged [F]; and marine criteria are flagged [M]. Freshwater criteria were given preference over marine criteria, if both are available.
[4] Lead retained for qualitative evaluation since no toxicity criteria are established.
[5] Protective of human health from toxic effects resulting from drinking water and fish consumption.
[6] Protective of human health from toxic effects resulting from fish consumption.
NA - Ambient water quality criteria: "None Available"; exposure point concentrations:"Not Applicable".

TABLE A-14
AREA B POND - SHALLOW SEDIMENTS
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	NOAA Screening Guidelines ER-M Conc.	Region III Residential Soil COPC Screening Level	Retained as COPC?	Exposure Point Conc. (mg/kg)
VOLATILES (ug/kg)									
1,1-Dichloroethane	2/5	15 J	31.6	44.10414	86.35374	NA	780000	No	NA
1,2-Dichloroethane	1/5	20	34.7	42.44496	87.39394	NA	7000	No	NA
1,2-Dichloroethene	3/5	230	121.5	110.7384	258.9779	NA	70000 [3]	No	NA
2-Butanone	5/5	1300	331.6	549.5592	1013.858	NA	4700000	No	NA
Acetone	3/5	2300	611	975.9969	1822.666	NA	780000	No	NA
Benzene	2/5	62	42.2	43.64716	96.38642	NA	22000	No	NA
Carbon disulfide	1/5	22 J	32.9	43.62539	87.0594	NA	780000	No	NA
Methylene chloride	2/5	97 J	30.5	37.84012	77.47719	NA	85000	No	NA
Trichloroethene	3/5	520	144.1	214.4343	410.3126	NA	58000	No	NA
Vinyl chloride	2/5	60	47.8	40.6088	98.21441	NA	340	Yes	0.060
Xylenes(total)	1/5	4 J	31.5	44.38891	86.60727	NA	16000000	No	NA
SEMIVOLATILES (ug/kg)									
1,2-Dichlorobenzene	1/5	49 J	961.8	1143.865	2381.868	NA	700000	No	NA
1,4-Dichlorobenzene	1/5	45 J	961	1144.664	2382.06	NA	27000	No	NA
2-Methylnaphthalene	1/5	30 J	958	1147.68	2382.805	670	NA	No	NA
2-Methylphenol	1/5	310 J	963	1132.02	2368.364	NA	390000	No	NA
2,4-Dimethylphenol	1/5	1000 J	816	558.0031	1508.741	NA	160000	No	NA
4-Methylphenol	2/5	15000	3393	6501.499	11464.38	NA	39000	No	NA
Benzo(a)anthracene	2/5	190 J	482	551.7314	1166.955	1600	880	No	NA
Benzo(a)pyrene	2/5	210 J	485.6	549.5492	1167.846	1600	88	No	NA
Benzo(b)fluoranthene	3/5	390 J	297.8	160.1381	496.6058	NA	880	No	NA
Benzo(k)fluoranthene	2/5	140 J	467	563.5668	1166.648	NA	8800	No	NA
Bis(2-ethylhexyl)phthalate	2/5	5300	2191	2575.448	5388.328	NA	46000	No	NA
Chrysene	3/5	350 J	289	163.875	492.445	2800	88000	No	NA
Fluoranthene	2/5	630 J	586	509.2814	1218.255	5100	310000	No	NA
Naphthalene	1/5	46 J	961.2	1144.464	2382.012	2100	310000	No	NA
Phenanthrene	2/5	200 J	491.2	543.5207	1165.962	1500	310000 [4]	No	NA
Phenol	2/5	1800 J	705.6	758.3036	1647.007	NA	4700000	No	NA
Pyrene	2/5	350 J	519	529.6154	1176.499	2600	230000	No	NA
PESTICIDES/PCBs (ug/kg)									
4,4'-DDD	5/5	4200	889.6	1850.84	3187.354	NA	2700	Yes	4.20
4,4'-DDE	5/5	850	185.28	371.7771	646.8282	27	1900	Yes	0.850
4,4'-DDT	1/5	4.4 B	10.07	12.22828	25.25098	46.1	1900	No	NA
Aldrin	1/5	5.95 J	5	6.22254	12.72506	NA	38	No	NA
alpha-Chlordane	4/5	31 K	7.652	13.18054	24.01518	NA	490 [5]	No	NA
Aroclor-1254	4/5	7600	1661.2	3321.172	5784.319	180 [6]	83	Yes	5.78
Dieldrin	5/5	86 K	24.23	34.96072	67.63251	NA	40	Yes	0.068
Endosulfan I	4/5	6.4 J	6.104	5.746397	13.23795	NA	47000 [7]	No	NA
Endrin	1/5	0.87 J	9.534	12.60542	25.18318	NA	23000	No	NA
Endrin aldehyde	3/5	180 K	41.05	77.74986	137.5737	NA	NA	No	NA
gamma-Chlordane	4/5	56 K	13.3	23.89464	42.96436	NA	490 [5]	No	NA
Heptachlor epoxide	1/5	0.96 J	4.002	6.428823	11.98316	NA	70	No	NA
Methoxychlor	1/5	4.6 J	17.72	19.94159	42.47678	NA	39000	No	NA

TABLE A-14
AREA B POND - SHALLOW SEDIMENTS
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA
(CONTINUED)

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	NOAA Screening Guidelines ER-M Conc.	Region III Residential Soil COPC Screening Level	Retained as COPC?	Exposure Point Conc. (mg/kg)
METALS (mg/kg)									
Aluminum	5/5	26800	9000	11090.28	22768.2	NA	NA	No	22768
Antimony	1/2	16 L	15.6	0.565685	16.30228	NA	3.1	Yes	16
Arsenic	4/5	42.7	16.76	17.09161	37.97863	70	0.37 [8]	Yes	38.0
Barium	4/5	211 J	109.62	89.95706	221.2985	NA	550	No	NA
Beryllium	1/5	0.76	0.73	0.507986	1.360647	NA	0.15	Yes	0.76
Cadmium	3/5	41.9	13.402	16.98059	34.48281	9.6	3.9	Yes	34.5
Calcium	5/5	13400 J	4692	4969.997	10862.08	NA	NA	No	NA
Chromium	5/5	225	59.76	94.42533	176.9857	370	39 [9]	No	NA
Cobalt	2/5	20.8	6.46	8.565585	17.09387	NA	470	No	NA
Copper	5/5	298	133.78	143.9806	312.5269	270	290	Yes	298
Iron	5/5	77800	27126	32185.65	67083.36	NA	NA	No	NA
Lead	5/5	497 J	187.78	214.6786	454.5442	218	400 [10]	Yes [11]	455
Magnesium	4/5	4950	1688.3	2129.873	4332.463	NA	NA	No	NA
Manganese	5/5	246	99.84	112.79	239.8649	NA	39	Yes	240
Mercury	3/5	0.35 K	0.4335	0.553008	1.12004	0.71	2.3	Yes	0.35
Nickel	3/5	42.3	15.49	17.27941	36.94178	223	160	No	NA
Potassium	5/5	3870	1301	1649.586	3348.903	NA	NA	No	NA
Silver	2/5	14.7	5.54	6.434905	13.52871	3.7	39	Yes	13.5
Sodium	1/5	793	174.2	345.9197	603.6472	NA	NA	No	NA
Vanadium	4/5	130	49.82	52.03145	114.4152	NA	55	Yes	114
Zinc	5/5	1020	439.92	447.2031	995.1069	410	2300	Yes	995

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] Soil COPC screening level is for mixture of cis- and trans- isomers.
[4] Napthalene COPC screening level used as a surrogate.
[5] Chlordane COPC screening level used as a surrogate.
[6] NOAA ER-M screening value for total PCBs.
[7] Endosulfan COPC screening level used as a surrogate.
[8] COPC screening level is for carcinogenic arsenic.
[9] COPC screening level is for chromium VI.
[10] Soil screening level for residential land use (USEPA, 1994b).
[11] Lead retained for qualitative evaluation since no toxicity criteria are established.
NA - Not applicable.

TABLE A-15
AREA B POND - DEEP SEDIMENTS
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	NOAA Screening Guidelines ER-M Conc.	Region III Residential Soil COPC Screening Level	Retained as COPC?	Exposure Point Conc. (mg/kg)
VOLATILES (ug/kg)									
1,2-Dichloroethene	1/1	87	87	NA	NA	NA	70000 [3]	No	NA
2-Butanone	1/1	140	140	NA	NA	NA	4700000	No	NA
Acetone	1/1	340	340	NA	NA	NA	780000	No	NA
Tetrachloroethene	1/1	120	120	NA	NA	NA	12000	No	NA
Trichloroethene	1/1	39	39	NA	NA	NA	58000	No	NA
Vinyl chloride	1/1	10 J	10	NA	NA	NA	340	Yes	0.01
SEMIVOLATILES (ug/kg)									
4-Methylphenol	1/1	320 J	320	NA	NA	NA	39000	No	NA
Benzo(a)pyrene	1/1	230 J	230	NA	NA	1600	88	No	NA
Benzo(b)fluoranthene	1/1	460 J	460	NA	NA	NA	880	No	NA
Bis(2-ethylhexyl)phthalate	1/1	1800 J	1800	NA	NA	NA	46000	No	NA
Chrysene	1/1	300 J	300	NA	NA	2800	88000	No	NA
Fluoranthene	1/1	340 J	340	NA	NA	5100	310000	No	NA
Pyrene	1/1	240 J	240	NA	NA	2600	230000	No	NA
PESTICIDES/PCBs (ug/kg)									
4,4'-DDD	1/1	1600 J	1600	NA	NA	NA	2700	No	NA
4,4'-DDE	1/1	60 L	60	NA	NA	27	1900	Yes	0.06
4,4'-DDT	1/1	4400	4400	NA	NA	46.1	1900	Yes	4.4
METALS (mg/kg)									
Aluminum	1/1	7440	7440	NA	NA	NA	7800	No	NA
Arsenic	1/1	31.3	31.3	NA	NA	70	0.37 [4]	No	NA
Barium	1/1	50.1	50.1	NA	NA	NA	550	No	NA
Beryllium	1/1	0.56	0.56	NA	NA	NA	0.15	Yes	0.56
Cadmium	1/1	12	12	NA	NA	9.6	3.9	Yes	12
Calcium	1/1	1650	1650	NA	NA	NA	NA	No	NA
Chromium	1/1	104	104	NA	NA	370	39 [5]	No	NA
Cobalt	1/1	8.4	8.4	NA	NA	NA	470	No	NA
Copper	1/1	126 J	126	NA	NA	270	290	No	NA
Iron	1/1	18100	18100	NA	NA	NA	NA	No	NA
Lead	1/1	169 J	169	NA	NA	218	400 [6]	No	NA
Magnesium	1/1	1510	1510	NA	NA	NA	NA	No	NA
Manganese	1/1	69.6	69.6	NA	NA	NA	39	Yes	69.6
Mercury	1/1	0.33	0.33	NA	NA	0.71	2.3	No	NA
Nickel	1/1	17	17	NA	NA	223	160	No	NA
Potassium	1/1	1000	1000	NA	NA	NA	NA	No	NA
Silver	1/1	3.1	3.1	NA	NA	3.7	39	No	NA
Vanadium	1/1	34.8	34.8	NA	NA	NA	55	No	NA
Zinc	1/1	336 K	336	NA	NA	410	2300	No	NA

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] COPC screening level is for mixture of cis- and trans- isomers.
[4] COPC screening level is for carcinogenic arsenic.
[5] Chromium VI COPC screening level.
[6] Soil screening level for residential land use (USEPA, 1994b).
NA - Not applicable.

TABLE A-16
AREA B - SCHOOL SURFACE WATER
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	Federal Ambient Water Quality Criteria		Virginia Water Quality Standards	
						Water and Organisms [3]	Organisms Only [3]	Public Water Supplies [5]	Other Surface Waters [6]
SEMIVOLATILES (ug/L)									
Diethylphthalate	1/2	0.9 J	2.95	2.899138	28.9973	23000	120000	NA	NA
2,4-Dimethylphenol	1/2	2 J	3.5	2.12132	22.559	NA	NA	NA	NA
PESTICIDES/PCBs (ug/L)									
4,4'-DDD	2/2	0.038 J	0.035	0.004243	0.073118	NA	NA	NA	NA
UNFILTERED METALS (ug/L)									
Antimony	1/2	20.6	14.8	8.202439	88.4948	14	8600	NA	NA
Arsenic	2/2	11.5 K	9.7	2.545584	32.5708	0.018	0.14	50	NA
Barium	2/2	46.3	41.4	6.929646	103.6594	1000 [P]	NA	2000	NA
Calcium	2/2	86300	54550	44901.28	457965.5	NA	NA	NA	NA
Copper	1/2	27.1	14.05	18.45549	179.8633	1300	NA	1300	NA
Iron	2/2	14300	10720	5062.885	56207.48	300 [P]	NA	NA	NA
Lead	2/2	53.6	31.1	31.81981	316.985	50 [P]	NA	15	NA
Magnesium	2/2	27000	17335	13668.37	140138.49	NA	NA	NA	NA
Manganese	2/2	574	463.5	156.2706	1867.513	50 [P]	100 [P]	50	NA
Potassium	2/2	8810	6005	3966.869	41645.33	NA	NA	NA	NA
Sodium	2/2	109000	65550	61447.58	617625.7	NA	NA	NA	NA
Zinc	2/2	199 J	132.5	94.0452	977.449	110 [F]	110 [F]	5000	NA

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] Recalculated values from IRIS, based on a risk level of 1E-06, are presented. If none are available, then published or freshwater/marine chronic criteria are presented, if available. Published criteria are flagged [P]; freshwater criteria are flagged [F]; and marine criteria are flagged [M]. Freshwater criteria were given preference over marine criteria, if both are available.
[4] Lead retained for qualitative evaluation since no toxicity criteria are established.
[5] Protective of human health from toxic effects resulting from drinking water and fish consumption.
[6] Protective of human health from toxic effects resulting from fish consumption.
NA - Ambient water quality criteria: "None Available"; exposure point concentrations:"Not Applicable".

TABLE A-17
AREA B - SHALLOW SEDIMENTS IN ELEMENTARY SCHOOL AREA
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	NOAA Screening Guidelines ER-M Conc.	Region III Residential Soil COPC Screening Level	Retained as COPC?	Exposure Point Conc. (mg/kg)
METALS (mg/kg)									
Arsenic	1/2	23	12	15.55635	151.766	70	0.37 [3]	No	NA
Chromium	1/2	34	17.5	23.33452	227.149	370	39 [4]	No	NA
Lead	1/2	94	47.5	65.76093	638.329	218	NA	No	NA
Mercury	1/2	0.6	0.3375	0.371231	3.672825	0.71	2.3	No	NA
Vanadium	1/2	58	29.5	40.30509	391.621	NA	55	No	NA

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] COPC screening level is for carcinogenic arsenic.
[4] COPC screening level is for chromium VI.
[5] Lead retained for qualitative evaluation since no toxicity criteria are established.
NA - Not applicable.

TABLE A-18
AREA B - DEEP-SEDIMENTS IN ELEMENTARY SCHOOL AREA
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc.	Arithmetic Mean Conc. [1]	Standard Deviation [1]	95% UCL for Arithmetic Mean [1][2]	NOAA Screening Guidelines ER-M Conc.	Region III Residential Soil COPC Screening Level	Retained as COPC?	Exposure Point Conc. (mg/kg)
METALS (mg/kg)									
Arsenic	1/1	6.4	6.4	NA	NA	70	0.37 [3]	No	NA
Chromium	1/1	21	21	NA	NA	370	39 [4]	No	NA
Lead	1/1	310	310	NA	NA	218	NA	Yes [5]	310
Mercury	1/1	0.8	0.8	NA	NA	0.71	2.3	Yes	0.8

Notes:
[1] One-half detection limit values for non-detects are included in statistical calculations.
[2] 95% UCL calculated for a normal distribution.
[3] COPC screening level is for carcinogenic arsenic.
[4] COPC screening level is for chromium VI.
[5] Lead retained for qualitative evaluation since no toxicity criteria are established.
NA - Not applicable.

TABLE A-19
BRIG FACILITY - INDOOR AIR
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc. (ug/m3)	Region III Ambient Air COPC Screening Level (ug/m3)	U.S. EPA VOC Data Base (ug/m3)	Maximum Detected Background Ambient Air Conc. (ug/m3)	Retained as COPC? (Yes/No)	Exposure Point Conc. (mg/m3)
VOLATILES (ug/m3)							
1,1,1-Trichloroethane	32/33	3400	100	0.04	11	Yes	3.40
1,2,4-Trichlorobenzene	1/33	0.4	21	NA	ND	No	NA
1,2,4-Trimethylbenzene	20/33	7.8	0.18	0.83	0.3	Yes	0.0078
1,3,5-Trimethylbenzene	8/33	4.4	0.15	NA	ND	Yes	0.0044
1,4-Dichlorobenzene	11/33	26	0.26	0.001	0.3	Yes	0.026
Benzene	27/33	1	0.22	1.39	0.7	Yes	0.001
Benzyl chloride	1/33	0.9	0.037	NA	ND	Yes	0.0009
Bromomethane	7/33	0.9	0.52	NA	0.6	Yes	0.0009
Chloroform	2/33	0.8	0.078	0.23	ND	Yes	0.0008
Chloromethane	9/33	1.1	0.99	NA	0.7	Yes	0.0011
Dichlorodifluoromethane	26/33	0.8	21	NA	0.7	No	NA
Ethylbenzene	6/33	0.7	100	0.49	ND	No	NA
Freon 113	1/33	0.9	NA	NA	ND	No	NA
Freon 114	1/33	0.4	NA	NA	ND	No	NA
Hexachlorobutadiene	1/33	1	0.081	NA	0.3	Yes	0.001
meta/para-Xylene	26/33	100	31 [1]	1.615	0.7	Yes	0.1
Methylene chloride	26/33	380	3.8	8.373	0.8	Yes	0.38
ortho-Xylene	12/33	32	73	0.615	0.3	No	NA
Styrene	2/33	8.9	100	NA	ND	No	NA
Tetrachloroethene	1/33	0.3	3.1	0.026	ND	No	NA
Toluene	32/33	110	42	5.49	1.6	Yes	0.11
Trichlorofluoromethane	21/33	6.5	73	NA	0.4	No	NA

Notes:
[1] COPC screening level is for para-xylene.
NA - Criterion not available; exposure concentration not applicable since chemical is not a COPC.
ND - Not detected.

TABLE A-20
ELEMENTARY SCHOOL - INDOOR AIR
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc. (ug/m3)	Region III Ambient Air COPC Screening Level (ug/m3)	U.S. EPA VOC Data Base (ug/m3)	Maximum Detected Background Ambient Air Conc. (ug/m3)	Retained as COPC? (Yes/No)	Exposure Point Conc. (mg/m3)
VOLATILES (ug/m3)							
1,1,1-Trichloroethane	15/15	27	100	0.04	11	No	NA
1,2,4-Trimethylbenzene	4/15	0.3	0.18	NA	0.3	Yes	0.0003
1,4-Dichlorobenzene	6/15	0.6	0.26	0.001	0.3	Yes	0.0006
Benzene	15/15	0.7	0.22	1.39	0.7	Yes	0.0007
Chloromethane	2/15	0.8	0.99	NA	0.7	No	NA
Dichlorodifluoromethane	12/15	1.1	21	NA	0.7	No	NA
Hexachlorobutadiene	4/15	0.3	0.081	NA	0.3	Yes	0.0003
meta-/para-Xylene	15/15	0.8	31 [1]	1.615	0.7	No	NA
Methylene chloride	15/15	0.8	3.8	8.373	0.8	No	NA
ortho-Xylene	1/15	0.3	73	0.615	0.3	No	NA
Toluene	15/15	1.9	42	5.49	1.6	No	NA
Trichlorofluoromethane	13/15	0.7	73	NA	0.4	No	NA

Notes:
[1] COPC screening level is for para-xylene.
NA - Criterion not available; exposure concentration not applicable since chemical is not a COPC.

TABLE A-21
AMBIENT AIR
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc. (ug/m3)	Region III Ambient Air COPC Screening Level (ug/m3)	U.S. EPA VOC Data Base (ug/m3)	Maximum Detected Background Ambient Air Conc. (ug/m3)	Retained as COPC? (Yes/No)	Exposure Point Conc. (mg/m3)
VOLATILES (ug/m3)							
1,1,1-Trichloroethane	6/9	14	100	0.04	11	No	NA
1,2,4-Trimethylbenzene	3/9	0.3	0.18	0.83	0.3	Yes	0.0003
Benzene	9/9	0.7	0.22	1.39	0.7	Yes	0.0007
Bromomethane	1/9	0.8	0.52	NA	0.6	Yes	0.0008
Dichlorodifluoromethane	6/9	0.6	21	NA	0.7	No	NA
Hexachlorobutadiene	3/9	0.3	0.081	NA	0.3	Yes	0.0003
meta-/para-Xylene	8/9	0.7	31 [1]	1.615	0.7	No	NA
Methylene chloride	6/9	1.2	3.8	8.373	0.8	No	NA
Toluene	9/9	1.2	42	5.49	1.6	No	NA

Notes:
[1] COPC screening level is for para-xylene.
NA - Criterion not available; exposure concentration not applicable since chemical is not a COPC.

TABLE A-22
BRIG FACILITY - OUTDOOR AIR (NEAR GAS WELL)
SELECTION OF COPCs AND EXPOSURE POINT CONCENTRATIONS
CAMP ALLEN LANDFILL, CTO-084
NORFOLK NAVAL BASE
NORFOLK, VIRGINIA

Parameter	Frequency of Detection	Maximum Detected Conc. (ug/m3)	Region III Ambient Air COPC Screening Level (ug/m3)	U.S. EPA VOC Data Base (ug/m3)	Maximum Detected Background Ambient Air Conc. (ug/m3)	Retained as COPC? (Yes/No)	Exposure Point Conc. (mg/m3)
VOLATILES (ug/m3)							
1,1,1-Trichloroethane	3/3	8.5	100	0.04	11	No	NA
1,2,4-Trimethylbenzene	2/3	0.5	0.18	0.83	ND	Yes	0.0005
Benzene	3/3	0.7	0.22	1.39	0.7	Yes	0.0007
Dichlorodifluoromethane	1/3	0.7	21	NA	0.7	No	NA
Ethylbenzene	3/3	0.6	100	0.49	ND	No	NA
Freon 114	2/3	42	NA	NA	ND	No	NA
meta-/para-Xylene	3/3	2.3	31 [1]	1.615	0.7	No	NA
Methylene chloride	3/3	1.4	3.8	8.373	0.8	No	NA
ortho-Xylene	3/3	0.7	73	0.615	0.3	No	NA
Toluene	3/3	670	42	5.49	1.6	Yes	0.67
Trichlorofluoromethane	1/3	0.2	73	NA	0.4	No	NA

Notes:
[1] COPC screening level is for para-xylene.

APPENDIX B - SHOWER INHALATION MODEL

APPENDIX B
CALCULATION SPREADSHEETS FOR SHOWER AIR MODEL AND DERMAL PERMEABILITIES

CALCULATION SPREADSHEETS
FOR
ANDELMAN SHOWER MODEL

TABLE B-1
VOLITILZATION OF COPCs FROM WATER DURING SHOWERING
CAMP ALLEN LANDFILL
AREA A SHALLOW AQUIFER WELL LOCATIONS B-20W AND B-20WSS
NORFOLK, VIRGINIA
CONTRACT TASK ORDER 0084

This spreadsheet calculates the average concentration of a volatile organic compound in shower air (mg/m3) over the duration of the shower. The air concentration is estimated by a balance between the rate of chemical release from the shower and the rate of air exchange between the shower and the bathroom and the rest of the house. The calculations are based on the efficiency of the volatilization of trichloroethene from shower water as observed in model showers, as well as in several homes. The model was developed by Dr. Julian B. Andelman at the Graduate School of Public Health, University of Pittsburgh.

$$Ca = Cinf[1+((1/kts))(exp(-kts)-1))]$$

Where:

$$Cinf = [(E)(Fw)(Ct/1000)]/Fa$$
$$k = Fa/Vb$$
$$E = (ETCE)(H)/(HTCE)$$

The following defines the parameters used in the Andelman Shower Model:

Parameter	Constant	Units	Description
Ca	Chem. Specific	mg/m3	Avg. Air Conc. over Shower Duration
Cinf	Chem. Specific	mg/m3	Asymptotic Conc. in Air
E	Chem. Specific	unitless	Efficiency of Release-Water to Air
H	Chem. Specific	m3-atm/mol	Henry's Constant for Chemical (i)
Ct	Chem. Specific	ug/L	Conc. of Chemical (i) in Shower Wate
ETCE	0.6	unitless	Efficiency of Release of TCE
HTCE	9.1E-03	m3-atm/mol	Henry's Constant for TCE
Fa	0.0483334	m3/min	Flow Rate of Air in the Shower
Vb	2.9	m3	Volume of Average Bathroom
k	0.01666668966	1/min	Rate Constant
Fw	20	L/min	Flow Rate of Water in Shower
ts	12	min	Showering Time

COPCs (Well B-20W)	H (atm-m3/mol)	E	Ct (ug/L)	Cinf (mg/m3)	Ca (mg/m3)
Benzene	5.50E-03	3.6E-01	310	4.7E+01	4.36
1,2-Dichloroethene	6.56E-03	4.3E-01	6100	1.1E+03	102
2-Butanone	2.74E-05	1.8E-03	4300	3.2E+00	0.301
4-Methyl-2-pentanone	9.40E-05	6.2E-03	16000	4.1E+01	3.84
Methylene chloride	2.03E-03	1.3E-01	0	0.0E+00	0.00
Tetrachloroethene	2.59E-02	1.7E+00	0	0.0E+00	0.00
Trichloroethene	9.10E-03	6.0E-01	0	0.0E+00	0.00
Toluene	6.37E-03	4.2E-01	5400	9.4E+02	87.9
Vinyl chloride	8.19E-02	5.4E+00	3300	7.4E+03	691

COPCs (Well B-20WSS)	H (atm-m3/mol)	E	Ct (ug/L)	Cinf (mg/m3)	Ca (mg/m3)
Benzene	5.50E-03	3.6E-01	0	0.0E+00	0.00
1,2-Dichloroethene	6.56E-03	4.3E-01	1600	2.9E+02	26.8
2-Butanone	2.74E-05	1.8E-03	0	0.0E+00	0.00
4-Methyl-2-pentanone	9.40E-05	6.2E-03	220	5.6E-01	0.053
Methylene chloride	2.03E-03	1.3E-01	57	3.2E+00	0.30
Tetrachloroethene	2.59E-02	1.7E+00	620	4.4E+02	41.0
Trichloroethene	9.10E-03	6.0E-01	1800	4.5E+02	41.9
Toluene	6.37E-03	4.2E-01	1200	2.1E+02	19.5
Vinyl chloride	8.19E-02	5.4E+00	0	0.0E+00	0.00

TABLE B-2
VOLITILZATION OF COPCs FROM WATER DURING SHOWERING
CAMP ALLEN LANDFILL
AREA A DEEP AQUIFER WELL LOCATION A-MW1B
NORFOLK, VIRGINIA
CONTRACT TASK ORDER 0084

This spreadsheet calculates the average concentration of a volatile organic compound in shower air (mg/m3) over the duration of the shower. The air concentration is estimated by a balance between the rate of chemical release from the shower and the rate of air exchange between the shower and the bathroom and the rest of the house. The calculations are base on the efficiency of the volatilization of trichloroethene from shower water as observed in model showers, as well as in several homes. The model was developed by Dr. Julian B. Andelman at the Graduate School of Public Health, University of Pittsburgh.

$$Ca = Cinf[1+((1/kts))(exp(-kts)-1))]$$

Where:

$$Cinf = [(E)(Fw)(Ct/1000)]/Fa$$
$$k = Fa/Vb$$
$$E = (ETCE)(H)/(HTCE)$$

The following defines the parameters used in the Andelman Shower Model:

Parameter	Constant	Units	Description
Ca	Chem. Specific	mg/m3	Avg. Air Conc. over Shower Duration
Cinf	Chem. Specific	mg/m3	Asymptotic Conc. in Air
E	Chem. Specific	unitless	Efficiency of Release-Water to Air
H	Chem. Specific	m3-atm/mol	Henry's Constant for Chemical (i)
Ct	Chem. Specific	ug/L	Conc. of Chemical (i) in Shower Wat
ETCE	0.6	unitless	Efficiency of Release of TCE
HTCE	9.1E-03	m3-atm/mol	Henry's Constant for TCE
Fa	0.0483334	m3/min	Flow Rate of Air in the Shower
Vb	2.9	m3	Volume of Average Bathroom
k	0.0166666897	1/min	Rate Constant
Fw	20	L/min	Flow Rate of Water in Shower
ts	12	min	Showering Time

COPCs (Well A-MW1B)	H (atm-m3/mol)	E	Ct (ug/L)	Cinf (mg/m3)	Ca (mg/m3)
1,2-Dichloroethane	9.78E-04	6.4E-02	38	1.0E+00	0.095
1,2-Dichloroethene	6.56E-03	4.3E-01	540	9.7E+01	9.05
Trichloroethene	9.10E-03	6.0E-01	100	2.5E+01	2.33
Vinyl chloride	8.19E-02	5.4E+00	100	2.2E+02	20.9

TABLE B-3
VOLITILZATION OF COPCs FROM WATER DURING SHOWERING
CAMP ALLEN LANDFILL
AREA B SHALLOW AQUIFER WELL LOCATION B-MW11A
NORFOLK, VIRGINIA
CONTRACT TASK ORDER 0084

This spreadsheet calculates the average concentration of a volatile organic compound in shower air (mg/m3) over the duration of the shower. The air concentration is estimated by a balance between the rate of chemical release from the shower and the rate of air exchange between the shower and the bathroom and the rest of the house. The calculations are base on the efficiency of the volatilization of trichloroethene from shower water as observed in model showers, as well as in several homes. The model was developed by Dr. Julian B. Andelman at the Graduate School of Public Health, University of Pittsburgh.

$$Ca = Cinf[1+((1/kts))(exp(-kts)-1))]$$

Where:

$$Cinf = [(E)(Fw)(Ct/1000)]/Fa$$
$$k = Fa/Vb$$
$$E = (ETCE)(H)/(HTCE)$$

The following defines the parameters used in the Andelman Shower Model:

Parameter	Constant	Units	Description
Ca	Chem. Specific	mg/m3	Avg. Air Conc. over Shower Duration
Cinf	Chem. Specific	mg/m3	Asymptotic Conc. in Air
E	Chem. Specific	unitless	Efficiency of Release-Water to Air
H	Chem. Specific	m3-atm/mol	Henry's Constant for Chemical (i)
Ct	Chem. Specific	ug/L	Conc. of Chemical (i) in Shower Wat
ETCE	0.6	unitless	Efficiency of Release of TCE
HTCE	9.1E-03	m3-atm/mol	Henry's Constant for TCE
Fa	0.0483334	m3/min	Flow Rate of Air in the Shower
Vb	2.9	m3	Volume of Average Bathroom
k	0.01666668966	1/min	Rate Constant
Fw	20	L/min	Flow Rate of Water in Shower
ts	12	min	Showering Time

COPCs (Well B-MW11A)	H (atm-m3/mol)	E	Ct (ug/L)	Cinf (mg/m3)	Ca (mg/m3)
Benzene	5.59E-03	3.7E-01	29	4.4E+00	0.414
1,2-Dichloroethane	9.78E-04	6.4E-02	58	1.5E+00	0.145
1,2-Dichloroethene	6.56E-03	4.3E-01	1600	2.9E+02	26.8
Trichloroethene	9.10E-03	6.0E-01	44	1.1E+01	1.02
Vinyl chloride	8.19E-02	5.4E+00	94	2.1E+02	19.7

TABLE B-4
VOLITILZATION OF COPCs FROM WATER DURING SHOWERING
CAMP ALLEN LANDFILL
AREA B DEEP AQUIFER WELL LOCATION B-MW19B
NORFOLK, VIRGINIA
CONTRACT TASK ORDER 0084

This spreadsheet calculates the average concentration of a volatile organic compound in shower air (mg/m3) over the duration of the shower. The air concentration is estimated by a balance between the rate of chemical release from the shower and the rate of air exchange between the shower and the bathroom and the rest of the house. The calculations are base on the efficiency of the volatilization of trichloroethene from shower water as observed in model showers, as well as in several homes. The model was developed by Dr. Julian B. Andelman at the Graduate School of Public Health, University of Pittsburgh.

$$Ca = Cinf[1+((1/kts))(exp(-kts)-1))]$$

Where:

$$Cinf = [(E)(Fw)(Ct/1000)]/Fa$$
$$k = Fa/Vb$$
$$E = (ETCE)(H)/(HTCE)$$

The following defines the parameters used in the Andelman Shower Model:

Parameter	Constant	Units	Description
Ca	Chem. Specific	mg/m3	Avg. Air Conc. over Shower Duration
Cinf	Chem. Specific	mg/m3	Asymptotic Conc. in Air
E	Chem. Specific	unitless	Efficiency of Release-Water to Air
H	Chem. Specific	m3-atm/mol	Henry's Constant for Chemical (i)
Ct	Chem. Specific	ug/L	Conc. of Chemical (i) in Shower Water
ETCE	0.6	unitless	Efficiency of Release of TCE
HTCE	9.1E-03	m3-atm/mol	Henry's Constant for TCE
Fa	0.0483334	m3/min	Flow Rate of Air in the Shower
Vb	2.9	m3	Volume of Average Bathroom
k	0.01666668966	1/min	Rate Constant
Fw	20	L/min	Flow Rate of Water in Shower
ts	12	min	Showering Time

COPCs (Well B-MW19B)	H (atm-m3/mol)	E	Ct (ug/L)	Cinf (mg/m3)	Ca (mg/m3)
Benzene	5.59E-03	3.7E-01	12	1.8E+00	0.171
Chloroform	2.87E-03	1.9E-01	1	7.9E-02	0.007
1,2-Dichloroethane	9.78E-04	6.4E-02	3	8.0E-02	0.007
1,2-Dichloroethene	6.56E-03	4.3E-01	16	2.9E+00	0.3
Trichloroethene	9.10E-03	6.0E-01	18	4.5E+00	0.42

ESTIMATION OF DERMAL PERMEABILITIES
FOR
ORGANIC COPCs IN GROUNDWATER AND SURFACE WATER

TABLE B-5
CALCULATION SUMMARY OF ADJUSTED DERMAL PERMEABILITY FACTORS
DERMAL EXPOSURES TO ORGANIC COPCs IN GROUNDWATER AND SURFACE WATER
CAMP ALLEN LANDFILL
CONTRACT TASK ORDER 084

This spreadsheet adjusts the dermal permeability constant (Kp) for use in the calculation of the dose absorbed (DA) during and after dermal exposure to each COPC detected in groundwater and surface water. Values for the adjusted dermal permeability constant (Kp') determined for a COPC in groundwater may vary from the Kp' value determined for the same COPC in surface water as a function of the duration of the exposure event (tevent). The following equations used in the calculation of Kp' values for groundwater and surface water COPCs detected at the Camp Allen Landfill site were obtained from USEPA's guidance document entitled Dermal Exposure Assessment: Principles and Applications, Interim Report, January, 1992. The Kp' value (cm/event) obtained for a COPC in this spreadsheet will be multiplied by the COPC water concentration in the groundwater/surface water dermal risk calculation spreadsheet(s) to derive a dermally absorbed dose (DAD), which is then used in the derivation of risk levels and hazard quotients.

If tevent < t*, then:

$$Kp' = 2 * Kp * [(6 * tau * tevent)/pi]^{0.5}$$

If tevent > t*, then:

$$Kp' = Kp * \{[tevent/(1 + B)] + 2 * tau * [(1 + 3B)/(1 + B)]\}$$

Where:
Kp' = Adjusted dermal permeability constant (cm/event)
Kp = Dermal permeability constant (cm/hour)
tevent = Duration of event (hours/event)
tau = Lag time (hours)
pi = 3.1416
B = Partitioning constant (unitless)

Parameter	Kp (cm/hour)	Kow	log Kow	MW (g/mole)	B	tau (hours)	t* (hours)	Kp' Groundwater (tevent = 0.2 hrs) (cm/event)	Kp' Surface Water (tevent = 2.6 hrs) (cm/event)
1,1-Dichloroethene	1.6E-02	1.3E+02	2.13	96.9	1.3E-02	3.4E-01	8.2E-01	0.01153	NA
1,2-Dichloroethane	5.3E-03	3.0E+01	1.48	99.0	3.0E-03	3.5E-01	8.4E-01	0.00388	0.01747
1,2-Dichloroethene	1.0E-02	7.2E+01	1.86	96.9	7.2E-03	3.4E-01	8.2E-01	0.00721	0.03271
1,4-Dichlorobenzene	6.2E-02	2.5E+03	3.39	147.0	2.5E-01	6.9E-01	3.3E+00	0.06366	NA
2-Butanone	1.1E-03	2.0E+00	0.29	72.0	1.9E-04	2.4E-01	5.8E-01	0.00067	0.00339
2,4-Dimethylphenol	1.5E-02	2.0E+02	2.30	122.0	2.0E-02	4.9E-01	1.2E+00	0.01298	NA
2-Methylphenol	1.0E-02	8.9E+01	1.95	108.1	8.9E-03	4.0E-01	9.6E-01	0.00782	NA
4,4'-DDD	2.8E-01	6.3E+05	5.80	320.0	6.3E+01	7.8E+00	3.7E+01	NA	3.48516
4,4'-DDE	2.4E-01	4.9E+05	5.69	318.0	4.9E+01	7.6E+00	3.6E+01	NA	2.94873
4-Methyl-2-pentanone	3.3E-03	1.5E+01	1.19	100.0	1.5E-03	3.6E-01	8.6E-01	0.00245	NA
4-Methylphenol	1.8E-02	8.7E+01	1.94	108.1	8.7E-03	4.0E-01	9.6E-01	0.01407	NA
Aldrin	1.6E-03	1.0E+03	3.01	365.0	1.0E-01	1.5E+01	3.6E+01	0.00766	NA
Aroclor-1254	3.7E-01	1.1E+06	6.04	328.0	1.1E+02	5.3E+01	2.5E+02	NA	11.94102
Benzene	2.1E-01	1.3E+02	2.13	78.1	1.3E-02	2.6E-01	6.3E-01	0.13236	0.65100
Bis(2-chloroethyl)ether	2.1E-03	2.0E+01	1.29	143.0	1.9E-03	6.5E-01	1.6E+00	0.00209	NA
Bis(2-ethylhexyl)phthalate	3.3E-02	1.3E+05	5.11	391.0	1.3E+01	2.1E+01	1.0E+02	0.18693	0.67397
Chlorobenzene	4.1E-02	6.9E+02	2.84	112.6	6.9E-02	4.3E-01	1.0E+00	0.03323	NA
Chloroform	8.9E-03	9.3E+01	1.97	119.4	9.3E-03	4.7E-01	1.1E+00	0.00754	0.03145
delta-BHC	2.6E-02	1.3E+04	4.10	291.0	1.3E+00	5.2E+00	3.7E+01	NA	0.26485
Dieldrin	1.6E-02	3.6E+04	4.56	381.0	3.6E+00	1.8E+01	9.4E+01	0.08391	0.30253
Heptachlor epoxide	6.7E-04	5.0E+02	2.70	389.0	5.0E-02	2.1E+01	5.0E+01	0.00375	0.01351
Methylene chloride	4.5E-03	1.8E+01	1.25	84.9	1.8E-03	2.9E-01	6.9E-01	0.00300	NA
Tetrachloroethene	4.8E-02	2.5E+03	3.40	165.8	2.5E-01	9.0E-01	4.3E+00	0.05629	0.20295
Toluene	4.5E-02	5.4E+02	2.73	92.1	5.4E-02	3.2E-01	7.7E-01	0.03147	NA
Trichloroethene	1.6E-02	2.6E+02	2.42	131.4	2.6E-02	5.5E-01	1.3E+00	0.01467	0.05904
Vinyl chloride	7.3E-03	2.3E+01	1.36	62.5	2.3E-03	2.1E-01	5.1E-01	0.00414	0.02202
Xylenes	8.0E-02	1.6E+03	3.20	106.2	1.6E-01	3.9E-01	1.4E+00	NA	0.25892
alpha-Chlordane	4.6E-02	3.0E+05	5.47	410.0	3.0E+01	2.8E+01	1.3E+02	NA	1.08481
gamma-BHC	1.4E-02	5.2E+03	3.72	291.0	5.2E-01	5.2E+00	3.5E+00	0.03946	NA
gamma-Chlordane	4.6E-02	3.0E+05	5.47	410.0	3.0E+01	2.8E+01	1.3E+02	NA	1.08481

APPENDIX C - TOXICITY PROFILES

APPENDIX C
TOXICOLOGICAL PROFILES

Date of Last Revision: 11/10/94
Revisor: Rich Hoff

ACENAPHTHENE

INTRODUCTION

Acenaphthene is a two-ringed polycyclic aromatic hydrocarbon (PAH). Although little specific information on acenaphthene is available, information on related PAHs suggests that acenaphthene is not very persistent in the environment and that biodegradation is the ultimate fate process. Acenaphthene has not been shown to be carcinogenic or mutagenic, but it does cause liver and kidney damage at high exposure levels.

CAS Number:	83-32-9
Chemical Formula:	$C_{12}H_{10}$
IUPAC Name:	Acenaphthene

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight:	154.21
Boiling Point:	279°C
Melting Point:	96.2°C
Specific Gravity:	1.225 at 0°C
Solubility in Water:	3.42 mg/liter at 25°C
Solubility in Organics:	Soluble in ethanol, toluene, chloroform, benzene, and acetic acid
Log K_{ow}:	4.33
Vapor Pressure:	1×10^{-3} mm Hg at 20°C
Vapor Density:	5.32

FATE AND TRANSPORT

Acenaphthene, like other polycyclic aromatic hydrocarbons (PAHs), can be emitted into the environment by both natural and anthropogenic sources. Since very little specific information is available on this compound, its environmental fate is largely inferred from data for PAHs in general. In air, acenaphthene can be transported as adsorbed matter on suspended particulates. Ambient air samples collected in Sydney, Australia, contained 0.07 μg/100 m^3, indicating that atmospheric transport occurs and that individuals in urban environments may be exposed to measureable levels.

In surface water, direct, rapid photolysis of dissolved acenaphthene may be an important water-related environmental fate. It is probable that singlet oxygen is the oxidant and that the reaction products are quinones. Volatilization may play a role in acenaphthene transport, depending on mixing rates in both the water and air columns. However, adsorption to sediments is probably the dominant aquatic transport process. Consideration of the log octanol/water partition coefficient for acenaphthene and the behavior of other PAHs indicate that acenaphthene can be strongly adsorbed onto suspended and sedimentary particulate matter, especially particulates high in organic content.

Based on information concerning related compounds, it is likely that bioaccumulation of acenaphthene is short term, especially for vertebrates. Although it is rapidly accumulated after

exposure, it also is rapidly metabolized and excreted. Consequently, bioaccumulation is not considered the ultimate fate process for acenaphthene. Based on information for related compounds, it is probable that acenaphthene is readily degraded by microbes. Biodegradation is likely to be more rapid in the soil than in aquatic systems. However, systems indicate that biodegradation may be more important in those aquatic systems that are chronically affected by PAH contamination.

HUMAN HEALTH EFFECTS

Negative results are reported for a test of acenaphthene carcinogenicity based upon neoplastic induction in the newt Triturus cristatus, but the reliability of the test system for predicting mammalian carcinogenicity is not established. Other carcinogenicity studies involving exposure to acenaphthene as one component of complex mixtures of PAHs and other substances report both positive and negative results. However, the relative importance of individual components in the mixtures tested cannot be determined, and no conclusions involving acenaphthene can be drawn. Studies using several different bacterial test systems provide no evidence of mutagenicity. No information concerning its teratogenicity or reproductive toxicity is available.

The most thoroughly investigated effect of acenaphthene is its ability to produce nuclear and cytological changes in a variety of microbial and plant species. Most of these changes, such as increases in cell size and DNA content, are associated with a disruption of the spindle mechanism during mitosis and the resulting induction of polyploidy. However, there is no known correlation between these effects and the biological impact of acenaphthene on mammalian cells.

Very little is known about the human toxicity of acenaphthene. It has been shown to be irritating to the skin and mucous membranes and to cause vomiting if swallowed in large quantities.

In both rats and mice, subchronic oral exposure causes loss of body weight, changes in peripheral blood, increased aminotransferase levels of blood serum, and mild morphological damage to the liver and kidneys. The oral LD_{50} is 10 g/kg for rats and 2.1 g/kg for mice. Kidney and liver damage is greater after subchronic exposure to acenaphthene than after acute exposure.

Toxicity to Wildlife and Domestic Animals

In acute toxicity tests for freshwater organisms, EC_{50} values of 41,200 and 1,700 μg/liter are reported for the cladoceran Daphnia magna and the bluegill, respectively. In saltwater species, 96-hour LC_{50} concentrations for the mysid shrimp and the sheepshead minnow are 970 and 2,230 μg/liter, respectively. A chronic value of 710 μg/liter is reported for the sheepshead minnow, and the acute-chronic ratio for this species is 3.1. No other aquatic life chronic data are available. The freshwater alga Selenastrum capricornutum and the saltwater alga Skeletonema costatum are both relatively sensitive to acenaphthene exposure, with 96-hour EC_{50} values for chlorophyll a and cell number of approximately 525 μg/liter and 500 μg/liter, respectively.

The steady state bioconcentration factor for acenaphthene in the bluegill is 387, with a tissue half-life of less than 1 day. By using the bluegill data and an adjustment factor to allow for differences in lipid content, the bioconcentration factor for acenaphthene and the edible portions of all freshwater and estuarine aquatic organisms consumed by Americans is estimated to be 242. Reports of acenaphthene in foods is limited. One study reports levels of 3.2 μg/kg (the detection limit) or greater in the tissues of shellfish of an unspecified species and location.

A study summarizing the toxicity of a variety of compounds to wild and domestic bird species indicates that the LD_{50} of acenaphthene for the redwinged blackbird is greater than 100 mg/kg. Furthermore, the study reports that acenaphthene did not significantly deter feeding by the blackbird even when it was present in food at relatively high concentrations.

REGULATORY LEVELS AND CRITERIA

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are inadequate for establishing final criteria. EPA did report the lowest valves known to cause toxicity in aquatic organisms.

Freshwater

Acute toxicity:	1,700 μg/liter
Chronic toxicity:	No available data

Saltwater

Acute toxicity:	970 μg/liter
Chronic toxicity:	710 μg/liter

Human Health

EPA Carcinogenic Classification (1):	Not available
Oral Reference Dose (RfD):	6×10^{-2} mg/kg/day
Organoleptic criterion:	20 μg/liter

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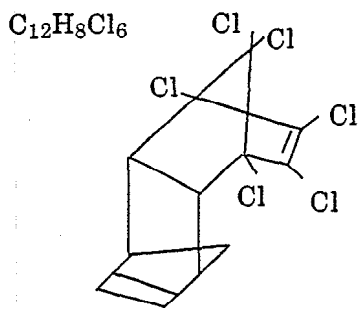
Revisor: Patrick B. Moroney

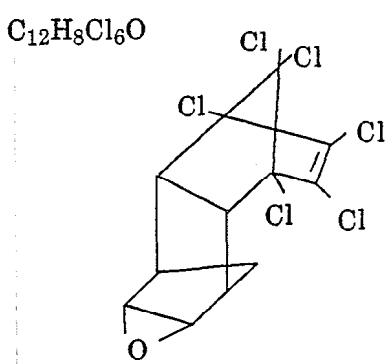
ALDRIN/DIELDRIN

INTRODUCTION

Chemical Name: Aldrin
Synonyms: Aldrex, Aldrite
CAS Number: 309-00-2;
Molecular Formula: $C_{12}H_8Cl_6$
Molecular Weight: 364.90 g/mole
Chemical Structure:



Chemical Name: Dieldrin
Synonyms: Alvit, Dieldrix
CAS Number: 60-57-1
Molecular Formula: $C_{12}H_8Cl_6O$
Molecular Weight: 308.93 g/mole
Chemical Structure:



Aldrin is an insecticide formerly used against termites and soil-dwelling pests such as ants, wireworms, whitegrubs, etc. It is a white crysalline powder in its pure form. The technical grade color is tan. Aldrin readily degrades to dieldrin upon entry into the environment or a

human receptor. Aldrin and dieldrin are structurally similar compounds. Hence, both compounds will be discussed simultaneously. Aldrin is not known to occur naturally (1,2).

Initially in 1970, the U.S. Department of Aqriculture cancelled all uses of aldrin based on the potentially carcinogenic nature of the compounds and the concern that these chemicals could cause severe aquatic environmental change. Aldrin has not been produced or used in the U.S. since 1987. Any past releases have probably been converted to its degradation product, dieldrin, which has also been cancelled for use (1,2).

FATE AND TRANSPORT

Aldrin

Log Koc: 4.69 (1)
Log Kow: 3.01 (1); 6.5 (2)
Half-life: see text
Henry's Law Constant: 3.2×10^{-4} atm-m^3/mole (1); 4.96×10^{-3} atm-m^3/mole (2)
BCF: 4,571 (molluscs) (2)
3,890 (golden orfe)
12,260 (algae)
Degradation Products: Dieldrin (1,2,3)
Solubility:
Water: 0.20 mg/L @ 25°C (1); 0.02 mg/L @ 20°C (2); 0.027 mg/L (3)
Organic Solvents: Very soluble in most organic solvents (1)
Vapor Pressure: 7.5×10^{-5} mm Hg @ 20°C; 1.4×10^{-4} mm Hg @ 25°C (1); 3.75×10^{-5} mm Hg @ 20°C (2); 6×10^{-6} mm Hg @ 25°C (3) Density: 1.7 @ 20°C (3)

Dieldrin

Log Koc: 3.87 (1); 3.87-4.08 (2)
Log Kow: 4.55 (1)
Half-life: 723 days (evaporation at 25°C)(1)
Henry's Law Constant: 1.51×10^{-5} atm-m^3/mole @ 25°C (1)
BCF: 100-10,000 (various aqueous species) (2) 3-6,000 (fish) (2)
Degradation Products: Photodieldrin (1)
Solubility:
Water: 0.18 mg/L @ 25°C (1); 0.2 mg/L @ 25°C (4)
Organic Solvents: Very soluble in benzene; slightly soluble in acetone, alcohol and ether (1)
Vapor Pressure: 1.78×10^{-7} mm Hg @ 20°C; 7.78×10^{-7} mm Hg @ 25°C (1); 2.8×10^{-6} mm Hg @ 20°C (4)
Density: 1.7 @ 20°C (1)

As stated previously, aldrin quickly degrades to dieldrin upon entry into the environment. Aldrin undergoes photolysis to dieldrin, which further degrades by ultraviolet radiation or microbial action into the more persistent photodieldrin (1).

Dieldrin is ubiquitous in the environment and very persistent. This is due to its higher resistance to biotransformation and abiotic degradation than aldrin. Consequently, dieldrin is found in low levels in all media. Dieldrin has a strong affinity for organic matter such as animal fat and plant waxes. Consequently, it bioconcentrates and biomagnifies through the terrestrial and aquatic food chains. This degradation product strongly adheres to soil and sediment, which minimizes mobility in this medium. However, aldrin and dieldrin can

volatilize from soil. Dieldrin is not water soluble. The presence of these compounds in surface water is attributed to runoff from contaminated soil (1).

Aldrin can enter the environment from accidental spills or leaks from storage containers at waste sites. In the past, aldrin entered the environment as a result of pesticide use on crops and use as termite control. Due to use on crops, aldrin and dieldrin have persisted as residue in soil and resulted in crop uptake. Because of the persistence and bioaccumulation of dieldrin, exposure can occur through the ingestion of contaminated water or food. The potential for exposure also exists in homes treated for termites. In this case, exposure via inhalation is significant (1).

PHARMACOKINETICS

Exposure to aldrin and dieldrin can occur via ingestion, dermal contact and inhalation. Inhalation and dermal contact exposure in air is possible due to the volatilization of these aldrin from contaminated surfaces. Hence, these compounds can enter the bloodstream via the stomach, skin, and lungs. As stated previously, aldrin quickly breaks down to dieldrin upon entry into the body. The dieldrin is very persistent in the fatty tissue. It can take a period of weeks to years for the compound to leave the body (1).

A study of the body burden of dieldrin showed that the bioconcentration and rate of elimination of dieldrin were related to the lipid content of the person. The highest levels of dieldrin in adipose tissue were found in the leanest individuals. These individuals also exhibited the lowest total body burden. However, the proportion of the total exposure dose retained in the adipose tissue was highest in those subjects with the greatest total body fat (1).

Placental transfer of dieldrin occurs. A study showed that the level of this pesticide was higher in fetal blood than the mother's blood (1). Aldrin and dieldrin are both toxic to the reproductive system and teratogenic. Reproductive effects include decreased fertility, increased fetal death and effects on gestation. Teratogenic effects include cleft palate, webbed foot and skeletal anomalies (4).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Aldrin and dieldrin cause similar adverse health effects. Short-term exposure to elevated levels of these compounds causes central nervous system excitation resulting in convulsions. Acute exposure can also damage the kidneys. Chronic exposure from an occupational setting also produces convulsions. Exposure to moderate levels of aldrin or dieldrin can cause headaches, dizziness or uncontrolled muscle movements. Animal studies support these health effect observations in humans (1).

It is unlikely that sufficient quantities of these pesticides could be ingested by persons living within vicinity of a hazardous waste site to result in death. Additionally, low levels of these compounds likely to be in air near such a site are significantly lower than the levels necessary to cause death (1).

The USEPA reference dose for chronic oral exposure to aldrin is $3x10^{-5}$ mg/kg/day. The reference concentration for chronic inhalation exposure is not available (2). The oral reference dose for chronic exposure to dieldrin is $5x10^{-5}$ mg/kg/day. The reference concentration for chronic inhalation exposure to dieldrin is not available (3).

Carcinogenic Effects

Epidemiological studies have been inadequate to determine whether aldrin and dieldrin cause cancer in exposed populations because of small sample size and the exposure of subjects to other chemicals in addition to aldrin and dieldrin. However, several studies conducted on mice indicate that aldrin and/or dieldrin may induce carcinoma in the liver. In addition, another study showed an increase in pulmonary, lymphoid and other tumors upon exposure to these pesticides. Most recent data indicates that aldrin and dieldrin may act primarily as tumor promoters (1).

An upper bound cancer slope factor (CSF) of 17 $(mg/kg/day)^{-1}$ for oral exposure to aldrin was estimated by USEPA. The inhalation CSF was 17 (mg/kg/day) (3). The oral CSF for dieldrin is 16 $(mg/kg/day)^{-1}$. The inhalation CSF is 16.1 $(mg/kg/day)^{-1}$ (3). The USEPA weight-of-evidence for aldrin and dieldrin is B2-Probable human carcinogen (2).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Aldrin and dieldrin are both acutely toxic to freshwater species at low concentrations. The LC_{50} values range from 1 to 46 µg/L for different fish species. Saltwater species are also sensitive to these pesticides. The range of LC_{50} values are similar to the freshwater range. The range for aldrin is 2 to 100 µg/L and 1 to 34 µg/L for dieldrin (4).

Terrestrial and Avian

These pesticides, particularly dieldrin, have been associated with large-scale bird and mammal kills in treated areas. Experimental feeding studies have shown that the chemicals are quite toxic to terrestrial wildlife and domestic animals at low levels (4).

REGULATORY LEVELS AND CRITERIA

National aldrin and dieldrin regulatory levels and criteria relative to a risk assessment are established under the Safe Drinking Water Act (SDWA), and the Ambient Water Quality Criteria (WQC).

SUMMARY OF CRITERIA

	Aldrin
Chronic Oral RfD:	$3x10^{-5}$ mg/kg/day (2)
Chronic Inhalation (RfC):	Not Available (2)
USEPA Weight-of-Evidence:	B2-Probable human carcinogen (2)
Oral Slope Factor:	17 $(mg/kg/day)^{-1}$
Inhalation Slope Factor:	17.1 $(mg/kg/day)^{-1}$
SDWA MCLG MCL:	Aldrin in an unregulated contaminant for which USEPA establishes a monitoring requirement, but which does not have an associated final MCLG or MCL (2)
Ambient Water Quality Criteria	
Human Health	
- Water and Fish Consumption	$7.4x10^{-5}$ µg/L (2)
- Fish Consumption Only	$7.9x10^{-5}$ µg/L (2)

Aquatic Organisms	
- Freshwater, Acute	3 µg/L (2,4)
- Freshwater, Chronic	None
- Marine, Acute	1.3 µg/L (2)
- Marine, Chronic	None
ACGIH TLV-TWA:	0.25 mg/m^3 (4)
OSHA PEL-TWA:	0.25 mg/m^3 (4)

Dieldrin

Chronic Oral Exposure (RfD):	5x10^{-5} mg/kg/day (3)
Chronic Inhalation (RfD):	Not Available (3)
USEPA Weight-of-Evidence:	B2-Probable human carcinogen (2)
Oral Slope Factor:	16 (mg/kg/day)$^{-1}$ (3)
Inhalation Slope Factor:	16.1 (mg/kg/day)$^{-1}$ (3)
SDWA MCLG, MCL:	Dieldrin in an unregulated contaminant for which USEPA establishes a monitoring requirement, but which does not have an associated final MCLG or MCL (2)
Ambient Water Quality Criteria	
Human Health	
- Water and Fish Consumption	0.00014 µg/L (5)
- Fish Consumption Only	0.00014 µg/L (5)
Aquatic Organisms	
- Freshwater, Acute	2.5 µg/L (4)
- Freshwater, Chronic	0.0019 µg/L (4)
- Marine, Acute	0.71 µg/L (4)
- Marine, Chronic	0.0019 µg/L (4)
ACGIH TLV-TWA:	0.25 mg/m^3 (4)
OSHA PEL-TWA:	0.25 mg/m^3 (4)

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Revisor: Patrick B. Moroney

ALUMINUM

INTRODUCTION

Chemical Name: Aluminum
Synonyms (1): Alaun; Alumina Fibre; Aluminum Dehydrated; Aluminium Flake
CAS Number (1): 7429-90-5
Molecular Formula (1): Al
Molecular Weight (1): 26.98 g/mole

Aluminum is a silvery white, crystalline solid which is the most abundant component of the earth's crust. It is the third most abundant of all chemicals and does not occur free in nature. Fine powder forms of aluminum are flammable and form explosive mixtures in the air.

HUMAN HEALTH EFFECTS

Aluminum compounds can affect absorption of other elements in the gastrointestinal tract and thereby alter intestinal function. Aluminum inhibits fluoride absorption and may decrease the absorption of calcium and iron compounds and possibly the absorption of cholesterol. It may alter gastrointestinal tract motility through inhibition of acetylcholine-induced contractions which may explain why aluminum-containing antacids often produce constipation.

A progressively fatal neurologic syndrome also has been reported in patients on long-term intermittent hemodialysis treatment for chronic renal failure. The disorder, which typically arises after three to seven years of dialysis treatment, may be due to aluminum intoxication. Sources of 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 dialysis fluid.

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

The chemistry of aluminum in surface water is complex due to the following five properties:

- aluminum is amphoteric; it is more soluble in acidic solutions than basic solutions;
- ions such as chloride and sulfate form soluble complexes with aluminum;
- aluminum can form strong complexes with fulvic and humic acids;
- hydroxide ions can connect aluminum ions to form soluble and insoluble polymers; and,
- under some environmental conditions, aluminum solutions slowly reach equilibrium (4).

Acute tests have been conducted on aluminum at pH values ranging between 6.5 and 9.0 with freshwater species in fourteen genera. In many tests, less than 50% were affected at the highest concentration tested. Some studies found that the acute toxicity of aluminum increased with pH, whereas others found the opposite to be true. Three studies have been conducted on the chronic toxicity of aluminum to aquatic animals. The chronic values of Daphnia magna, Ceriodaphnia dubia, and the fathead minnow were 742.2, 1,908, and 3,288 µg/L, respectively. The diatom, Cyclotella meneghiniana, and the green alga, Selenastrum capricornutum were affected by concentrations of aluminum in the range of 400 to 900 µg/L. Bioconcentration factors from 50 to 231 were obtained in tests with young brook trout and striped bass. At a pH of 6.5 to 6.6, 169 µg/L caused a 24% reduction in the growth of young brook trout, and 174 µg/L killed 58% of the exposed striped bass.

The Ambient Water Quality Criteria indicates that, except possibly where a locally important species is very sensitive, freshwater aquatic organisms and their uses should not be affected adversely, when pH is between 6.5 to 9.0 if the four-day average concentration of aluminum does not exceed 87 µg/L more than once every three years on the average, and if the one-hour average concentration does not exceed 750 µg/L more than once every three years on average.

Terrestrial and Avian

Information regarding the toxicity of aluminum to terrestrial and avian wildlife or domestic animals was not available.

REGULATORY LEVELS AND CRITERIA

ACGIH TLV-TWA (5):

2 mg/m^3 (soluble salts)
2 mg/m^3 (alkyls)
5 mg/m^3 (welding fumes)

OSHA PEL-TWA (6):

15 mg/m^3 (total dust)
5 mg/m^3 (respirable fraction)

Chronic Oral RfD: 1.0 mg/kg/day (7)

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ANTIMONY

INTRODUCTION

Chemical Name:	Antimony
CAS Number (1):	7440-36-0
Molecular Formula (1):	Sb
Molecular Weight (1):	151.18 g/mole

Antimony is a silvery or gray lustrous metal that comes from the ores stibnite, dermasite, tetrahedrite, livingstonite, and jamisonite (1). Stibnite is the most common naturally occurring form of antimony. Antimony is used as a hardening alloy for lead, solder, sheet and pipe, semiconductor technology, production of fireproofing chemicals, ceramics, glassware, and pigments, and pyrotechnics (2,3). Antimony exists in four valence states (−3, 0, +3, and +5) (4). Antimony is a common air pollutant from industrial emissions but exposure in the general population is largely from food (3).

FATE AND TRANSPORT

Antimony occurs as a soluble oxide or as antinomite (+3) salt in most natural water systems. It may change to volatile stibine in a reducing environment. Stibine is very soluble in water, but it is not stable in under aerobic conditions. Sediment beds offer a reducing environment allowing for remobilization of antimony previously removed from solution. Sorption to clays and minerals is the most important mechanism resulting in the removal of antimony from solution. Insoluble forms of antimony compounds may be formed when heavy metals in solution react with antimonite or antimonate. Bioaccumulation represents a minor fate process for antimony. (4)

PHARMACOKINETICS

Antimony is slowly absorbed from the gastrointestinal tract. The metal accumulates in lung tissue when humans are exposed to antimony dust in an occupational setting. Trivalent antimony is concentrated in red blood cells and the liver. Pentavalent antimony is mostly found in plasma. Both trivalent and pentavalent antimony are excreted in feces and urine. More trivalent antimony is excreted in the urine, where more pentavalent antimony is excreted in the feces (3).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Studies have reported that antimony trioxide, antimony trichloride, and antimony pentachloride may be mutagenic. Reproductive studies have indicated that female workers exposed to metallic antimony dust, antimony trioxide, and pentoxide had an increase in incident of gynecological disorders and late spontaneous abortions. Antimony was found in the breast milk, placental tissue, amniotic tissue, and blood of the umbilical cord in exposed workers. Intraperitoneal administration of antimony in rats supported the findings of the human reproductive effects (4).

Cardiovascular effects in humans consist of changes in electrocardiograms, cardiac edema, myocardial fibrosis, and other signs of myocardial structural damage when exposed to either trivalent or pentavalent antimonial compounds. Animal studies have supported these findings (4).

Antimony and/or its compounds are known to cause pneumoconiosis upon inhalation exposure. Chronic exposure by inhalation causes rhinitis, pharyngitis, tracheitis, and bronchitis. Dermatitis may occur in humans dermally exposed to antimony (4). Also transient skin eruptions, termed "antimony spots", may occur in workers with chronic exposure to antimony (3). An oral RfD of 4.0×10^{-4} has been established by EPA.

Carcinogenic Effects

Antimony production workers have demonstrated higher incidences of lung cancer. Also, animal studies involving rats have indicated that antimony trioxide may produce lung and liver tumors (4).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Although criteria have not been established for antimony, the lowest values known to be toxic in aquatic organisms have been reported. These values are as follows (5):

Freshwater:

Acute toxicity:	88 µg/L (proposed criteria)
Chronic toxicity:	30 µg/L (proposed criteria)

Marine:

Acute toxicity:	1,500 µg/L (proposed criteria)
Chronic toxicity:	500 µg/L (proposed criteria)

These values were established from a study involving the exposure of antimony potassium tartrate and antimony trichloride to Daphnia magna. The LC_{50} and EC_{50} values for Daphnia magna and the fathead minnow ranged from 9,000 to 21,900 µg/L. The chronic value for the fathead minnow and Daphnia magna are 1,600 and 5,400 µg/L, respectively. No detectable bioconcentration of antimony by the bluegill was observed (4).

Terrestrial and Avian

Studies regarding the toxicity of antimony to wildlife and domestic animals were not located in the available literature (4).

REGULATORY LEVELS AND CRITERIA

Oral RfD (7):	4×10^{-4} mg/kg/day
EPA Carcinogenic Classification (7):	Group D - not classified as a carcinogen

Ambient Water Quality Criteria (5):	
Water and Organisms	146 μg/L
Organisms Only	45,000 μg/L
MCL	0.006 mg/L
MCLG	0.006 mg/L
OSHA PEL-TWA:	0.5 mg/m^3 (antimony and its compounds as Sb)
ACGIH TLV-TWA:	0.5 mg/m^3 (antimony and its compounds as Sb)

SUMMARY OF CRITERIA

The Oral RfD for antimony was reported as $4.0x10^{-04}$. The study supporting this value involved the exposure of 5 ppm of antimony potassium tartrate in drinking water to rats (7).

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ARSENIC

INTRODUCTION

Chemical Name:	Arsenic
CAS Number:	7440-38-2
Molecular Formula:	As
Molecular Weight:	74.92 g/mole
Chemical Structure:	As

Arsenic (elemental) exists as a silvery to black, brittle, crystalline and amorphous metalloid. Arsenic is used in the production of glass, enamels, ceramics, oil, cloth, linoleum, electrical semiconductors, pigments, fireworks, pesticides, fungicides, veterinary pharmaceuticals and wood preservatives. Arsenic also has been shown to occur in municipal sewage (7).

FATE AND TRANSPORT

BCF (1):	Accumulates to toxic levels in food chain organisms
Degradation Products:	None
Solubility:	
In Water (5):	Insoluble
In Organics (6):	Unknown (5); soluble in nitric acid
Vapor Pressure (6):	1 mm Hg @ 372° C (sublimes)
Specific Gravity (5):	5.727

Arsenic can occur in soil, water, or air. Since it is an element, it cannot be degraded by environmental processes. However, transformation from one arsenic compound to another is possible.

In the environment, arsenic can occur in four different oxidation states (-3, 0, +3, +5). The particular chemical speciation is important in determining mobility. Interconversions between the +3 and +5 states, as well as organic complexation, are most important (8).

In the soil, the concentration and chemical form in which arsenic occurs is affected by pH, soil type and iron and aluminum content of the soil. Lowered pH and reducing conditions tend to favor the development of arsine, a toxic gas comprised of arsenic and oxygen (7).

In the aquatic environment, volatilization is an important mechanism when biological activity or highly reducing conditions favor the production of arsine or methylarsenics. Sorption of arsenic onto sediments is also an important process in aquatic transport processes. While arsenic may cycle considerably in the environment given its mobility, the deep ocean probably serves as a sink for most inorganic arsenic (7).

PHARMACOKINETICS

Human and animal studies have shown that gastrointestinal absorption of arsenic is very high (>90 to 95 percent). Absorption of arsenic via the inhalation and dermal routes is limited in both animal and human studies. In terms of the developing fetus, inorganic arsenic has been shown to rapidly cross the transplacental barrier after oral administration to mice and rats (5).

Most animals and humans tend to clear arsenic rapidly from the blood and other tissues (including the liver, kidneys, and lungs). Arsenic has been shown to be retained in the brain of experimental animals (5). Arsenic has a tendency to accumulate in the skin and desquamous tissues, such as hair and nails of animals (2).

The main route of excretion for absorbed arsenic is via the urine. Studies demonstrate that only six to nine percent of ingested arsenic appears in the feces, indicating nearly complete gastrointestinal absorption of the metal. The biological half-life is on the order of ten hours, with 50 to 80 percent excreted in about three days (2).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Trivalent compounds of arsenic are the principal toxic forms. Arsenic's principal mode of toxic action is at the cellular level, where it affects mitochondrial enzymes that are critical in tissue respiration (2).

Ingestion of large doses of arsenic can be acutely fatal. Symptoms include fever, anorexia, cardiac arrhythmia and eventual cardiovascular failure. Additionally, central nervous system (CNS) effects, including peripheral neuropathy and sensory loss, are usually noted (2).

Chronic long-term exposure is characterized by liver injury. This is usually reflected as jaundice, and may progress to cirrhosis. Also, peripheral vascular disease has been observed in persons chronically exposed to arsenic (2).

USEPA has established an oral RfD of 3×10^{-4} mg/kg/day for arsenic. This is based on keratosis and hyperpigmentation (1).

Carcinogenic Effects

Arsenic has been implicated as a carcinogen by the inhalation route in both animal and human studies.

Studies of populations living near arsenic-using pesticide manufacturing plants were shown to have an increased incidence of lung cancer. Also, case reports of arsenical pesticide applicators have demonstrated an association between arsenic exposure and lung cancer (1).

Evidence for the carcinogenicity of arsenic via oral exposure comes from an epidemiological study where an arsenic-contaminated water supply was associated with a significant increase in cancer of the bladder, lung, liver, kidney, skin and colon (1).

Because of arsenic's carcinogenic potential in humans, the EPA has classified it as a Group A carcinogen-human carcinogen. The carcinogenic slope factor for arsenic by inhalation exposure is 15.1 $(mg/kg/day)^{-1}$. Also, a carcinogenic slope factor of 1.75×10^0 $(mg/kg/day)^{-1}$ has been derived for ingestion exposure to this element (1,4).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

While various forms of inorganic arsenic seem to have roughly similar toxicities in aquatic organisms, they all seem to be much more toxic than the organic forms. Acute toxicity of adult freshwater animals has been shown to occur at arsenic trioxide levels as low as 812 µg/L and as low as 40 µg/L in early life stage organisms (8).

Ambient Water Quality Criteria for the protection of aquatic organisms are as follows: (1)

Freshwater:

Acute Toxicity:	3.6×10^2 µg/L (Arsenic III)
Chronic Toxicity:	1.9×10^2 µg/L (Arsenic III)

Marine:

Acute Toxicity:	6.9×10^1 µg/L (Arsenic III)
Chronic Toxicity:	3.6×10^1 µg/L (Arsenic III)

Terrestrial and Avian

Information on arsenic toxicity among terrestrial wildlife is very limited. However, arsenic poisoning has been known to occur on rare occasions in domestic animals. Arsenic poisoning in domestic animals leads to hyperemia and edema of the gastrointestinal tract, hemorrhage of the cardiac serosal surfaces and peritoneum, and pulmonary congestion and edema (8).

REGULATORY LEVELS AND CRITERIA

The following regulatory levels and criteria have been established for arsenic:

OSHA PEL-TWA (9):	10 µg/m^3 (inorganic)
ACGIH TLV-TWA (10):	0.01 mg/m^3
MCL(1):	0.05 mg/lL
EPA Ambient Water Quality Criteria (5):	
Ingestion of Water and Aquatic Organisms:	2.2×10^{-6} mg/L
Ingestion of Organisms Only:	1.75×10^{-5} mg/L

SUMMARY OF CRITERIA

EPA Carcinogenic Classification (1):	Group A-human carcinogen
Cancer Slope Factor (Inhalation) (1):	15.1 $(mg/kg/day)^{-1}$
Cancer Slope Factor (Oral) (4):	1.75×10^0 $(mg/kg/day)^{-1}$
Oral RfD (1):	3×10^{-4} mg/kg/day

Inhalation RfD (4):	Not Determined
NOAEL (4):	0.009 mg/L

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Date of Last Revision: 11/10/94
Revisor: Rich Hoff

BARIUM

INTRODUCTION

Chemical Name:	Barium
CAS Number:	7440-39-3
Molecular Formula:	Ba
Molecular Weight:	137.3 g/mole
Chemical Structure:	Ba

Barium is a silvery-white, slightly lustrous, somewhat malleable metal. It is used in various alloys, paints, soap, paper, and rubber, and in the manufacture of ceramics and glass. Barium compounds also are used in diagnostic radiology (3,4).

FATE AND TRANSPORT

BCF (1):	Not Significant
Solubility (1):	
In Water:	Decomposes; combines with sulfate present in natural waters to form $BaSO_4$, which has a solubility of 1.6 mg/L @ 20° C
In Organics:	Soluble in alcohol, insoluble in benzene
Vapor Pressure:	Not Reported
Specific Gravity (1):	3.5

Barium is extremely reactive, decomposes in water, and readily forms insoluble carbonate and sulfate salts. It is generally present in surface water and groundwater only in trace amounts. Barium is not soluble at more than a few parts per million in water that contains sulfate at more than a few parts per million. It is rare to find barium in drinking water at concentrations in excess of 1 mg/L (1). In terms of atmospheric transport, barium can adhere to particulates and be transported via the wind (1).

PHARMACOKINETICS

The absorption, metabolism and excretion of barium and its compounds depend upon the solubility of the specific barium compound. Insoluble forms, such as barium sulfate, are not toxic by either ingestion or inhalation. The soluble salts of barium are absorbed, and small amounts are accumulated in the skeleton (3). However, the biological half-life for barium is generally less than 24 hours (1).

The primary route of barium elimination is through the feces (3). Urinary excretion of barium occurs to a much lesser extent because it is reabsorbed by the renal tubules of the kidney.

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Soluble salts of barium, such as barium chloride and barium carbonate, are highly toxic by exposure via ingestion or inhalation (1).

The most important effect of acute barium poisoning is a strong, prolonged stimulant action of muscle including smooth, cardiac, and skeletal muscles. This tends to lead to a transient increase in blood pressure due to vasoconstriction (1,3).

Effects on the hematopoietic system and cerebral cortex have also been reported in humans. Accidental ingestion of soluble barium salts has resulted in gastroenteritis, muscular paralysis and ventricular fibrillation (1).

Baritosis, a benign form of pneumoconiosis, is an occupational disease arising from the inhalation of barium sulfate, barium oxide, or barium carbonate dust (1,3).

The EPA has established an oral RfD for barium of 7×10^{-2} mg/kg/day, based on increased blood pressure. EPA has placed a confidence rating of "medium" on this RfD (2). This value is based on a study involving the administration of barium chloride in drinking water to 11 male volunteers. The volunteers were given 1.5 L/day of distilled and charcoal-filtered water containing 0 mg/L barium for the first two weeks, 5 mg/L for weeks 3 to 6, and 10 mg/L for weeks 7 to 10. There were no changes in systolic or diastolic blood pressures or serum chemistry. There was an increase in serum calcium levels that was attributed to a decrease in serum albumin levels. However, these increases were not clinically significant (2). The RFDi was based on a four month inhalation study in rats that demonstrated fetotoxicity. A NOEL of 0.8 mg/cu m was identified in this study (5).

Carcinogenic Effects

Currently, there are no reports of carcinogenicity associated with exposure to barium (1). The EPA has not evaluated barium with respect to carcinogenic potential.

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Information regarding the aquatic impact potential of barium was not readily available (1).

Terrestrial and Avian

Adequate data for characterization of toxicity to wildlife and domestic animals are not available (1).

REGULATORY LEVELS AND CRITERIA

Maximum Contaminant Level (MCL) (2):	2.0 mg/L
MCLG (2)	2 mg/L
OSHA PEL-TWA (1):	0.5 mg/m^3 (soluble compounds)

ACGIH TLV-TWA (1):	0.5 mg/m^3 (soluble compounds)
EPA Carcinogenic Classification (2):	Group D-not classified as a carcinogen
Oral RfD (2):	7×10^{-2} mg/kg/day
Inhalation RfD(5):	1.43×10^{-4} mg/kg/day

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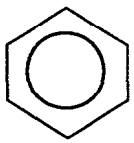
Date of Last Revision: 11/10/94
Revisor: Patrick B. Moroney

BENZENE

INTRODUCTION

Chemical Name:	Benzene
CAS Number:	71-43-2
Common Name:	Annulene, Benzol, Coal naphtha
Molecular Formula:	$C_6 H_6$
Molecular Weight:	78.12 g/mole
Chemical Structure:	



Benzene appears colorless to light yellow. It is a mobile, nonpolar liquid with a high refractive nature. In the vapor state it burns with a smoky flame emitting an aromatic odor (2).

Benzene is used as a solvent and in the manufacturing of rubber, in oil refineries, chemical plants, retail stations, and shoe manufacturing (2).

FATE AND TRANSPORT

K_{oc} (2):	0.3-100
Log K_{ow} (4):	1.95 - 2.15
$t_{1/2}$ (6) :	Expressed as degradation rate of 200-330 mg/L/day (estuarine)
Henry's Law Constant (7):	5.5×10^{-3} atm- m^3/mole
BCF:	Not Available
Degradation Products (7):	Benzene glycol, catechol
Solubility (7):	
In Water:	1780 mg/L at 25°C
In Organics:	Miscible
Vapor Pressure (1):	75 mm Hg at 20°C
Specific Gravity (1):	0.879 at 20°C

Benzene may appear in the ambient air, water, and soil. Although benzene is released into the environment by both natural and man-made sources, the contribution from the man-made source is most significant (4).

The combustion of gasoline is the most significant source of benzene release. Other minor sources are septic tank effluent, structural fires, and exhaled air of smokers (4).

Volatilization is the major transport process while atmospheric destruction of benzene is the most likely fate process (4).

In both soil and surface water, sorption is the primary removal method. Although bioaccumulation of benzene is low, the rate of biodegradation is enhanced by the presence of other hydrocarbons (4).

PHARMACOKINETICS

Benzene can be absorbed into the body by inhalation, ingestion, and dermal contact. Metabolic transformation must occur before benzene can exert its toxic effect. Benzene is distributed to the blood (approx. 30%) and to the bone marrow, adipose tissue, and liver (>50%) (4). Benzene exposure may lead to immunosuppression or sensitization, and cause neurotoxic effects (4).

Humans expire unmetabolized benzene in the breath and phenolic metabolites in the urine. The liver is the major site of benzene metabolism.

Humans eliminate unchanged benzene in exhaled air and as benzene metabolites in urine. Only a small amount of benzene is excreted in the feces (4).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Acute exposure to moderate concentrations of benzene may cause drowsiness, dizziness, headache, and nausea. If exposure continues, unconsciousness may occur (4). Long-term exposure to benzene may affect normal blood production resulting in severe anemia and internal bleeding (4). Prolonged or repeated dermal absorption of benzene may cause blistering, erythema, and dermatitis (4).

In humans, there is not sufficient evidence to link benzene to spontaneous abortions and miscarriages in pregnant women. Animal studies indicate adverse health effects on unborn test animals (4).

Benzene is genotoxic, causing structural chromosomal aberrations (4).

Carcinogenic Effects

The EPA has classified benzene as a Group A carcinogen - a human carcinogen. It has been determined that prolonged exposure to benzene vapors can result in the development of leukemia (4). The primary epidemiogical study supporting the carcinogenic effects from benzene inhalation indicates that the exposure duration ranged from less than five years to as many as 30 years. Based on this primary study, the EPA has derived a unit risk of 8.3×10^{6} per $\mu g/m^3$ from which, an inhalation cancer slope factor of 2.9×10^{-2} $(mg/kg/day)^{-1}$ can be derived (3). Although benzene has been shown to be carcinogenic via the inhalation route, data relating the ingestion or dermal route of exposure to carcinogenic effects is insufficient (4). However, the EPA has derived an oral cancer slope factor of 2.9×10^{-2} $(mg/kg/day)^{-1}$ based on the risk posed from the inhalation route of exposure.

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

The available data for benzene indicate that acute toxicity to freshwater life occurs at concentrations

as low as 5,300 μg/L. For saltwater aquatic life, acute toxicity occurs at concentrations as low as 700 μg/L (5).

No data concerning chronic exposure to benzene in aquatic organisms is readily available (5).

Terrestrial and Avian

Information regarding the toxicity of benzene to terrestrial and avian wildlife and domestic animals was not found in the available literature.

SUMMARY OF REGULATORY LEVELS AND CRITERIA

EPA Carcinogenic Classification (3):	Class A - Human carcinogen
AWQC (3):	
Ingestion of Water and Organisms:	6.6×10^{-1} μg/L
Ingestion of Organisms Only:	$4.0 \times 10^{+1}$ μg/L
Reportable quantity(3):	10 lbs
ACGIH TLV(4):	10 ppm
OSHA TWA(4):	1 ppm
OSHA STEL(4):	5 ppm
Cancer Slope Factor (oral) (3):	2.9×10^{-2} $(mg/kg/day)^{-1}$
Cancer Slope Factor (inhalation) (3):	2.9×10^{-2} $(mg/kg/day)^{-1}$
Oral RfD:	Not Applicable
Inhalation RfD (8):	1.71×10^{-3}
MCL (drinking) (3):	5 μg/L
MCLG (3):	0 μg/L

SUMMARY OF TOXICOLOGICAL INDICES (3)

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Date of Last Revision: 12/14/94
Revisor: Patrick B. Moroney

BERYLLIUM

INTRODUCTION

Chemical Name:	Beryllium
CAS Number:	7440-41-7
Molecular Formula:	Be
Molecular Weight:	9.01 g/mole

Beryllium is a dark gray metal from the alkaline earth family. Most of the beryllium ore that is mined is converted into metal alloys which are used in the fields of electronics and metal fabrication. Pure beryllium is used in nuclear weapons and reactors and x-ray transmission windows and mirrors (4).

FATE AND TRANSPORT

BCF:	100 for freshwater and marine plants, invertebrates, and fish (4)
Solubility:	
In Water:	Insoluble
In Organics (1):	Soluble in dilute acid and alkali; insoluble in alcohol, ether, and CCl_4
Specific Gravity (1):	1.85 @ 20° C

Beryllium can be present in the air, water, and soil although it can not be degraded by environmental fate processes. However, it may transform from one beryllium compound to another. The major source of its emissions to the environment is the release of particulates and fly ash into the atmosphere through the combustion of coal and fuel oil. Other sources of emission are associated with ore processing, metal fabrication, and beryllium oxide production and use. Beryllium is most likely found in the environment in the particulate form rather than the dissolved form (4).

There is no information regarding the aquatic or soil biotransformation of beryllium and its compounds (4).

The removal of beryllium from the atmosphere occurs through wet and dry deposition. Because beryllium displaces divalent cations which share common sorption sites, in most types of soil, it is expected to be tightly adsorbed. Since the BCF is reported at <1000, beryllium will not significantly bioaccumulate (4).

PHARMACOKINETICS

Beryllium enters the body through inhalation of air, consumption of food, and contact with water. The primary route of exposure is by inhalation. Based on animal studies, pulmonary absorption does not appear to be extensive, and absorption through the gastrointestinal tract and the skin appears to be minimal. Dermal exposure to soluble beryllium compounds can cause contact dermatitis in humans (4).

Animal studies indicate that once beryllium enters the body via inhalation, it is circulated in the blood as an orthophosphate colloid and then distributed to the bone, liver, and kidneys in both humans and animals. Oral exposure of soluble beryllium in animal studies showed that distribution of the compound was mainly to the liver and intestine whereas insoluble beryllium was found mainly in the intestine.

Although beryllium is not biotransformed, soluble beryllium compounds are partially converted to more insoluble forms in the lungs (4).

Due to the poor absorption of beryllium compounds through the gastrointestinal tract, excretion of beryllium after oral administration of the compound is primarily in the feces, as demonstrated in animal studies. Following oral exposure to animals, only a very small amount appears in the urine; however, if beryllium is inhaled, a large portion of the absorbed beryllium is excreted in the urine (4).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

The most profound effect from inhalation appears in the lungs. Exposure to beryllium oxide or to soluble beryllium compounds may lead to the development of acute chemical pneumonitis. Beryllium and chemical pneumonitis in humans may result from exposures ranging from < 2 to 1000 $\mu g/m^3$. Chronic exposure to insoluble forms may lead to berylliosis. The most common clinical symptom of berylliosis is a granulomatous lung inflammation. Additional respiratory effects are rhinitis, pharyngitis, and tracheobronchitis. Besides the lung, beryllium also accumulates in the bone, liver and kidneys (1,4). No studies were identified describing the toxicity of beryllium when administered to humans by the oral route (4).

Beryllium can be absorbed through the skin; however, this route is minor. Exposure levels producing skin lesions associated with acute dermal exposure were not available (4). Also, direct contact with the eyes may cause inflammation to the conjunctiva (1).

No human data were available concerning decreased longevity. One study involving mice reported acute oral LD_{50}s of 18 to 20 mg/kg. The EPA has verified an oral RfD of 0.005 mg/kg/day. This figure was based on a study where rats were exposed to beryllium sulfate in drinking water at a concentration of 5 mg/L. A NOAEL was calculated at 5 mg/L (3).

Carcinogenic Effects

This chemical is among the substances classifed as a probable human carcinogen. This classification is based on limited evidence of carcinogenicity in humans from epidemiologic studies (3). Studies indicating increased lung cancer in individuals who have been exposed to beryllium failed to account for confounding factors such as lung cancer associated with smoking, and did not include employees of the beryllium industry who were not exposed to the beryllium (control groups) (4).

There has been sufficient information to link beryllium to lung cancer via inhalation in animals; osteosarcomas have been observed in rabbits via intravenous or intramedullary injection (3). Also, there has been sufficient information to link beryllium to an induction of tumors via inhalation and

intratracheal instillation in rats and monkeys, and the induction of osteosarcomas in rabbits by intravenous or intramedullary injection in multiple studies.

Slight increases in cancer incidence were reported in Long-Evans rats that were administered 5 ppm beryllium sulfate in drinking water for a lifetime. An increase in reticulum cell carcomas of the lungs was seen in male Wistar-derived rats administered beryllium sulfate in the diet at 5 and 50 ppm, but not at 500 ppm.

Osteogenic sarcomas were induced in rabbits by intravenous injection of beryllium compounds. Bone tumors were induced by beryllium oxide, zinc beryllium silicate, beryllium phosphate, beryllium silicate, and beryllium metal.

Lung tumors (adenomas and adenocarcinomas) have been induced via the inhalation route in both male and female Sprague-Dawley rats during exposure periods of up to 72 weeks by beryllium sulfate, in Sherman and Wistar rats by beryllium phosphate, beryllium fluoride, and zinc beryllium silicate, in male Charles River CR-CD rats by beryl ore, and in both male and female rhesus monkeys by beryllium sulfate. Positive results were seen in rats exposed to beryllium sulfate at concentrations as low as 2 $\mu g/m^3$ (3).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Available data are not adequate to develop ambient water quality criteria for aquatic life protection. However, the EPA has reported the lowest concentration of beryllium known to cause toxic effects. The criterion for acute beryllium exposure to freshwater organisms is 130 $\mu g/L$. The chronic exposure for freshwater organisms is 5.3 $\mu g/L$. No saltwater acute or chronic studies are available (4).

Terrestrial and Avian

No data are available for terrestrial and avian life forms (4).

REGULATORY LEVELS AND CRITERIA

OSHA Ceiling Limit (4):	5 $\mu g/m^3$
OSHA PEL-TWA (4):	2 $\mu g/m^3$
ACGIH TLV-TWA (4):	2 $\mu g/m^3$
Ingestion of water and fish (3):	6.8×10^{-3} $\mu g/L$
Ingestion of organisms only (3):	1.17×10^{-1} $\mu g/L$
Reportable quantity (3):	10 lbs
MCL (3):	0.004 mg/L
MCLG (3):	0.004 mg/L

SUMMARY OF CRITERIA

EPA Carcinogenic Classification (3):	Group B2-Probable human carcinogen
Cancer Slope Factor (Oral) (3):	4.3 $(mg/kg/day)^{-1}$

Cancer Slope Factory (Inhalation) (3):	8.4 $(mg/kg/day)^{-1}$
Oral RfD (3):	0.005 mg/kg/day
Inhalation RfD (3):	Not available
NOAEL (7):	50 mg/kg/day

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Date of Last Revision: 11/10/94
Reviser: Patrick B. Moroney

BIS (2-ETHYLHEXYL) PHTHALATE

INTRODUCTION

Chemical Name: Bis (2-ethylhexyl) phthalate
CAS Number (1): 117-81-7
Molecular Formula (1): $C_{24}H_{38}O_4$
Molecular Weight (1): 390.5 g/mole

Bis (2-ethylhexyl) phthalate (BEHP) is a man-made chemical that is added to plastics to increase their flexibility. This compound is essentially a colorless liquid. BEHP is also known by the tradenames of Palatinol AH, Octoil, Sicol 150, Bisoflex 81, and Eviplast 80. It is present in a variety of plastics, especially vinyl materials, which have BEHP contents as high as 40 percent. Materials containing BEHP include rainwear, footwear, upholstery materials, imitation leather, waterproof gloves, tablecloths, shower curtains, food packaging materials, floor tiles, paints, flexible tubing, and plastic bags. BEHP has been detected in commercial organic solvents (2).

FATE AND TRANSPORT

Log K_{ow} (1):	5.11
Surface Water $t_{1/2}$: (2):	2-3 weeks
Log K_{oc} (1):	4 to 5
Henry's Law Constant (1):	1.1×10^{-5} atm·m^3/mole
BCF (1):	100 to 10,000 for fish and invertebrates
Water Solubility (1):	0.3 mg/L at 25° C
Vapor Pressure (1):	6.45×10^{-6} mmHg at 25° C

BEHP is released into the atmosphere through emissions from manufacturers and other industries and will exist in the gaseous phase. It is also released to the atmosphere via volatilization from plastic materials and the burning of plastics (2). BEHP released to the air will be carried for significant distances in the troposphere with wash out by rain being a significant removal process (1).

If released into a water system, BEHP biodegrades fairly rapidly with a half-life of approximately 2-3 weeks. BEHP also will strongly adsorb to soils and sediments and accumulate in aquatic organisms (1). As a result, evaporation and hydrolysis are not significant removal processes in the aquatic environment.

As mentioned above, BEHP has a strong tendency to adhere to soils and sediments. Therefore, when BEHP comes into contact with surface soils, release via evaporation or leaching into groundwater is negligible. Some studies suggest that BEHP may biodegrade in soil under aerobic conditions following acclimation.

PHARMACOKINETICS

The absorption of BEHP following inhalation, dermal contact, and oral exposure has been studied in humans and/or animals. Absorption following exposure to airborne BEHP has not been quantified in either humans or animals although data suggest it is absorbed via this route (2). Oral exposure in humans has resulted in urinary excretion of 11-15% of the administered dose. However, the total oral absorption rate is probably higher (approximately 20-25%) since it has been demonstrated in animals that 15-20% of the absorbed dose is eliminated via the bile (2). In terms of dermal absorption, the available animal studies indicate that absorption is poor via this pathway. One study suggests a dermal absorption rate of BEHP of approximately five percent. No data are available on the absorption of BEHP following dermal exposure in man (2).

No studies are available describing the distribution of BEHP and its metabolites following exposure in man. The tissue distribution in rats and monkeys is mainly to the liver, kidney, blood, and testes following oral exposure to BEHP. Dermal contact in rats resulted in the largest accumulation in the muscle, followed by the liver, kidney, and fat. No data were available regarding distribution of BEHP in animals after inhalation exposure (2).

In terms of BEHP metabolism, the available data in both humans and animals suggest that a complex series of reactions occur resulting in the production of 30 or more metabolites. The initial metabolic stop involves the lipolytic cleavage of DEHP to mono(2-ethylhexyl)phthalate (MEHP) and, after a series of primary metabolic reactions, the oxidized derivatives of MEHP undergo glucuronidation prior to excretion. It should be noted that the integrity of the aromatic phthalic acid moiety is maintained during these metabolic conversions (2).

The excretion of BEHP varies with the route of primary exposure. Following oral exposure to BEHP, a majority of the metabolites are excreted in the urine with lesser quantities eliminated via the bile in both humans and animals. A similar pattern of excretion was evident following dermal exposure in rats. No data are available regarding BEHP excretion following inhalation exposure in humans or animals (2).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

There are currently no data available suggesting that human exposure to BEHP results in the development of systemic effects. However, animal data suggest that exposure may affect the liver, testes, kidney, thyroid, and pancreas (2).

Oral exposure of rodents to BEHP has resulted in significant effects on the liver. Hepatic effects of BEHP in rodent systems includes increased hyperplasia, decreased cholesterol synthesis and degredation, alterations in the morphology of bile ducts, and appearance of precancerous altered cell foci, nodules, and tumors of the liver. The degree of liver perturbation is a function of both the dose and duration of exposure (2).

A number of studies in rats have demonstrated that BEHP exposure results in increased weight of the testes, prostate, seminal vesicles, and epididymus. The severity of testicular damage appears to be greater in younger than older male rats (2).

Chronic exposure to BEHP in animals has resulted in focal cystic changes of the kidney cells, decreased creatinine clearance, and an accumulation of lipofuscin deposits in the tubular epithetial cells (2).

The U.S. EPA has developed an oral reference dose (RfD) for BEHP based on an increased relative liver weight in guinea pigs (3). The guinea pigs were orally fed BEHP-containing feed and a lowest observed adverse effect level (LOAEL) of 19 mg/kg/day was established. An uncertainty factor of 1,000 was applied to the LOAEL to derived an oral RfD of 2×10^{-2} mg/kg/day for BEHP (3).

Carcinogenic Effects

The U.S. EPA has classified BEHP as a Group B2 carcinogen -- probable human carcinogen. This classification is based on sufficient evidence of carcinogenicity in animals and inadequate evidence in humans (3).

Based upon an NTP study involving Fisher 344 rats and B6C3F1 mice, an oral slope factor for BEHP was derived. Male and female rats were fed doses of 0, 6,000 and 12,000 ppm BEHP for 103 weeks and examined histologically for evidence of carcinogenicity. The histological examination revealed a statistically significant increase in the incidence of hepatocellular carcinomas and combined incidence of carcinomas and adenomas. A positive trend between increasing dose and increased cancer incidence also was apparent. An oral slope factor of 1.4×10^{-2} $(mg/kg/day)^{-1}$ was developed by the U.S. EPA using the linearized multistage model (3). There is currently no slope factor available for assessing carcinogenicity of BEHP by the inhalation route.

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Date of Last Revision: 2/23/95

Revisor: Patrick Moroney

2-BUTANONE

INTRODUCTION

Chemical Name: 2-butanone

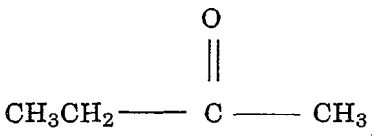
Synonyms (1): Methyl Ethyl Ketone, MEK, Methyl acetone

CAS Number (1): 78-93-3

Molecular Formula (2): C_4H_8O

Molecular Weight (2): 72.10 g/mole

Chemical Structure (1):



2-Butanone is a colorless liquid which has an acetone-like odor. It is derived by distillation from mixed n-butylenes and sulfuric acid, by controlled oxidation of butane, and by fermentation. It's primarily used is as a solvent in the coatings industry to manufacture gums, resins, and nitrocellulose (3). 2-Butanone is also produced to make cements and adhesives, printing ink, cleaning fluids, smokeless powder and wax (1).

FATE AND TRANSPORT

Log K_{ow} (1): 0.29

K_{oc} (4): 4.5 ml/g

$t_{1/2}$ (4):

Air: 0.58 days

Surface Water: 10 days

Henry's Law Constant (1): 4.35×10^{-5} atm-m^3/mol

BCF (1): 9.00×10^{-2}

1.86

Solubility:

In Water (1): 353,000 mg/L @ 10°C

In Solubility (7): Miscible with alcohol, ether, benzene and acetone

Vapor Pressure: 70.6 mm Hg @ 20°C (1)

90.6 mm Hg @ 25°C (2)

Density (1): 0.8050 g/ml @ 20°C

Vapor Density (7): 2.41

Specific Gravity (7): 0.805 @ 20°C

2-Butanone is released into the atmosphere through emissions from manufacturers and other industries and will exist in the gaseous phase. Degradation occurs by reaction with photochemically produced hydroxyl radicals resulting in acetaldehyde. 2-Butanone has been detected in rainwater and therefore, is assumed that it may be removed from the atmosphere through a rain episode. 2-Butanone may also be subject to direct photolysis (2).

If released into a water system, it will evaporate into the atmosphere within 3 days if released to a river, and 12 days if released to lakes. Because of its aliphatic nature, biodegradation will occur in both freshwater and saltwater; however, the process will be slow. Biodegradation studies have suggested that 2-butanone may slowly biodegrade in groundwater after a long acclimation period. 2-Butanone will not significantly hydrolyze, photooxidize, adsorb to sediment, or bioconcentrate in aquatic organisms (2).

Upon release to the soil, 2-butanone will partially evaporate to the atmosphere and may subsequently leach into groundwater. Biodegradation screening studies have indicated that 2-butanone may be subject to slow biodegradation in soil and groundwater (2).

PHARMACOKINETICS

Information addressing the absorption, distribution, metabolism, and excretion of 2-butanone was not available in the reviewed literature.

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

When Spraque-Dawley rats were exposed to 1000 to 3000 ppm 2-butanone vapors for 7 hours per day on days 6 through 15 of gestation, no maternal toxicity effects were observed; however, fetal development and increase in the number of anomalous skeletons were apparent. The fetal anomalies were exhibited as acaudia (no tails), imperforated anus, and brachygnathia (shortened lower jaw). No embryotoxicity or teratogenicity effects were observed in the rats (1).

Short-term acute exposure to animals caused irritation to eyes, nose, and throat, central nervous system depression, emphysema, and congestion of the liver and kidney (1).

One animal study involving the infusion of 2-butanone into the circulatory system of rats at a rate of 70 µm/kg/min for 60 minutes, showed depression of vestibulo-ocular reflex (VOR). Depression of VOR is considered an early sign of intoxication prior to the onset of a general depression of the central nervous system (1).

Chronic toxicity in Fisher 334 rats exposed to 5000 ppm 2-butanone for 6 hours per day, 5 days per week for 90 days, consist of a decrease in mean body weight, increase in liver weight and liver to body weight ratios, depression of brain weight in females, decreased serum glutamic-pyruvic transaminase activity and increased alkaline phosphatase, potassium, and glucose values in females (1).

Studies have suggested that potentiation of hexane-induced neurotoxicity by 2-butanone was the result of the persistence of the seurotoxic metabolite in the blood and not the enhanced biotransformation of precursor hexacarbons to 2,5-hexanedione (1).

Occupational studies have indicated that adverse health effects can be associated with overexposure in the workplace. An incidence was reported where a male working in a confined space during paint removal suffered optic nerve toxicity induced by methanol formed from the metabolism of 2-butanone (1). Also, factory workers using 2-butanone suffered from severe dermatitis and numbness in the fingers, arms and legs. These symptoms disappeared after the use of 2-butanone was discontinued (1).

Human studies addressing skin exposure has shown that partial dehydration of the stratum corneum occurs upon exposure. Also, spontaneous trancient whitening of the skin is caused by excessive exposure to 2-butanone due to the change in structure and removal of skin lipids rather than vasoconstriction (1).

In case studies involving humanomit addiction to inhaled 2-butanone solvents, severe toxic polyneuropathy was apparent. The peripheral motor defects required 2 to 3 years to appear after the exposure was initiated. These effects were due to an axonal transmission disorder which destroyed peripheral and central axons (1).

Findings that defend that neurotoxic properties of hexane can be potentiated by 2-butanone have been supported by the "Berlin Poisoning Affair". In this incident, a solvent manufacturer changed the formula of a solvent thereby reducing the hexane content; however, 11% 2-butanone was added as a denaturant. When released to the public, neurological effects, which included weight loss, muscular weakness affecting extremities or paralysis of extremities, extreme muscular atrophy, and respiratory disorders, were reported. The original formula which did not contain the 2-butanone did not cause the above health effects. However, it unclear whether the observed effects resulted from the 2-butanone alone or from a combination of the 2-butanone and hexane (1).

Carcinogenic Effects

The EPA has classified 2-butanone as a Group D carcinogen - not classifiable as to human carcinogenicity. The basis for this classification is the lack of human carcinogenicity data and inadequate animal data (5).

Studies addressing the carcinogenic potential of 2-butanone by the oral or inhalation routes were not available. One study reported results of the dermal exposure of two groups of male C_3H/He mice. These mice were exposed to 50 mg of solution containing 25 or 29% 2-butanone in 70% dodecylbenzene twice a week for 1 year. The group treated with 25% 2-butanone developed no skin tumors. One tumor was reported in the group treated with 29% 2-butanone (5).

Animal studies have revealed that there is no evidence of genotoxicity when tested in TA102 and TA104 strains of Salmonella typhimurium (1). Studies involving Chinese V79 hamster cells proved to be marginally positive. This assay demonstrates the ability of a compound to

inhibit gap junction-mediated intercellular communication (1). Also, 2-butanone strongly induces mitotic aneuploidy in diploid yeast strain D61.M of Saccharomyces cerevisiae (1). And finally, no significant increases in micronucleated polychromatic erythrocytes in the bone marrow cells of Chinese hamster receiving 411 mg/kg 2-butanone intraperitoneally were observed (1).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

EPA ambient water quality criteria have not been established for 2-butanone. However, an LC_{50} concentration of 5,600 µg/L was reported for two freshwater fishes. Also, an LC_{50} concentration for brine shrimp was reported to be 1,950 mg/L (7).

Terrestrial and Avian

No information on the toxicity of 2-butanone to terrestrial wildlife or domestic animals was found in the available literature (7).

REGULATORY LEVELS AND CRITERIA

OSHA PEL (8-hr TWA) (1):	200 ppm
OSHA STEL (15-min) (1):	300 ppm
NIOSH IDLH (30-min) (1):	3,000 ppm
NIOSH REL (10-hr TWA) (1):	200 ppm
ACGIH TLV (R) (8-hr TWA) (1):	200 ppm
ACGIH STEL (15-min) (1):	300 ppm
EPA Health Advisories (1):	
1-day (child):	80 mg/L
10-day (child):	8 mg/L
longer-term (child):	3 mg/L
longer-term (adult):	9 mg/L
lifetime (adult):	0.2 mg/L

SUMMARY OF CRITERIA

Subchronic Oral RfD (6):	2.0 mg/kg/day
Chronic Oral RfD (5):	0.6 mg/kg/day
Subchronic Inhalation RfD (6):	0.286 mg/kg/day
Chronic Inhalation RfD (5):	0.286 mg/kg/day
EPA Carcinogenic Classifications (5):	Group D-not classifiable as to human carcinogenicity

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Date of Last Revision: 12/14/94
Revisor: Patrick B. Moroney

CADMIUM

INTRODUCTION

Chemical Name:	Cadmium
CAS Number:	7440-43-9
Molecular Formula:	Cd
Molecular Weight:	112.4 g/mole

Cadmium is a soft, blue-white, malleable metal or grayish-white powder. It is obtained as a by-product from the treatment of copper, lead, and iron (1). Its primary uses are as a pigment, and in batteries, specialized metal alloys, and photography (2).

FATE AND TRANSPORT

BCF:	81 (4)
Solubility (4) :	Salts are water soluble; metal is insoluble
Specific Gravity (1):	8.642

Cadmium can be present in the air, water, and soil. Most often, cadmium exists in small particles which tend to be more persistent. Photochemical reactions are not involved in the atmospheric fate of cadmium. The largest source of atmospheric cadmium occurs as a result of fossil fuel combustion (4).

In water, cadmium is relatively mobile and may exist as a hydrated ion or as metal inorganic complexes. Many inorganic cadmium compounds are soluble in water; however, cadmium oxide and sulfide have very low solubilities (4).

Cadmium exists in soil as free cadmium compounds. It is strongly accumulated by organisms through food and water. The bioaccumulation capabilities of cadmium allow for elevated cadmium concentrations in mollusks, crustaceans, fish, and aquatic plants. Elevated cadmium levels in beef and poultry are often a result of cadmium-containing fertilizers (4).

PHARMACOKINETICS

Cadmium readily enters the body through ingestion of water or food and the inhalation of ambient air. Increased cadmium levels are largely attributed to plumbing, municipal incinerators, coal combustion, food grown in phosphate fertilizers, and smoking. Dermal exposure to cadmium is not considered a significant pathway (4).

Absorption of inhaled cadmium through the lung is dependent on particle size and whether or not the particles are respirable. It has been estimated that roughly 30 to 60 percent of inhaled cadmium is absorbed. Cadmium ingested in food or water is poorly absorbed (one to six percent) from the gastrointestinal tract of humans (4).

Cadmium is distributed to most organs in the body; however, the areas of greatest accumulation are the kidneys and liver. The primary route of excretion is through the urine. However, since only a small amount of cadmium is excreted per day, there is a strong tendency for cadmium to accumulate in the body over time. The half-life in humans ranges from 17 to 38 years (4).

Cadmium is not known to undergo any direct metabolic conversions in vivo such as oxidation, reduction, or alkylation. However, the cadmium ion does bind to protein and nonprotein sulfhydryl groups and to anionic groups in proteins and other molecules (4).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

The primary adverse health effect in humans associated with long-term cadmium exposure is renal dysfunction. Manifestation, associated with abnormal renal function include the formation of renal stones, formation, disturbances in mineral metabolism, and renal functional lesions (4).

In the industrial environment, acute pulmonary exposure may lead to pneumonitis and severe pulmonary edema. Also, bone disorders, such as, osteomalacia, osteoporosis, and spontaneous bone fracture, have been observed in humans who are chronically exposed to cadmium (4). In recent studies, it has been found that relatively low doses of cadmium can alter the immune response in experimental animals; however, there is little evidence for suppression of the immune response in chronically exposed human populations (4). Other systemic effects include immunosuppression in animals, anemia, possible effects on the endocrine system, and defects in sensory function in humans. The LD_{50} in rats is 225 mg/kg (4). Cadmium is also an animal teratogen and reproductive toxin in laboratory animals.

The EPA has established an oral RfD values of 5.0×10^{-4} mg/kg/day (water) and 1.0×10^{-3} mg/kg/day (food). A concentration of 200 μg cadmium/gm wet human renal cortex is the highest renal level not associated with significant proteinuria. The toxicokinetic model assumes that 0.01% day of the cadmium body burden is eliminated per day. Assuming 2.5% of absorption of cadmium from food or 5% from water, the toxicokinetic model predicts that the NOAEL for chronic cadmium exposure is 0.005 and 0.01 mg cadmium/kg/day from water and food, respectively.

Carcinogenic Effects

Cadmium is classified as an EPA Group B1 carcinogen, a probable human carcinogen by inhalation. An Inhalation Cancer Slope Factor of 6.3 has been established by EPA (3).

There is a significant association between cadmium and bronchogenic carcinomas and total malignant neoplasms in animals (4). Also, there is limited evidence which links cadmium inhalation exposure to an increased incidence of prostate cancer in exposed workers.

Studies with experimental animals have shown cadmium to produce significant increases in lung tumors. Also, administration of cadmium by injection has led to tumor formation at the injection site (4). Based on a large number of studies, it has been hypothesized that cadmium is not directly mutagenic, but impedes genetic repair (4).

It should be noted that carcinogenic effects have not been reported in either humans or laboratory animals exposed orally or dermally to cadmium (4).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Laboratory experiments suggest that cadmium may have adverse effects on reproduction in fish at levels present in lightly to moderately polluted water (1).

Freshwater fish and invertebrates exhibit an acute LC_{50} ranging from 100 to 1000 µg/L. Saltwater species were 10-fold more tolerant to cadmium. The bioconcentration factors are generally less than 1000, but as high as 10,000 for some freshwater fish (1).

Ambient Water Quality Criteria have been established for cadmium and are as follows (3):

Freshwater:	
Acute toxicity:	3.9 µg/L (1-hr)*
Chronic toxicity:	1.1 µg/L (4-day)*
Marine:	
Acute toxicity:	$4.3 \times 10^{+1}$ µg/L (1-hr)
Chronic toxicity:	9.3 µg/L (4-day)

*Hardness dependent criteria

Terrestrial and Avian

No data characterizing adverse effects on domestic or wild animals are available (1).

SUMMARY OF REGULATORY LEVELS AND CRITERIA

EPA Carcinogenic Classification (3):	B1-Probable human carcinogen via inhalation
Cancer Potency Factor (inhalation) (3):	6.3×10^{0} $(mg/kg/day)^{-1}$
Oral RfD (3):	5×10^{-4} mg/kg/day (water)
	1×10^{-3} mg/kg/day (food)
Ingestion of water and organisms (3):	1.0×10^{1} µg/L
Reportable quantity (3):	10 lbs
OSHA PEL-TWA (7):	5 $\mu g/m^3$
MCL:	5.0 µg/L
MCLG (6):	5 µg/L
WHO (drinking water) (4):	0.005 mg/L
ACGIH TLV-TWA (8):	0.01 mg/m^3 (total dust/particulate)
	0.002 mg/m^3 (respirable fraction)

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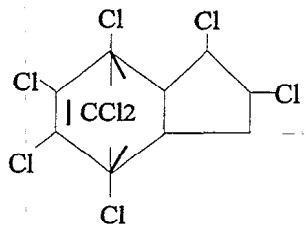
Date of Last Revision: 11/10/94
Revisor: Rich Hoff

CHLORDANE

INTRODUCTION

Chemical Name:	1,2,4,5,6,7,8,8-Octachloro-2,3,3a,4,7,7a-hexahydro-4,7-methamoindene	
Synonyms:	cis-Chlordane:	alpha-Chlordane
	trans-Chlordane:	gamma-Chlordane
CAS Number:	Chlordane (mixture):	57-74-9
	cis-Chlordane:	5103-74-2
	trans-Chlordane:	5103-71-9
Molecular Formula:	$C_{10}H_6Cl_8$	
Molecular Weight:	409.8	
Chemical Structure:		



Chlordane is an organochlorine pesticide that was formerly used on field crops and is presently used to control structural pests in homes. Technical chlordane is a complex mixture that includes two isomers of chlordane, heptachlor, and two isomers of nonachlor. It is very persistent in the environment and is strongly bioaccumulated in fish and other aquatic organisms. Chlordane causes liver tumors in mice, and the results of a mutagenicity assay were positive. It also has adverse reproductive effects in mice, and chronic exposure causes liver changes and adversely affects the central nervous system. Chlordane is very toxic to aquatic organisms (1).

Technical chlordane is a complex mixture, the major components of which are cis-Chlordane and trans-Chlordane. The technical product also contains a variety of other chlorinated hydrocarbons, including heptachlor. It is a viscous amber-colored liquid. Much of the available literature does not distinguish between the chlordane isomers and appears to discuss mixtures of these compounds (1).

FATE AND TRANSPORT

Boiling Point:	175°C at 2 mm Hg	
Melting Point:	cis-Chlordane	107-109°C
	trans-Chlordane	103-105°C
Specific Gravity:	1.59 - 1.635 at 16°C (technical chlordane)	
Solubility:		
In Water:	From 0.056 to 1.85 mg/liter at 25°C	
In Organics:	Miscible in aliphatic and aromatic solvents (technical chlordane)	
Log Octanol/Water Partition Coefficient:	2.78	

Vapor Pressure:	1×10^{-5} mm Hg at 20°C (refined product)
Flash Point:	Minimum 81°C (technical chlordane)

Chlordane is very persistent in the environment, resisting chemical and biological degradation into harmless substances. Chlordane in water is somewhat volatile, which is an important loss process. Loss of chlordane from aquatic systems occurs when organics are present, and when residue concentrations in sediment are higher than in water. Therefore, sorption to sediments is an in removing the chemical from the aquatic environment. Chlordane binds tightly to soil particles and persists for years in soil after surface application. However, chlordane applied as an emulsifiable concentrate is more readily volatilized than when it is applied as a granular formulation. Certain food and feed crops accumulate residues by absorption from the soil. Atmospheric transport of vapors and contaminated dust particles from soil application sites can occur (1).

HUMAN HEALTH EFFECTS

Mixtures of cis-Chlordane and trans-Chlordane produce liver cancer in mice. Chlordane also has mutagenic effects in at least one test system. Reproductive effects, including developmental defects and neonatal metabolic and biochemical disorders, are observed in the offspring of mice exposed to chlordane. Tests with laboratory animals, primarily rodents, demonstrate acute and chronic toxic effects. Either isomer alone, or a mixture of the two, appears to exhibit approximately equal toxicity. Acute effects include anorexia, weight loss, tremors, convulsions, and death. Chronic exposure to chlordane causes liver changes and induces or suppresses a variety of enzyme systems. In addition, chlordane may act as a cumulative neurotoxin. The oral LD_{50} in the rate is 283 mg/kg. Oxychlordane, an epoxide metabolite formed from either chlordane isomer, is significantly more acutely toxic than chlordane. The oral LD_{50} of oxychlordane administered to rats in corn oil is 19 mg/kg and 43 mg/kg when administered in an aqueous suspension (1).

Acute oral or skin exposure to chlordane can cause vomiting, seizures, electroencephalographic dysrhythmia, convulsions, and death in humans. However, most reports of human toxicity are inconclusive. Oxychlordane has been found in a high percentage of human adipose tissue samples and also in human milk samples (1).

ENVIRONMENTAL HEALTH EFFECTS

The toxic effects of chlordane are seen at relatively low concentrations in some fish and invertebrate species. Chlordane also shows strong tendencies for bioaccumulation in some aquatic and terrestrial organisms. It can concentrate at levels thousands of times greater than the surrounding water medium in a variety of aquatic organisms, including bacteria, algae, daphnids, and fish. The EPA criteria for acute exposure to freshwater species is 2.4 μg/L, and it is 4.3E-3 μg/L for chronic exposure. The corresponding acute and chronic values for saltwater species are 0.09 μg/L and 0.0040 μg/L. Very little information exists concerning the biotransformation of chlordane. Although biotransformations may be important for the ultimate degradation of chlordane, these processes are likely to be very slow (1).

Chlordane or oxychlordane residues have been found in a wide variety of wildlife and domestic animal species, but usually at relatively low levels. Chlordane does not appear to be extensively concentrated in the higher members of the terrestrial food chain. Studies indicate that chlordane may produce toxic effects in certain soil invertebrates after surface application. Although little

information concerning bioaccumulation in these organisms is available, the potential bioconcentration of chlordane or oxychlordane by terrestrial insectivores is of concern. Little information on the toxic effects of chlordane to mammalian wildlife and domestic animal species is available. Chlordane or oxychlordane residues have been found in crops, meat, fish, and poultry, dairy products, and eggs. Oral LD_{50} values for chlordane ranging from 331 to 858 ppm in the diet (approximately 25 to 50 mg/kg) are reported for a variety of wild bird species. Oral LD_{50} values ranging from 100 to 1,000 mg/kg are reported for a variety of animals, including rodents, goats, sheep, and chickens (1).

REGULATIONS AND STANDARDS

Ambient Water Quality Criteria (2):

- Aquatic Life
  - Freshwater
    - -- Acute Toxicity: 2.4 μg/L
    - -- Chronic Toxicity: 0.0043 μg/L
  - Saltwater
    - -- Acute Toxicity: 0.09 μg/L
    - -- Chronic Toxicity: 0.004 μg/L

- Human Health
  - Water and Organisms: 4.6E-4
  - Organisms only: 4.8E-4

Summary of Toxicological Indices (3):

- Weight of Evidence: B2 - probable human carcinogen
- Oral Cancer Slope Factor: 1.3 $(mg/kg/day)^{-1}$
- Inhalation Cancer Slope Factor: 1.29 $(mg/kg/day)^{-1}$
- Oral RfD: $6x10^{-5}$

Other Regulations

- OSHA PEL-TWA: 0.5 mg/m^3
- ACGIH TLV-TWA (skin): 0.5 mg/m^3
- Department of Transportation: Combustible Liquid
- SDWA MCL: 2 $\mu g/L$
  SDWA MCLG: 0 $\mu g/L$

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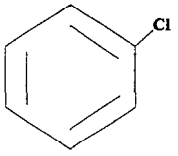
Date of Last Revision: 12/14/94
Revisor: Rich Hoff

CHLOROBENZENE

INTRODUCTION

Chemical Name:	Chlorobenzene
Synonyms (1):	Monochlorobenzene; Chlorobenzol; Phenyl Chloride; MCB; Benzene chloride
CAS Number (1):	108-90-7
Molecular Formula (2):	C_6H_5Cl
Molecular Weight (2):	112.56 g/mole
Chemical Structure (2):	



Chlorobenzene is a colorless liquid with an almond-like odor. It is produced commercially by the chlorination of benzene in the presence of a catalyst (2). It is primarily used as a solvent and degreasing agent. In the past, chlorobenzene has been used as a feedstock, and in the production of phenol, nitrobenzenes, DDT and aniline (1). The production of chlorobenzene has declined over the years due to the replacement of chlorobenzene by cumene in phenol production and the cessation of DDT production (2).

FATE AND TRANSPORT

Log K_{oc} (2):	2.52
Log K_{ow} (2):	2.84
$t_{1/2}$ (3): Air:	3.5 days
Surface Water:	0.30 days
Henry's Law Constant (4):	3.45×10^{-3} atm-m^3/mole
BCF:	10.3 (estimated for edible aquatic organisms) (1)
	33 (estimated) (1)
	447 (fathead minnows) (4)
Solubility (2):	
In Water:	500 mg/L @ 20°C
In Organics:	Soluble in alcohol, ether, and benzene
Vapor Pressure:	8.8 mm Hg @ 20°C (2)
	11.9 mm Hg @ 25°C (1)
Vapor Density (5):	3.88
Specific Gravity (5):	1.11 @ 20°C (liquid)

Chlorobenzene is released into the atmosphere through fugitive emissions associated with its primary uses. In the atmosphere it exists almost entirely in the vapor phase. The dominant removal mechanism is by reaction with hydroxyl radicals. During this process, formation of chlorophenols

takes place. Photolysis will occur to some extent; however, it will be at much slower rate. Monochlorobiphenyls are produced during the photolysis process (4).

If released to a water system, chlorobenzene will evaporate with the rate of evaporation depending on the wind speed and water movement. Biodegradation is possible during the warmer seasons. The rate of the biodegradation process depends on the type of water system. Also, chlorobenzene biodegrades more rapidly in fresh water than in estuarine and marine systems. Adsorption will occur onto organic sediments (4).

Upon release to soil, chlorobenzene will volatilize. However, if it penetrates the upper soil layers, it is relatively mobile in sandy soil and aquifer material. Once chlorobenzene interacts with the soil, biodegradation occurs very slowly or not at all. As a result, it is expected that chlorobenzene will leach into the ground water (4).

PHARMACOKINETICS

The absorption of chlorobenzene via inhalation exposure has been studied by observing two workers who were exposed to 0.84 and 0.50 ppm of chlorobenzene. These results indicate that the chlorobenzene was absorbed in percentages of 38 and 45, respectively. Rats absorbed concentrations of chlorobenzene through inhalation exposure at concentrations up to 700 ppm. Studies also indicate that chlorobenzene is moderately well absorbed from the gastrointestinal tract following oral exposure. For example, studies revealed that a human volunteer absorbed 31%, a rat absorbed 18%, and rabbits absorbed 22% of an administered dose of chlorobenzene. No information was available regarding the dermal uptake of chlorobenzene in humans or animals (2).

Only one study addressed the distribution of chlorobenzene. This study involved administering rats C^{14}-labeled chlorobenzene vapor. The chlorobenzene was found in all tissues with radioactivity increasing as the concentration of chlorobenzene increased. The concentration was highest in the epididymal and perirenal fat. Animal and human studies focusing on the distribution of chlorobenzene following oral or dermal exposure were not available. Also, human studies concerning the distribution of chlorobenzene following inhalation exposure were not available (2).

The primary metabolites of chlorobenzene are p-chlorophenylmercapturic acid and 4-chlorocatechol. The in vitro metabolites are o-chlorophenol, m-chlorophenol, and p-chlorophenol. In a 57 year old male exposed to an oral dose of 0.3 mmol/kg chlorobenzene, the main urinary metabolite reported was 4-chlorocatechol. In animals studied, there was more urinary mercapturic acid than 4-chlorocatechol and o- and m-chlorophenylmercapturic acids (2).

Studies addressing the excretion of chlorobenzene following inhalation exposure in rats demonstrated that respiratory elimination of C^{14}-labeled chlorobenzene increased disproportionately with increasing dose. Modeling of respiratory elimination kinetics revealed a biphasic elimination pattern. A fast alpha phase of up to 4 hours post-exposure and a slower beta phase lasting longer than 48 hours was observed. The study that reported the 0.84 and 0.50 ppm chlorobenzene exposure in workers revealed that the excretion of p-chlorophenylmercapturic acid was markedly lower than that of 4-chlorocatechol. Studies which focused on the excretion of chlorobenzene following oral exposure to a 57 year old male found that the excretion of p-chlorophenylmercapturic acid was markedly lower than that of 4-chlorocatechol. A rabbit study which involved the oral exposure of a C^{14}-labeled chlorobenzene resulted in excretion of 22% of the chlorobenzene in the urine while

the remaining portion was excreted in the expired air. Human and animal studies regarding the dermal exposure of chlorobenzene were not available (2).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Death due to exposure to chlorobenzene in humans has not been reported. However, animals experienced death at high doses of chlorobenzene for brief periods of exposure. Death occurred within 2 hours after 8,000 ppm exposure and 7 hours after 3,700 ppm exposure. Death was due to central nervous system depression resulting in respiratory failure (2).

No evidence of hepatic toxicity in humans by inhalation, oral, or dermal exposure was reported. However, animal studies revealed changes in liver weights and enzyme levels, degeneration, necrosis, and alterations in microsomal enzymes. The precise mechanism for liver damage is unknown. Although, it is suggested that direct binding of chlorobenzene metabolites to cellular protein may be involved. Based on these animal studies, liver toxicity may be an area of concern in humans (2).

Renal effects in humans caused by chlorobenzene exposure have not been reported. Animal studies suggested kidney effects at concentrations similar to those which affect the liver. Signs of toxicity include tubular degeneration and necrosis as well as changes in organ weight at doses of $\geq$ 250 mg/kg/day. Necrosis is associated with covalent binding of substantial amounts of radiolabeled chlorobenzene to kidney protein (2).

Immunological effects have not been determined in humans. Histopathologic evaluations in animals suggest immunotoxicity. However, direct tests have not been performed (2).

Chlorobenzene-induced neurological effects have been reported in humans. These effects include disturbances in the central nervous system, but changes in structure of the brain or other parts of the nervous system were not evident. Workplace studies involving the inhalation of chlorobenzene for up to 2 years revealed headaches, dizziness and sleepiness in humans exposed to concentrations of chlorobenzene of 2,400 to 2,900 ppm. When accidentally ingested, unconsciousness, lack of response to skin stimuli, and muscle spasms were exhibited. Animal studies have identified chlorobenzene as a potential neurotoxin (2).

There are no human studies which address the developmental effects of chlorobenzene. However, negative response in two animal species indicate that chlorobenzene does not cause developmental toxicity (2).

No studies were located that addressed the reproductive effects of chlorobenzene in humans. Animal studies are inconclusive since only one species was tested. In this study, chlorobenzene did not adversely affect various reproductive parameters in rats (2).

The EPA has established an oral RfD value of 2.0×10^{-02} mg/kg/day with an uncertainty factor of 1000. The Environmental Criteria and Assessment Office (ECAO) established an inhalation RfD of 5.71×10^{-3} mg/kg/day. The NOAEL for the study was 19 mg/kg/day; and the LOAEL was 54.5 mg/kg/day. This study involved the oral administration of chlorobenzene to beagle dogs via

capsules at doses of 27.25, 54.5, or 272.5 mg/kg/day for 5 days/week, for 13 weeks. Death, body weight loss, changes in hematology, clinical chemistry, and urine analysis, and pathologic changes in the liver, kidney, gastrointestinal mucosa, and hematopoietic tissue were observed at 272.5 mg/kg/day (6).

Carcinogenic Effects

The EPA assigned chlorobenzene a carcinogenic classification of Group D-not classifiable as to human carcinogenicity. This classification indicates that no human data, inadequate animal data and predominantly negative genetic toxicity data in bacterial, yeast, and mouse lymphoma cells were found (6).

The animal study, which proved to be inadequate involved the administration of chlorobenzene by gavage in corn oil 5 days per week for 103 weeks to F344/N rats and B6C3F1 mice. No chlorobenzene-related signs of clinical toxicity were observed in the rats or mice during the study. However, the frequency of high-dose male rats with neoplastic nodules of the liver was slightly increased (6).

As mentioned previously, the genotoxic effects of chlorobenzene in humans is unknown. However, genotoxic effects have been examined in short-term animal bioassays. In vivo animal studies were moderately positive for genotoxicity. In vitro studies involving bacterial and yeast assay systems with and without metabolic activation were negative. The genotoxicity involved the transformation of epithelial cells but not the hepatocytes (2). These studies indicate inconclusive results for the determination of genotoxicity in animals and man.

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Although data are inadequate for establishing criteria, the EPA has reported the lowest values of chlorobenzene known to be toxic in aquatic organisms. These values are as follows (1,7):

Freshwater:

Acute toxicity:	250 μg/L
Chronic toxicity:	50 μg/L (for 7.5 days exposure)

Marine:

Acute toxicity:	160 μg/L
Chronic toxicity:	129 μg/L

Terrestrial and Avian

Information regarding the toxicity of chlorobenzene to terrestrial and avian wildlife or domestic animals was not found in the available literature.

REGULATORY LEVELS AND CRITERIA

OSHA PEL TWA (2):		75 ppm
ACGIH TLV TWA (2):		10 ppm
Reportable Quantity (2):		100 lbs
Health Advisories (2):		
1 day:		2 mg/L
10 days:		2 mg/L
Longer term:	Child:	2 mg/L
	Adult:	7 mg/L
Lifetime:		100 mg/L
Ambient Water Quality Criteria (2):		
Ingesting water and organisms:		4.88×10^{-1} mg/L

SUMMARY OF TOXICOLOGICAL INDICES

EPA Carcinogenic Classification (6):	Group D - Not classifiable as to human carcinogenicity
Oral RfD (6):	2.0×10^{-2} mg/kg/day
Inhalation RfD (8):	5.71×10^{-3} mg/kg/day

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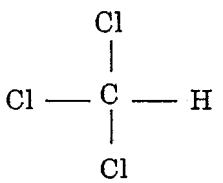
Revisor: Patrick B. Moroney

CHLOROFORM

INTRODUCTION

Chemical Name: Chloroform
CAS Number: 67-66-3
Molecular Formula: $CHCl_3$
Molecular Weight: 119.38 g/mole
Chemical Structure:



Chloroform is a colorless, highly refractive, heavy, volatile liquid. It has a characteristic odor and a sweet taste. Chloroform is used in the manufacture of fluorocarbon plastics, as a solvent, fumigant, and insecticide, and in analytical chemistry (7).

FATE AND TRANSPORT

Log K_{oc} (4) : 1.64
Log K_{ow} (4): 1.90-1.97
$t_{1/2}$ (5): 1.2-31 days reported for aquatic systems
Henry's Law Constant (4): 0.0032 atm-m^3/mole @ 25° C
BCF (1): Not strongly bioaccumulated
Degradation Products (4): Methylene chloride
Solubility:
In Water (1): 8,200 mg/L @ 20° C
In Organics (4): Soluble in acetone, miscible with ethanol, ether, benzene and ligroin
Vapor Pressure (4): 160 mm Hg @ 20° C
Specific Gravity (4): 1.4832 @ 20/4° C

The majority of chloroform is released into the atmosphere. Release to water and soil will be lost to the environment through volatilization. Chloroform has the capability to be transported long distance through the atmosphere. In the gas-phase, it reacts with photochemically produced hydroxyl radicals.

Chloroform does not significantly adsorb to sediment. Also, it is not expected to bioconcentrate in aquatic organisms(s).

PHARMACOKINETICS

Chloroform is readily absorbed through the lungs and intestinal tract, and to a lesser extent through the skin. Absorption from the gastrointestinal tract is rapid and complete and follows first-order absorption kinetics. With oral exposure, a dose-dependent, first-pass phenomenon occurs with pulmonary elimination of unchanged chloroform. Pulmonary uptake occurs by diffusion which also follows first-order kinetics (3).

Chloroform is lipid soluble and passes through cell membranes readily. It appears to accumulate in body fat and the liver of humans when inhaled. Distribution of chloroform after oral exposure to animals was found to be in body fat and liver. No data on dermal contact were available (6).

The main organ for chloroform metabolism is the liver. Chloroform is metabolized by the cytochrome P-450 system into trichloromethanol, which spontaneously dechlorinates into phosgene. The ultimate end product of chloroform metabolism is carbon dioxide. It is believed that phosgene and other reactive intermediates, possibly free radicals, are responsible for the toxic effects of chloroform (5). Reactive intermediates of chloroform have been shown to covalently bind with liver proteins and to deplete glutathione levels (3).

There are two primary pathways for the elimination of chloroform from the body. One pathway is the elimination of unchanged chloroform in exhaled air. The other pathway involves metabolic conversion of chloroform in the liver and other organs with the subsequent elimination of end products by respiration (5).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

The primary effect of high-level exposure to chloroform is on the central nervous system. Exposure to very high levels can cause liver and kidney damage as well as cardiac arrhythmia (3). Repeated exposure to lower, subnarcotic doses of chloroform can cause liver and kidney toxicity; however, these effects are typically not seen (3).

The EPA has established an oral RfD for chloroform of 1×10^{-2} mg/kg/day. This is based on a long-term feeding study of chloroform in beagle dogs. A lowest observable adverse effect level (LOAEL) of 12.9 mg/kg/day was determined. Uncertainty factors totaling 1000 were applied in deriving the RfD. The EPA considers the confidence in this RfD to be "medium to low" (2). An RfD for inhalation exposure is not available at this time (2).

Carcinogenic Effects

While the database for conclusively establishing chloroform as a human carcinogen is deemed to be inadequate, there is a body of evidence to support the carcinogenic potential of chloroform in laboratory animals (2).

Chloroform has been tested for carcinogenicity in eight strains of mice, two strains of rats, and in beagle dogs. In gavage studies with mice and rats, significant increases in kidney epithelial tumors and hepatocellular carcinomas were observed (2).

The EPA has established cancer slope factors for chloroform from both the oral and inhalation exposure routes. The oral slope factor, based on tumor incidence in the kidney, is 6.1×10^{-3} $(mg/kg/day)^{-1}$. The inhalation slope factor, based on development of liver tumors, is 8.1×10^{-2}

(mg/kg/day)$^{-1}$. Based on the carcinogenic potential demonstrated in animals, chloroform has been classified as a Group B2 carcinogen-probable human carcinogen (2,6).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Limited information is available regarding the toxicity of chloroform to aquatic organisms at known concentrations. LC_{50}s for two freshwater fish and one invertebrate species range from 28.9 to 115 mg/L. Twenty-seven-day LC_{50} values of 2.03 and 1.24 mg/L were reported for embryo-larval tests with rainbow trout (1).

The only reliable value for toxicity of chloroform to marine species is a 96-hour LC_{50} value of 81.5 mg/L for pink shrimp (1).

The available data for chloroform indicates that toxicity to freshwater aquatic life occurs at the following concentrations. Data regarding saltwater species was not available. (2)

Acute toxicity: 2.89×10^4 µg/L
Chronic toxicity: 1.24×10^3 µg/L

Terrestrial and Avian

No specific information regarding effects of chloroform to terrestrial and avian species was readily available. However, chloroform is thought to be widely distributed throughout the environment and has been detected in water fowl (1).

REGULATORY LEVELS AND CRITERIA

OSHA TWA (5):	50 ppm (ceiling limit)
ACGIH TWA (5):	10 ppm
Water and Fish Consumption (2):	1.9×10^{-1} µg/L
Fish Consumption (2):	1.57×10^{-1} µg/L
Reportable Quantity (2):	10 lbs

SUMMARY OF CRITERIA

EPA Carcinogenic Classification (2):	Group B2-probable human carcinogen
Oral RfD (2):	1×10^{-2} mg/kg/day
Inhalation RfD (2):	Not established
Cancer Slope Factor (oral) (2,6):	6.1×10^{-3} (mg/kg/day)$^{-1}$
Cancer Slope Factor (inhalation) (2,6):	8.1×10^{-2} (mg/kg/day)$^{-1}$
MCL (2):	0.10 mg/L (total trihalomethanes)
MCLG (2):	0 mg/L

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Date of Last Revision: 12/15/94
Revisor: Patrick B. Moroney

CHROMIUM

INTRODUCTION

Chemical Name:	Chromium
CAS Number:	Chromium (VI) 7440-47-3; Chromium (III) 16065-83-1
Molecular Formula:	Cr
Molecular Weight:	52 g/mole
Chemical Structure:	Cr

Chromium is a semi-gray heavy metal that generally exists in either a trivalent (III) or hexavalent (VI) state. Chromium occurs naturally in soil; however, it is no longer mined in the United States.

FATE AND TRANSPORT

BCF:	16 (2)
Degradation Products:	None
Solubility:	Metallic chromium is not soluble in water, but it is soluble in some acids and strong alkalies (4). Some chromium salts are water soluble (3).
Specific Gravity (3):	7.20 @ 28°C

Chromium and compounds can be present in air, water, and soil. In air, chromium is primarily associated with particulate matter which results from surface soil dispersion or particulate emission from industrial sources (3). Transport of chromium from water to air is not likely because chromium compounds do not volatilize from water. In the atmosphere, Cr(VI) can be reduced to Cr(III) at a significant rate by V^{+2}, V^{+3}, VO_2^{+}, Fe^{+2}, HSO^{-}_3, and As^{+3}. The residence time of atmospheric chromium is expected to be less than 10 days (1).

Cr(VI) as a component of a complex anion is quite soluble in water. Consequently, it is not adsorbed to any significant degree by clays or hydrous metal oxides. The anionic form of Cr(VI), therefore, is mobile in the aquatic environment (3). Cr(VI) is a moderately strong oxidizing agent. In water, it will react with a reducing agent to form Cr(III) which will subsequently hydrolyze to chromium hydroxide and precipitate out of solution. This Cr(III) precipitate tends to adsorb to sediment. Air deposition, runoff, and leaching from soil can introduce chromium into surface and groundwater. The residence time of Cr(III) in lake water is approximately 4.5 to 18 years (1).

Chromium probably occurs as insoluble $Cr_2O_3 \cdot nH_2O$ in soil, since the organic matter in soil is expected to convert soluble chromate into insoluble Cr_2O_3 (1). Cr(III), as an insoluble salt, tends to strongly adhere to clay particles and organic matter, whereas soluble Cr(VI) is not strongly adsorbed to soil (3).

PHARMACOKINETICS

The primary route of entry of chromium into the body is through the gastrointestinal tract. Inhalation and dermal absorption are the primary routes of entry into the body for occupational exposure (7).

Chromium is distributed in humans following inhalation to the lungs, lymph nodes, kidney, liver, bladder, and bone. Distribution of chromium after oral exposure resulted in accumulation in the lymph nodes and lungs followed by the spleen, liver, kidney, and heart. There was no information available among the reviewed literature concerning the distribution of chromium after dermal exposure (7).

In vitro studies indicate that chromium (VI) is reduced to chromium (III). During this process an intermediate, chromium V, appears to be formed which may represent the form of chromium that interacts with cellular macromolecules (7).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Human studies have identified chromium as a nephrotoxin that produces renal tubular necrosis. There is not sufficient evidence, however to quantitatively describe the effects. Limited data suggest that exposure by inhalation to chromium compounds can result in hepatic effects in humans and animals. Sufficient evidence is not available to relate chromium exposure to adverse reproductive or developmental effects. It should be noted that Cr(III) by the oral route is an essential trace element (1).

Chromium (III)

The verified chronic oral RfD for metallic Cr(III) (insoluble salts) is 1 mg/kg/day. The RfD is based on a no observed adverse effects level (NOAEL) derived from a study in which 60 male and female rats were fed chromic oxide baked in bread at a dietary level of 5 percent. They were fed bread 5 days/week for 600 feedings (840 total days). It was determined that the average amount of chromic oxide ingested was 1,800 g/kg body weight. An uncertainty factor of 100 and a modifying factor of 10 were applied to the NOAEL when calculating the RfD. The modifying factor was derived to address factors that if determined would lower the RfD (1,5). The confidence in the RfD is low; however, it is a conservative number. No effects due to chromic oxide were observed at any dose level (1). An Inhalation RfD for Chromium III is currently under review by the EPA (5).

Chromium (VI)

The EPA has established an oral RfD for Chromium VI of 5.0×10^{-3} mg/kg/day. No LOAEL was identified in the supporting study. However, a NOAEL of 2.4 mg/kg/day was reported. The supporting study involved the administration of drinking water containing 1 to 11 ppm chromium VI to Sprague-Dawley rats for one year. No significant adverse effects were seen on appearance, weight gain, or food consumption, and there were no pathologic changes in the blood or other tissues (5).

An inhalation RfD for Chromium VI is currently under review by the EPA (5).

Carcinogenic Effects

Chromium (III)

Cr(III) has been classified by the EPA as a Group D carcinogen (1). This means the chemical has not been classified because there is inadequate evidence of carcinogenicity in animals (5).

Chromium (VI)

The verified cancer unit risk for lifetime exposure by inhalation to Cr(VI) is 1.2×10^{-2} $(\mu g/m^3)^{-1}$. Inhaled Cr(VI) has been classified by EPA as a Group A-human carcinogen (1). There is sufficient evidence to support an association between exposure by inhalation and cancer (2). The potency factor is based on an epidemiology study in which chromate plant workers were studied and showed an increased incidence of lung cancer. The study documented total chromium exposure and did not differentiate between Cr(III) and Cr(VI) and consequently, the health risk may be underestimated (1,5). Although CR(VI) is carcinogenic by the inhalation route, there is no evidence that it is carcinogenic by the oral route (1).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Chromium is an essential nutrient. It is accumulated in a variety of aquatic and marine biota to levels quite higher than in ambient water. Therefore, it appears that the food chain is a more efficient pathway for chromium uptake than direct uptake from seawater (1).

Water and aquatic species characteristics modify the toxic effects of chromium on aquatic life. Cr(III) appears to be more acutely toxic to fish than Cr(VI); however, the reverse is true in chronic exposure studies (1).

The Ambient Water Quality Criteria for the level of chromium that will not affect aquatic organisms and their uses include the following:

- 4-day averages that should not be exceeded more than once every three years (5,6).

	Freshwater	Saltwater
Cr(VI)	11 μg/L	None
Cr(III)	$e^{(0.8190\ [\ln\ [hardness]+1.561])}$ or 210 μg/L (hardness dependent)	--

- 1-hour average that should not be exceeded more than once every 3 years (6).

	Freshwater	Saltwater
Cr(VI)	16 μg/L	1,100 μg/L
Cr(III)	$e^{(0.8190\ [\ln\ [hardness]+3.688])}$ or 1,700 μg/L (hardness dependent)	10,300 μg/L (hardness dependent)

Terrestrial

No plants typically used as food or animal feed are chromium accumulators. Plants that absorb chromium do so through root uptake and do not efficiently transport it to the leaves. Also, there is

little tendency for inorganic Cr(III) to accumulate along the food chain. No data are available for organochromium compounds (3).

REGULATORY LEVELS AND CRITERIA

OSHA PEL-TWA (10):	0.5 mg/m^3 (Chromium III)
	1.0 mg/m^3 (insoluble salts or chromium metal)
OSHA Ceiling Level (10):	1 mg/10 m^3 (chromic acid and chromates)
MCL (6):	0.1 mg/L
ACGIH TLV-TWA (9):	0.5 mg/m^3 (chromium metal)
	0.5 mg/m^3 (Chromium II and III compounds)
	0.05 mg/m^3 (water soluble Chromium VI)

Health Advisories (1):

One-day (Chromium VI):	Use 10-day valve
10-day (Child) (Chromium VI):	1.4 mg/L
Longer-Term (Child) (Chromium VI):	0.24 mg/L
Longer-Term (Adult) (Chromium VI):	0.84 mg/L
Lifetime (Adult) (Chromium VI):	0.120 mg/L

Ingestion of water and fish (Chromium III) (1):	170 mg/L
Ingestion of organisms only (Chromium III) (1):	3,433 mg/L

SUMMARY OF CRITERIA

Cr(III) -	EPA Carcinogenic Classification (1):	Group D-Not Classified
Cr(VI) -	EPA Carcinogenic Classification (1):	Group A-Human Carcinogen (Inhalation)

Cancer Slope Factor (1,5)

Cr(III):	Not Applicable
Cr(VI):	1.2×10^{-2} $(\mu g/m^3)^{-1}$ (Inhalation unit risk)
	42 $(mg/kg/day)^{-1}$ (Inhalation slope factor)

RfD (Oral) (1,5)

Cr(III):	1 mg/kg/day
Cr(VI):	5×10^{-3} mg/kg/day

Oral Drinking Water (7)

Cr (VI):	2×10^{-2} mg/kg/day

NOAEL (7)

Cr (III):	5%
Cr (VI):	2.4 mg/kg/day

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Date of Last Revision: 12/14/94
Revisor: Rich Hoff

COPPER

INTRODUCTION

Chemical Name:	Copper
Synonyms (1):	Bronze Powder, C.I. 77400
CAS Number (1):	7440-50-8
Molecular Formula:	Cu
Molecular Weight (1):	63.546 g/mole
Chemical Structure:	Cu

Copper is a reddish colored metal that occurs naturally in rock, soil, water, sediment, and air. Also, copper occurs naturally in plants and animals. A concentration of approximately 50 ppm can be found in the earth's crust. Copper is considered to be ductile and an excellent conductor. The primary uses of copper consist of use as the metal or alloy in the manufacture of wire, sheet metal, pipe, and other metal products. Copper compounds occur both naturally and are man-made and exhibit a characteristic blue-green color. Copper compounds are used in agriculture to treat plant diseases, for water treatment, and as preservatives for wood, leather, and fabrics (1,2,3).

FATE AND TRANSPORT

Fish BCF (4):	200 L/kg
Solubility(3):	In Water : Insoluble
Density (3):	8.92

The largest release of copper to the environment is to the land with an estimated 97% deposited to this media. Tailings and overburdens from copper mines and tailings from mills are the primary contributors. It is also released through mining operations, agriculture, solid waste, and sludge from publicly-owned treatment works. The majority of copper deposited in the soil is strongly adsorbed and remains in the upper few centimeters of soil. The copper adsorbs to organic matter, carbonate minerals, clay minerals, or hydrous iron and manganese oxides (3).

Copper is released to water bodies primarily through natural weathering of soil and discharges from industries and sewage treatment plants. The majority of copper in water is found in the form of particulates. Approximately 2.4% of total copper released to the environment is attributed to release into a waterway. An estimated 68% of release to water is associated with natural weathering or disturbed soil. Copper sulfate use represents 13% of releases to water. Only 2% is contributed by urban runoff. The copper discharged into waterways is in the form of particulate matter. The particulate matter either settles out, precipitates out, or adsorbs in organic matter, hydrous iron and manganese oxides, and clay in sediment. When copper is present as the Cu(I) ion, it tends to disproportionate to Cu(II) and copper metal. The concentration of dissolved copper depends on factors such as pH, the oxidation-reduction potential of the water, and the presence of competing cations, anions of insoluble cupric salts and organic and inorganic complexing agents (3).

Copper emitted to the air occurs from windblown dust, volcanos, and manufacturing processes such as primary copper smelters and ore processing facilities. An estimated 0.04% of all copper released to the environment is to the air. Copper is released to the atmosphere in the form of particulate matter or adsorbed to particulate matter. Gravitational settling accounts for the removal of particles that are >5 μm while smaller particles are removed by either dry deposition or washout by rain. Deposition rates in urban areas are 0.119 and 0.164 kg/ha/yr for dry and wet deposition, respectively. It is assumed that copper transformations in the atmosphere exist as oxides. As the oxides age, sulfatization may occur (3).

PHARMACOKINETICS

No data were located that studied absorption of copper through inhalation or dermal exposure. However, via oral exposure, copper is absorbed in the stomach and small intestine. In humans, approximately 60% of an oral dose of Cu^{64} as copper acetate was absorbed from the gastrointestinal tract. Once absorbed from the gastrointestinal tract, copper forms ionic copper or binds to amino acids. The factors which may affect copper absorption include: competition with other metals, the amount of copper in the stomach, certain dietary components, and forms of copper. High dietary ascorbic acid has been shown to impair copper absorption (3).

Studies regarding distribution of copper through inhalation and dermal exposure are not available. Through oral exposure, copper is distributed by loosely binding to plasma albumen and amino acids in the portal blood and transported to the liver. Studies have also shown that copper binds to transcuprein, a plasma protein. Once in the liver, copper is incorporated into ceruloplasmin and released into the plasma (3).

Copper is metabolized mainly by its transfer to and from various organic ligands. Several binding proteins for copper have been identified that are important in the uptake, storage, and release of copper from tissues (3).

Oral exposure is the only pathway which has been studied with regard to excretion. The major pathway for the excretion of copper is through bile following oral exposure. Studies have shown that 72% of ingested radioactive copper was excreted as copper acetate in the feces. Reabsorption of biliary copper is negligible; however, a fraction of fecal copper is of endogenous biliary origin (3).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Wilson's disease (hepatolenticular degeneration), is the only significant manifestation of copper toxicity in humans. Death has been reported upon ingestion of large amounts of copper and attributed to extensive hepatic centrilobular necrosis (3).

Systemic effects induced by copper ingestion include gastrointestinal irritation, manifested as vomiting, nausea, diarrhea, and anorexia. In individuals totally poisoned from copper, centrilobular necrosis of the liver and vecrosis and sloughing of tubular cells in the kidney have been observed (3).

Immunological effects in humans have been exhibited as allergic dermatitis upon contact with copper. Additionally, inhalation studies in mice have found that after exposure to copper, an impaired immune function is present (3).

Neurological effects are manifested as Wilson's disease. Wilson's disease affects the central nervous system and includes symptoms of poor coordination, psychological impairment, tremor, disturbed gait, and rigidity. Increased copper levels in the brain have been observed in humans with Wilson's disease and in animals exposed to high levels of copper (3).

Mice, mink, and hamsters injected with copper or fed a diet high in copper have shown developmental effects. These effects include fetal mortality and developmental abnormalities. No developmental effects have been reported in humans; however, there is a possibility that there may be an increased incidence of spontaneous abortion and miscarriage in women exposed to high levels of copper. Developmental effects have not been observed in healthy humans or in the offspring of mothers with Wilson's disease (3).

In humans, reproductive effects have not been reported when exposed to high levels of copper. Mink did not exhibit adverse effects when fed a diet high in copper. However, when a copper wire was inserted into the vas deferens or uterus of monkeys, rats, hamsters, and rabbits prior to conception or at gestational day 3, decreased fertility was observed (3).

The Drinking Water Criteria Document concluded toxicity data were inadequate for calculation of an RfD for copper.

Carcinogenic Effects

The EPA carcinogen classification for copper is Group D-inadequate evidence of carcinogenicity. An elevated incidence of cancer has not been observed in humans or animals exposed to copper via inhalation, oral, or dermal route of exposure (3).

Although there are no data on the mutagenicity of copper in humans, in vivo and in vitro studies of mammalian systems suggest that copper is a potential human mutagen (3).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Available data are not adequate for establishing water quality criteria. However, the bioconcentration factor of copper in fish has been developed. The results of these studies indicate a low potential for bioconcentration with a range of factors for fish of 10-100. The bioconcentration factor for molluscs, such as oysters was considerably higher at a peak of 30,000. Evidence suggests that there is no biomagnification of copper in the food chain. The biomagnification ratio in fish studied from lakes that were known to have received elevated loadings of copper, revealed that the ratio was <1 (3).

Terrestrial and Avian

In bioaccumulation studies of pollutant concentrations in the muscle and livers of 10 mammal species in Donana National Park in Spain, no evidence of bioaccumulation was obtained. No evidence of copper biomagnification in the food chain was observed (3).

REGULATORY LEVELS AND CRITERIA

OSHA PEL-TWA	
Fumes (3):	0.1 mg/m^3
Dusts and Mists (3):	1.0 mg/m^3
EPA SMCL (3):	1.0 mg/L
MCL (3):	1.3 mg/L (Action Level)
MCLG (3):	1.3 mg/L
EPA Reportable Quantity (3):	5000 lbs
ACGIH TLV-TWA:	
Fumes (3):	0.2 mg/m^3
Dusts and Mists (3):	1.0 mg/m^3
Ambient Water Quality Criterion (3):	1.0 mg/L
1-Day Health Advisory (3):	1.3 mg/L

SUMMARY OF CRITERIA

EPA Classification (6):	Group D - Inadequate evidence of carcinogenicity
Noncarcinogenic Effects	
Oral RfD (5):	3.71×10^{-2} mg/kg/day
Inhalation RfC (6):	Not determined

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Date of Last Revision: 11/10/94
Revisor: Rich Hoff

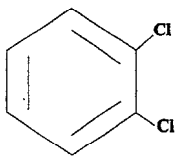
DICHLOROBENZENES

INTRODUCTION

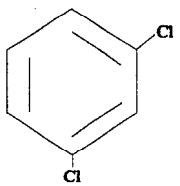
Chemical Name:	Dichlorobenzene	
Synonyms CAS Number (1):	1,2-Dichlorobenzene (1,2-DCB)	95-50-1
	1,3-Dichlorobenzene (1,3-DCB)	541-73-1
	1,4-Dichlorobenzene (1,4-DCB)	106-46-7

Molecular Formula (2): $C_6H_4Cl_2$
Molecular Weight (2): 147.01 g/mole
Chemical Structure (2):

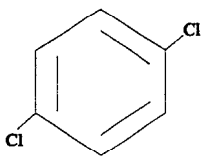
1,2-DCB



1,3-DCB



1,4-DCB



FATE AND TRANSPORT

Specific Gravity:	1,2-DCB:	1.3 mg/liter at 20°C
	1,3-DCB:	1.288 mg/liter at 20°C
	1,4-DCB:	1.458 mg/liter at 20°C
Solubility in Water:	1,2-DCB:	100 mg/liter at 22°C
	1,3-DCB:	69 mg/liter at 22°C
	1,4-DCB:	49 mg/liter at 22°C

Solubility in Organics: Soluble in alcohol, ether, acetone, benzene, carbon tetrachloride, and ligroin

Log Octanol/Water (log K_{ow}) Partition Coefficient:	1,2-DCB:	3.38 mg/liter at 22°C
	1,3-DCB:	3.38 mg/liter at 22°C
	1,4-DCB:	3.39 mg/liter at 22°C
Vapor Pressure:	1,2-DCB:	1 mm Hg at 20°C
	1,3-DCB:	2.3 mm Hg at 20°C
	1,4-DCB:	0.6 mm Hg at 20°C

Relatively little information concerning the environmental fate of DCB is available. DCB is expected to volatilize at a relatively rapid rate, and atmospheric transport can occur. It has an estimated half-life for removal from agitated surface water of 9 hours or less. Dichlorobenzenes are reported to be reactive toward hydroxyl radicals in air with a half-life of about 3 days, but indirect evidence suggests that DCB does not hydrolyze at a significant rate under normal environmental

conditions. The high log octanol/water partition coefficient for DCB suggests that adsorption to organic matter in aquatic systems and soil is probably an important environmental fate process. Indirect evidence suggests that bioaccumulation may also be an important fate process. DCB appears to be resistant to biodegradation. However, it may be broken down to some degree by pollutant-acclimatized microorganisms. Sorption, bioaccumulation, and volatilization with subsequent atmospheric oxidation are likely to be competing processes, with the dominant fate being determined by local environmental conditions. If volatilization doesn't occur, DCB is believed to be persistent.

PHARMACOKINETICS

Only pharmacokinetic data for 1,4-dichlorobenzene is reported in this toxicological profile. Quantitative absorption data is lacking for 1,4-dichlorobenzene in animals or humans. 1,4-Dichlorbenzene is structurally similar to benzene and it is assumed to be 100 percent absorbed upon oral administration. Upon inhalation for a three-hour period, 1,4-dichlorobenzene was 20 percent absorbed. Dermal absorbtion has not been assessed.

Animal studies have shown that upon absorption, 1,4-dichlorobenzene is distributed primarily to adipose tissue, with much lower levels in the liver and kidney. Detectable levels have been exhibited in the blood, lung, heart and brain, as well.

The primary urinary metabolite of 1,4-dichlorobenzene in both animals and humans is 2,5-dichlorophenol. This compound was conjugated with glucuronic and sulfuric acids.

1,4-Dichlorobenzene is mainly excreted in the urine, with some elimination in feces and expired air. There is also evidence that 1,4-dichlorobenzene is reabsorbed in enterohepatic circulation and is excreted in bile.

HEALTH EFFECTS

It is generally thought that the available data are inadequate for assessing the carcinogenic potential of DCB in animals and humans. One case study suggests an association between exposure to dichlorobenzene and several cases of leukemia. DCB is reported to be nonmutagenic in Salmonella typhimurium tester strains. Mutagenic and clastogenic activity reportedly occurs in some plant test systems. No data are available for evaluating the teratogenic or reproductive effects in animals or humans.

Symptoms of acute inhalation intoxication in humans include headache, nausea, and throat irritation. DCB is also a skin and eye irritant.

A variety of other symptoms, including weakness, fatigue, and anemia, have been observed after chronic dermal and inhalation exposure to dichlorobenzene.

Inhalation of DCB causes eye and upper respiratory tract irritation, central nervous system depression, and liver and kidney damage in experimental animals. An LC_{20} of approximately 4,900 mg/m^3/7 hours is reported for the rat. No toxic effects were observed after daily 7-hour inhalation exposures of up to 560 mg/m^3 for as much as 7 months in several species of experimental animals. Hepatic porphyria is reported to occur in rats after daily tracheal incubation of 455 mg/m^3

for up to 15 days. Oral exposure results in stimulation of liver microsomal enzyme systems and cumulative toxicity. The oral LD_{50} for the rat is 500 mg/kg. Chronic oral exposure to 188 mg/kg/day causes liver and kidney damage in rats. Exposure to 0.01-0.1 mg/kg/day produces changes in the hematopoietic system, increased prothrombin time, and altered conditioned reflexes and enzyme activities in chronically exposed rats. In general, toxicity increases in the order 1,4-DCB, 1,3-DCB, 1,2-DCB.

The oral Cancer Slope Factor (CSF) of 2.4×10^{-2} $(mg/kg/day)^{-1}$ is based upon a 103 week gavage study in mice in which liver tumors were exhibited.

TOXICITY TO WILDLIFE AND DOMESTIC ANIMALS

The 48-hour and 96-hour LC_{50} values for Daphnia and blue-gills, respectively, tested under static conditions, were 2,440 and 5,590 µg/liter (1,2-DCB); 28,100 and 5,020 µg/liter (1,3-DCB); and 11,000 and 4,280 µg/liter (1,4-DCB). Two flow through 96-hour LC_{50} tests using fathead minnows and rainbow trout gave values of about 3,000 µg/liter. A freshwater chronic value of 2,000 µg/liter is reported for the fathead minnow. Acute values for three saltwater species ranged from 1,970 µg/liter for the mysid shrimp to 9,660 µg/liter for the sheepshead minnow. No saltwater chronic values are available. A whole body bioconcentration factor of about 80 is reported for the bluegill.

The 96-hour median effect levels for chlorophyll a and cell number are 179,000 and 149,000 µg/liter, respectively, in the freshwater alga Selenastrum capricornutum. In the saltwater alga Skeletonema costatum the corresponding values are 44,200 and 44,100 µg/liter, respectively.

REGULATORY LEVELS AND CRITERIA

The following regulatory levels and criteria have been established for dichlorobenzene(s):

Ambient Water Quality Criteria (USEPA):

Aquatic Life		
Freshwater Acute	1,120	µg/L*
Freshwater Chronic	763	µg/L*
Marine Acute	1,970	µg/L*
Human Health		
Water and Organisms	400	µg/L
Organisms Only	260	µg/L

*Lowest Observed Effects Levels, Not Criteria

Maximum Contaminant Level:

1,2-dichlorobenzene	600	µg/L
1,3-dichlorobenzene	600	µg/L
1,4-dichlorobenzene	75	µg/L

USEPA Carcinogenic Classification:

1,2-dichlorobenzene	D (not classifiable)
Oral RfD	$9x10^{-2}$ mg/kg/day
1,3-dichlorobenzene	D (not classifiable)
Oral RfD	Pending
1,4-dichlorobenzene	B2 (Probable human carcinogen)(12)
Inhalation RfD(11):	$2.29x10^{-1}$ mg/kg/day
Oral Cancer Slope Factor (12):	$2.4x10^{-2}$ mg/kg/day^{-1}

OSHA PEL-TWA:	1,2-dichlorobenzene	50 ppm
	1,4-dichlorobenzene	75 ppm
ACGIH TLV-TWA(1):	1,2-dichlorobenzene	25 ppm
	1,4-dichlorobenzene	10 ppm

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Date of Last Revision: 2/4/92

Revisor: Susan C. Johnston

1,2-DICHLOROETHANE

INTRODUCTION

Chemical Name: 1,2-Dichloroethane

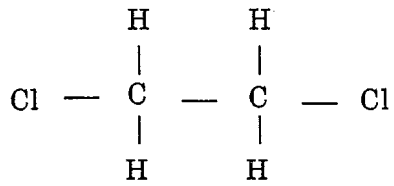
Synonyms (1): 1,2-DCA; 1,2-Ethylene dichloride; Dutch oil; ethylene chloride; ethylene dic hloride; freon 150; Brocide; Borer sol; ENI 1656; glycol dichloride

CAS Number (1): 107-06-2

Molecular Formula (1): $C_2H_4Cl_2$

Molecular Weight (1): 98.96 g/mole

Chemical Structure (1):



1,2-Dichloroethane (1,2-DCA) is a colorless chlorinated organic compound that is a man-made liquid not naturally occurring in the environment. Physical characteristics include a pleasant smell and a sweet taste. 1,2-DCA is produced in the United States mostly for the manufacture of vinyl chloride. However, it is also used in the production of tetrachloroethylene, trichloroethylene, 1,1,1-trichloroethane, vinylidene chloride, and ethyleneamines. In addition to the production of other chemicals, 1,2-DCA is also used as a degreasing agent, solvent, fumigant for grain, upholstery and carpets, paint, varnish, and finish remover, wetting and penetrating agent, and as a gasoline additive to remove lead. Of the chlorinated organic compounds produced in the United States. 1,2-DCA represents the largest quantity. Because of the toxicity and flammability of 1,2-DCA, its use as a solvent is being replaced with less hazardous chemicals as they become available (1),(3).

FATE AND TRANSPORT

Log K_{oc} (1): 1.14 to 1.28 (Range)

Log K_{ow} (2): 1.48

$t_{1/2}$ (4):

Air: 36 to 127 days

Surface Water: 0.17 days

Henry's Law Constant (2): 4.50×10^{-2} atm-m^3/mole

Fish BCF: 1.2 (4)

1.4 (3)

2.0 (bluegills) (3)

Solubility:

In Water (4): $8.52 \times 10^{+3}$ mg/L

In Organics (1): Miscible with alcohol, chloroform, ether, chlorinated solvents, and in common organic solvents

Vapor Pressure (1): 40 mm Hg @ 10°C

61 mm Hg @ 20°C

105 mm Hg @ 30°C

Density (3): 1.2530 g/mL @ 20°C

Specific Gravity (1): 1.253 @ 20°C

1,2-DCA is released into the atmosphere primarily from its production and use as a chemical intermediate, solvent, and as a lead scavenger in gasoline. 1,2-DCA degrades by reacting with hydroxyl radicals which are formed photochemically in the atmosphere and producing end products of CO2 and HCL. This photooxidation reaction results in an atmospheric half-life of approximately one month. It has also been reported that long distance atmospheric transport of 1,2-DCA is possible.

Evaporation is the primary loss pathway for release of 1,2-DCA from a water system. The estimated half-life is equated to several hours to 10 days. Chemical and biological degradation is expected to be very slow; and adsorption to sediment, hydrolysis, and bioconcentration in aquatic organisms is not expected (2).

Because of 1,2-DCA's high vapor pressure, releases to the land will evaporate fairly rapidly. The solvent is capable of rapidly leaching through sandy soil into groundwater. Once in the

groundwater, 1,2-DCA becomes persistent. Significant biodegradation in soil or groundwater is not expected; and hydrolysis is not significant (2).

PHARMACOKINETICS

In both humans and animals, 1,2-DCA is readily absorbed through the lungs following inhalation exposure. Studies examining the absorption of 1,2-DCA via the oral exposure pathway were not available. However, it is inferred from case studies that accidental or intentional ingestion of 1,2-DCA by humans reaches the systemic circulation. Human dermal exposure studies indicate that percutaneous absorption via contaminated water or the chemical itself may be a significant route of exposure. Animal studies have shown that dermal absorption is low when compared to absorption of other compounds (1).

Following inhalation exposure, 1,2-DCA was measured in the blood, liver, lung, and body fat of rats. Measurable concentrations were also found in the blood and tissues. The highest concentration of 1,2-DCA was found in the body fat of the rats. In studies that exposed pregnant rats to 1,2-DCA via inhalation, 1,2-DCA was identified in the maternal blood, placental tissue, amniotic fluid and fetal tissue. Distribution following oral administration of 1,2-DCA is similar to that of the inhalation route. It was also determined that bioaccumulation does not occur with repeated oral exposure. In terms of dermal contact, human studies indicate that 1,2-DCA can be distributed to breast milk following percutaneous exposure. No animal studies were available addressing dermal exposure to 1,2-DCA. However, it is thought that the distribution pattern may follow that of the other routes of exposure (1).

Studies addressing the metabolism of 1,2-DCA in humans following inhalation, oral, or dermal exposure were not available. However, the biotransformation of 1,2-DCA has been studied in both rats and mice in vivo and in vitro. The in vivo studies show that 1,2-DCA is readily metabolized in animals. The primary route of metabolism involved conjugation with glutathione to yield nonvolatile urinary metabolites. The enzymes involved in the biotransformation of 1,2-DCA were saturable at approximately 25 mg/kg/day via gavage and 50 ppm via inhalation. However, the metabolic pathway of 1,2-DCA is linear at low doses. At higher doses, the amount of conjugate glutathione produced is proportionally greater than the administered dose because the P450 enzymes become saturated (1).

Limited information was available regarding the excretion of 1,2-DCA following inhalation and dermal exposure. However, the available information indicates that 1,2-DCA is rapidly eliminated in the breath unchanged. No studies were identified examining excretion following human oral exposure. Animal studies have shown that 1,2-DCA is rapidly excreted through either urinary excretion or by exhalation (1).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Acute exposure to high concentrations of 1,2-DCA has resulted in respiratory distress prior to death in humans. Autopsies confirmed the presence of pulmonary congestion, edema, and irritation of the upper and lower respiratory tracts in these cases. Animal studies have shown that at concentrations equal to or greater than 1,000 ppm 1,2-DCA, respiratory distress and pulmonary congestion are possible (1).

Neurological effects, such as central nervous system excitation and depression, have been observed in both humans and animals prior to death. Morphological alterations of the nervous system have also been observed at autopsy (1).

Studies which addressed the acute effects of 1,2-DCA on the liver revealed hepatotoxicity evidenced in the form of liver dysfunction, enlarged livers, liver cirrhosis, and hepatocellular necrosis. In animals exposed to 1,2-DCA via inhalation or ingestion during acute or intermediate exposure, increased liver weight, enzyme induction, fatty infiltration, and hepatic degeneration were observed (1).

Animal studies also indicate that acute exposure to 1,2-DCA can result in perturbations of the immune system. It has been reported that 1,2-DCA reduces the animal's ability to resist infection by microbial pathogens. Also, a reduction in IgM levels, cell-mediated immune responses, and leukocyte numbers were observed in mice following acute ingestion of 1,2-DCA. It should be noted that these immunological effects have not been exhibited in humans (1).

Renal effects in humans exposed to 1,2-DCA via inhalation and ingestion consist of kidney failure and tubular and diffuse necrosis. Animal studies have revealed that renal effects, such as increased kidney weight, fatty metamorphosis and cloudy swelling, and degeneration of the tubular epithelium, have resulted from acute high-dose and intermediate lower-dose

inhalation exposure. Nephrotoxicity was not evident following long-term oral exposure to low concentrations of 1,2-DCA rats (1).

Developmental effects in association with 1,2-DCA exposure have not been observed in humans. Additionally, animal studies have revealed that mice, rats, and rabbits exposed to 1,2-DCA via inhalation and oral exposure exhibited no teratogenic effects (1).

Reproductive effects in humans have not been observed. However, some animal studies suggest that exposure to 1,2-DCA via inhalation may result in increased embryo mortality and decreased fertility in rats (1).

Although data is available describing the noncarcinogenic effects of 1,2-DCA, the EPA has not developed an RfD for either the inhalation or oral route of exposure.

Carcinogenic Effects

The EPA classifies 1,2-DCA as Group B2 carcinogen-probable human carcinogen. This classification indicates that there is sufficient evidence of carcinogenicity in animals, and inadequate evidence of carcinogenicity in humans. The basis for this classification is the induction of several tumor types in rats and mice treated by gavage and lung papillomas in mice after topical application (5).

In studies where 1,2-DCA was administered to male and female Osborne-Mendel rats and B6C3F1 mice at varied doses (47 to 299 mg/kg/day) for 32 to 78 weeks all high-dose male rats died after 23 weeks of observation and all female rats dies within 15 weeks. Male rats had increased incidence of forestomach squamous-cell carcinomas and circulatory system hemangiosarcomas. Female rats and mice had increased mammary adenocarcinoma incidence. Both sexes of mice developed alveolar/bronchiolar adenomas. In addition, the female mice developed endometrial stromal polyps and sarcomas. The male mice developed hepatocellular carcinomas (5). In another study, ICR/Ha Swiss mice treated topically with 1,2-DCA had exhibited increase incidence of benign lung papillomas (5).

In addition to the long-term animal studies, short-term genotoxic assays also support the carcinogenicity of 1,2-DCA. For example, 1,2-DCA is genotoxic in animals following oral and intraperitoneal treatment. Other studies have shown that 1,2-DCA is a weak, direct-acting mutagen that can be activated to a more patent species by glutathione and glutathione S-

transferases. These results were from short-term non-mammalian test systems. Increased mutagenic activity has been observed in studies with metabolic activation and supplemented with glutathione. 1,2-DCA binds to DNA and produces DNA damage in bacterial and human cells in vitro. It also produces both somatic and sex-linked recessive lethal mutations in Drosophila (1).

The EPA has derived cancer slope factors for 1,2-DCA using the data from the Osborne-Mendel rat and BFC3FI mice studies. The cancer slope factor for both the oral and inhalation route of exposure is 9.1×10^{-2} $(mg/kg/day)^{-1}$.

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

1,2-DCA is one of the chlorinated ethanes least toxic to aquatic life. Although data are not adequate for establishing criteria, the EPA has reported the lowest values of 1,2-DCA known to be toxic in aquatic organisms. These values are as follows (5):

Freshwater:

Acute toxicity: $1.18 \times 10^{+5}$ µg/L
Chronic toxicity: $2.0 \times 10^{+4}$ µg/L

Marine:

Acute toxicity: $1.13 \times 10^{+5}$ µg/L
Chronic toxicity: Not available

Terrestrial and Avian

Information on the toxicity of 1,2-DCA to domestic animals or terrestrial wildlife was not available (6).

REGULATORY LEVELS AND CRITERIA

EPA MCL (1):	0.005 mg/L
EPA MCLG (1):	0 mg/L
OSHA TWA (1):	1 ppm

ACGIH TLV-TWA (1):	10 ppm
NIOSH TWA (1):	1 ppm
NIOSH IDLH (1):	1000 ppm
Reportable quantity (1):	5,000 lbs
	100 lbs (proposed)
Health Advisories (1):	
1-day (child):	0.74 mg/L
10-day (child):	0.74 mg/L
Longer term (child):	0.74 mg/L
Longer term (adult):	2.6 mg/L
Water and Fish Consumption (5):	9.4×10^{-1} µg/L
Fish Consumption Only (5):	$2.43 \times 10^{+2}$ µg/L

SUMMARY OF CRITERIA

EPA Carcinogenic Classification:	Group B2-Probable Human Carcinogen
Inhalation and Oral	
Cancer Slope Factor (7):	9.1×10^{-2} $(mg/kg/day)^{-1}$

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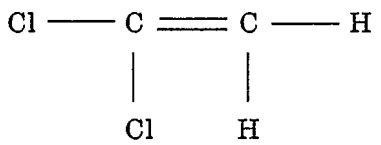
Date of Last Revision: 11/10/94

Revisor: Patrick B. Moroney

1,1-DICHLOROETHENE

INTRODUCTION

Chemical Name: 1,1-Dichloroethene
Synonyms (1): 1,1-DCE; VDC; Vinylidene chloride; Vinylidene dichloride
CAS Number (1): 75-35-4
Molecular Formula (1): $C_2H_2Cl_2$
Molecular Weight (2): 96.95 g/mole
Chemical Structure (1), (2):



1,1-Dichloroethene (1,1-DCE) is a clear, colorless liquid that has a mild, sweet smell. It is a man-made chemical that does not naturally occur in the environment. The most common use of 1,1-DCE is in the production of plastics such as packaging materials and flame-retardant fabrics. 1,1-DCE is also used for coating the interior of ship tanks and railroad tank cars, as well as steel piles and structures. Another use is as an intermediate in the production of 1,1,1-trichloroethane and in the manufacture of modacrylic fibers where it is combined with acrylonitrile (1),(2)

FATE AND TRANSPORT

Log K_{oc} (2): 1.81
Log K_{ow} (2): 2.13
(3): 1.84
$t_{1/2}$ (2): Air - 2.00 days
Surface Water - (1.00 - 6.00 days)
Henry's Law Constant (3): 3.40×10^{-2} atm-m^3/mol
Fish BCF (3): 5.6
Solubility:
In Water (3): $2.25 \times 10^{+3}$ mg/L
In Organics (2): Soluble
Vapor Pressure (2): 500 mm Hg @ 20°C
591 mm Hg @ 25°C
720 mm Hg @ 30°C
Density (1): 1.21 g/ml @ 20°C

The majority of 1,1-DCE released to the environment partitions to the atmosphere. 1,1-DCE is water soluble, and when released to the environment if will dissolve and remain in solution, resulting in surface water or groundwater contamination. 1,1-DCE has a low propensity for binding to organic or particulate matter, and therefore its movement within and between media is not inhibited by the adsorption processes (2).

In the atmosphere, 1,1-DCE reacts with OH and NO radicals to produce chloroacetyl chloride, phosgene, formaldehyde, carbon monoxide and nitric acid. Rapid photolysis occurs in the presence of nitrogen oxides. Also, rapid photodecomposition occurs in the presence of NOx and artificial lighting conditions. Because of a short atmospheric half-life, 1,1-DCE does not accumulate in the troposphere (2).

In waterways, biotransformation is the primary degradation pathway for 1,1-DCE. Biotransformation can occur either under aerobic or anaerobic conditions although results under aerobic conditions are conflicting. However, under anaerobic conditions, 1,1-DCE is nearly completely and quantitatively reduced to vinyl chloride after 108 days. Other degredation pathways, such as photolysis, hydrolysis, and oxidation are not significant transformation mechanisms for 1,1-DCE (2).

Information regarding soil degradation processes under aerobic conditions is lacking. Because of the tendency of 1,1-DCE to partition into the atmosphere, ambient concentrations in surface soil are expected to be low (2).

PHARMACOKINETICS

No human studies were available in the reviewed literature regarding absorption of 1,1-DCE by the inhalation, oral, or dermal exposure pathways. However, animal studies have revealed that 1,1-DCE rapidly absorbs following inhalation exposure. The 1,1-DCE absorption patterns suggest that the cumulative uptake was linear for levels up to 150 ppm. Steady-state was not achieved for higher levels. This indicates that 1,1-DCE uptake is saturable at higher levels. Animal studies involving absorption via oral exposure have indicated that 1,1-DCE ranging from 10 to 100 mg DCE/kg body weight is rapidly absorbed from the gastrointestinal tract of rats and mice. Because of the lack of human and animal studies involving dermal contact with 1,1-DCE, only assumptions on the absorption process can be made. Based on the physical and chemical properties of 1,1-DCE, it is thought that percutaneous absorption of the liquid form of 1,1-DCE is possible (2).

Human and animal studies regarding the distribution of 1,1-DCE through dermal exposure are not available. Studies have shown that 1,1-DCE is found in the liver and kidneys of rats following inhalation and oral exposure. Only small amounts were present in other tissues (2).

Studies denoting the human metabolism pattern of 1,1-DCE following exposure are not available. Animal studies indicate that the metabolic process is dependent upon the route of exposure and dose level. The initial step in the metabolism of 1,1-DCE is the formation of an epoxide intermediate. The main detoxification pathway for 1,1-DCE is purported to involve conjugation with hepatic glutathione. Conjugation with monochloroacetic acid followed by B-thionase activity appears to be the major metabolic route on a quantitative basis in rats. Thiodiglycollic acid is the predominant urinary metabolite (2).

No studies were available that addressed the excretion of 1,1-DCE in humans following inhalation, oral or dermal exposure. In rats, the majority of absorbed material is eliminated as metabolites in the urine. Approximately 1% of the parent compound is eliminated unchanged in expired air following inhalation. However, a greater portion may be eliminated unchanged through the lung once the metabolic processes become saturated. Excretion through the oral route of exposure is similar to the inhalation route. No studies were available concerning the excretion of 1,1-DCE following dermal contact (2).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Studies have shown that 1,1-DCE is toxic to both humans and animals. The source of human studies are primarily from accidental exposure to high concentrations for short periods of time. 1,1-DCE toxicity is mediated by metabolism to a reactive intermediate that ultimately compromises the viability of target organs. Animal studies have shown that the major target organs of 1,1-DCE toxicity are the liver, kidney, and lungs (2).

Respiratory effects in animals consist of irritation of the mucous membranes of the upper respiratory tract and pulmonary congestion, hyperemia, and morphological changes. These effects were induced by administration of high levels of 1,1-DCE via inhalation. It is believed in humans, that local, nonspecific irritation is exhibited upon inhalation of 1,1-DCE (2).

Hepatic effects in animals were exhibited following both inhalation and oral exposure to 1,1-DCE. Manifestations include biochemical changes and mild marked histological changes such as midzonal and/or centrilobular vacuolization, swelling, degeneration and necrosis. It is assumed that humans who have either been exposed to high levels of 1,1-DCE in an occupational setting, or those with compromised hepatic function, are at risk for 1,1-DCE induced liver toxicity (2).

Renal effects consisting of enzyme changes, hemoglobinuria, increases in organ weight, and tubular swelling, degeneration and necrosis are observed following both inhalation and oral exposure to 1,1-DCE in animals. Renal effects triggered by long-term exposure to 1,1-DCE in humans are not known (2).

Exposure to high concentrations of 1,1-DCE in humans affects the central nervous system. Complete recovery is possible if exposure is not prolonged. Also, central nervous system effects were observed in animals exposed via the inhalation route (2).

1,1-DCE has also exhibited teratogenic potential in animals. Developmental toxicity appears to be enhanced by inhalation exposure to 1,1-DCE as compared to oral exposure. Studies have shown that inhalation exposure at 80 to 160 ppm 1,1-DCE for 7 hours/day on gestation days 6 - 18 produced maternal toxicity, increased resorption, and skeletal alterations (2).

An oral RfD for 1,1-DCE has been derived by the EPA using the data from a drinking water study with Sprague-Dawley rate. Male and female rats were administered 50, 100 or 200 ppm 1,1-DCE in drinking water over a two year period and observed for treatment-related effects. From this study, a lowest observed adverse effect level (LOAEL) of 9 mg/kg/day was determined based on Sepotic lesions in the female rats. An uncertainty factory of 1000 was applied to this LOAEL to arrive at an oral RfD of 9×10^{-3} mg/kg/day.

Carcinogenic Effects

The EPA classified 1,1-DCE as a Group C carcinogen - possible human carcinogen. This classification is based on limited evidence of carcinogenicity in animals (4).

Studies involving the use of Swiss mice in the inhalation administration of 1,1-DCE have shown that at 25 ppm 1,1-DCE 4 hours daily, 5 days per week for 52 weeks, 1,1-DCE induces malignant tumors. Renal adenocarcinomas developed in 16% of male mice and 0.6% of the female mice (1). Mammary gland carcinomas and pulmonary adenomas were also observed in this study. The calculated inhalation and oral cancer slope factor for 1,1-DCE is 1.2 $(mg/kg/day)^{-1}$ (2). These cancer slope factors were derived using the data from the inhalation

study with Swiss mice described above. In another study, mice that had been topically administered 1,1-DCE showed an increased incidence of skin papillomas (1).

Supporting short-term assays are available for the carcinogen effects seen in animals. Genotoxicity studies suggest that 1,1-DCE exhibited mutagenic properties upon metabolic activation in bacteria and yeast. It is also capable of inducing gene conversion in yeast. In green plants, 1,1-DCE proved to be mutagenic without metabolic activation by mammalian systems. Chromosomal aberrations and sister chromatid exchanges in cultured mammalian cells in vitro and DNA damage in mice in vivo were exhibited in 1,1-DCE studies (2).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Ambient water quality lowest effect levels for the protection of aquatic organisms are as follows (4):

Freshwater:
- Acute Toxicity: $1.16 \times 10^{+4}$ µg/L
- Chronic Toxicity: None

Marine:
- Acute Toxicity: $2.24 \times 10^{+5}$ µg/L
- Chronic Toxicity: None

These quantifications occur at lower concentrations among species that are more sensitive than those tested (5).

Terrestrial and Avian

Information regarding the toxicity of 1,1-DCE to terrestrial and avian wildlife or domestic animals was not available (6).

REGULATORY LEVELS AND CRITERIA

OSHA TWA (8-hr) (1):	1 ppm
ACGIH TLV (8-hr TWA) (1):	5 ppm
ACGIH (15-min) (1):	20 ppm
EPA MCLG (1):	7 µg/L
EPA MCL (1):	7 µg/L
EPA Health Advisories (1):	
1-day (child):	2 mg/L
10-day (child):	1 mg/L
Longer-term (child):	1 mg/L
Longer-term (adult):	4 mg/L
Lifetime (adult):	7 µg/L
Reportable Quantity (1):	100 lbs
Ambient Water Quality Criteria (4):	
Water and Fish Consumption:	3.3×10^{-2} µg/L
Fish Consumption Only:	1.85 µg/L

SUMMARY OF CRITERIA

EPA Carcinogenic Classification (4):	Group C-Possible human carcinogen
Oral RfD (4):	9.00×10^{-3} mg/kg/day
Oral Cancer Slope Factor (4):	6.00×10^{-1} (mg/kg/day)$^{-1}$
Inhalation Cancer Slope Factor (4):	1.75×10^{-1} (mg/kg/day)$^{-1}$
LOAEL (4):	9 mg/kg/day

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Revisor: Patrick B. Moroney

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

INTRODUCTION

Chemical Name:	1,2-Dichloroethene (cis- and trans-)
Synonyms (1):	1,2-Dichloro-(z)ethene; Cis-1,2-dichloroethene; Cis-1,2-dichloroethylene; Trans-1,2-dichloroethylene; 1,2-DCE
CAS Number (2):	Cis- 156-59-2 Trans- 156-60-5
Molecular Formula (2):	$C_2H_2Cl_2$
Molecular Weight (2):	96.94 g/mole
Chemical Structure (2):	

cis trans

1,2-Dichloroethene (1,2-DCE) exists as the cis and trans isomers. It is a colorless, low-boiling liquid. The use of 1,2-DCE in the United States has not been widely developed due to its flammability characteristics. The trans isomer is used more frequently than the cis isomer or the 60:40 cis/trans mixture. The primary uses include a low-temperature extraction solvent or direct solvent in materials such as dyes, perfume oils, waxes, resins and thermoplastics (1).

FATE AND TRANSPORT

	Cis-	Trans-
K_{oc} (3):	49	59
Log K_{ow} (2):	2.06	1.86
1/2t:		
Air (3):	2.10 days	130 days
Surface Water (3):	1.0 to 6.0 days	1.0 to 6.0 days
Henry's Law Constant (2): (atm-m3/mole)	3.37×10^{-3}	6.72×10^{-3}
Fish BCF (3):	1.6	1.6
Solubility (2):		
In Water:	6.3 g/L	3.5 g/L
In Organics:	Miscible with alcohol, ether, and acetone; very soluble in benzene and chloroform	
Vapor Pressure (2):	340 mm Hg @ 25°C	200 mm Hg @ 35°C

Cis- and trans-1,2 DCE are released into the environment primarily in air emissions and wastewater during its production and use in the manufacturing of perfumes, lacquers, and thermoplastics. Also, 1,2-DCE is formed from trichloroethylene, tetrachloroethylene, and 1,1,2,2-tetrachloroethane. It

may exist in landfills, aquifers, and sediments. Cis-1,2-DCE is the most common isomer found; however, it is frequently misreported as trans-1,2-DCE, a priority pollutant. Most analytical procedures do not distinguish between the two isomers (2).

When released on to the soil, 1,2-DCE will either evaporate or leach into the ground water where very slow biodegradation occurs (2).

1,2-DCE will volatilize upon introduction into a water system with a half-life of 3 hours in a model river. Because of the relatively low K_{oc} value, it is suggested that biodegradation and adsorption to sediment will not be significant for either isomer (2).

In the atmosphere, 1,2-DCE will be reactive with photochemically produced hydroxyl radicals. The long half-life, 8 days, will allow for atmospheric dispersion prior to scavenging by rain (2).

1,2-DCE is not expected to bioconcentrate significantly in aquatic organisms. The bioconcentration factor, 1.6, was estimated from the log K_{ow} using a recommended regression equation (2).

PHARMACOKINETICS

Studies indicating the absorption, distribution, metabolism, and excretion of cis- and trans-1,2-DCE were not available. Because 1,2-DCE is not widely used, toxicologic data are limited.

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

The majority of 1,2-DCE studies are old an must be considered suspect. The purity of the samples and the ratio of the isomers were not always identified (1).

Oral LD_{50}s have been established for male and female mice exposed to trans-1,2-DCE. In male mice, an oral LD_{50} of 2122 mg/kg was reported while the LD_{50} for female mice was 2391 mg/kg.

Mice exposed to 1,2-DCE at concentrations of 0.1,1.0, or 2.0 mg/ml in drinking water for 90 days showed minimal toxic effects. At a concentration of 2.0 mg/ml, hepatic glutathione levels were significantly reduced in male mice and hepatic aniline hydroxylase activity was reduced in female mice at all three dose concentrations (1). In another study involving trans-1,2-DCE exposure at 402, 1311, or 3114 mg/kg/day in male rats and 353, 1257, or 2809 mg/kg/day in female rats for 90 days, no treatment related effects were observed which effected fluid consumption, body weight, hematology, serum chemistry, or urinalysis. A significant dose-dependent decrease in kidney weight was observed in female rats at the 1257 and 2809 mg/kg/day level (1). Lung effects were also reported in rats exposed to a single 8-hour exposure to 200 ppm of trans-1,2-DCE (1).

Other chronic toxicity studies have produced conflicting results. Some studies have demonstrated no adverse effects in rats, rabbits, guinea pigs and dogs exposed to either 500 or 1000 ppm of a 60% cis- / 40% trans- mixture of 1,2-DCE. The exposure lasted for 7 hours per day, 5 days per week for 6 months. However, other studies have reported that rats exposed to vapors of 200 ppm of trans-1,2-DCE for 8 hours per day, 5 days per week for 16 weeks, produced adverse liver and lung effects, as well as decreased leukocyte counts (1).

Human studies are limited. It is known that 1,2-DCE was once used as a general anesthetic. In its limited industrial usage, no toxic effects due to occupational exposure have been noted. Symptoms associated with exposure to 2000 ppm of trans-1,2-DCE include burning of the eyes, vertigo and nausea. Because of its limited use, no long-term epidemiological studies have been conducted for 1,2-DCE exposure (1).

Data examining teratogencity, embryotoxicity and reproductive effects were not available (1).

HEAST has assigned an oral RfD value of 2.0×10^{-2} mg/kg/day to trans-1,2-DCE. From this study, a lowest observed adverse effect level (LOAEL) of 175 mg/kg/day and a no observed adverse effect level (NOAEL) of 17 mg/kg/day were determined. For cis-1,2-DCE, an oral RfD of 1.0×10^{-2} mg/kg/day has been established. This oral RfD value is based on a NOAEL of 32 mg/kg/day.

The oral trans-1,2-DCE RfD value was based on a study involving the administration of trans-1,2,DCE in drinking water to male and female CD-1 mice in concentrations ranging from 17 to 452 mg/kg/day for 90 days. In the male mice, there were significant increases in serum alkaline phosphatase levels at the highest concentrations of trans-1,2-DCE. In female mice, the thymus weight and lung weight were decreased at the higher concentrations of trans-1,2-DCE (7). The oral cis-1,2-DCE value was based on a study involving administration via gavage to rats over a 90-day time frame. Critical end points for this study were decreased hematocrit and hemoglobin in the exposed rats.

Carcinogenic Effects

The EPA classifies cis-1,2-Dichloroethene is a Group D carcinogen - not classifiable as to human carcinogenicity. This classification is based on the available human and animal studies (7). Information regarding the carcinogenicity of trans-1,2-dichloroethene has not been evaluated by the EPA for evidence of human carcinogenic potential (7).

The available supporting carcinogenicity data do not indicate a carcinogenic potential in animals. Genotoxic assays revealed negative results when 1,2-DCE was tested on four standard strains of Salmonella typhimurium. Negative results were also obtained in E. coli K12 reversion tests in the presence of mouse liver enzymes. In studies involving S. cerevisiae in suspension and in a host-mediated assay, both isomers were able to induce convertants in the suspension assay but only with metabolic activation. Only the cis isomer was capable of inducing revertants and convertants in the host-mediated assay. No increase in chromosomal aberrations or sister chromatid exchanges, with or without metabolic activation, was found in studies involving Chinese hamster lung cells treated with 1,2-DCE isomers (1).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Although data are not adequate for establishing criteria, the EPA has reported the lowest values of dichloroethylenes known to be toxic in aquatic organisms. These values are as follows (5):

Freshwater:

Acute Toxicity:	$11.6 \times 10^{+4}$ µg/L
Chronic Toxicity:	Not available

Marine:

Acute Toxicity:	$2.24 \times 10^{+5}$ µg/L
Chronic Toxicity:	Not available

Terrestrial and Avian

Information regarding the toxicity of 1,2-trans-DCE to wildlife and domestic animals is limited. For bluegill, the reported 96-hour LC_{50} value under static conditions is 1.35 x 10+5 µg/L (4).

No information addressing the toxicity of 1,2-cis-DCE to terrestrial and avian wildlife or domestic animals was available (4).

REGULATORY LEVELS AND CRITERIA

OSHA TWA (8-hr) (1):	200 ppm
ACGIH TLV (8-hr TWA) (1):	200 ppm
MCL (1):	Cis- 70 µg/L
	Trans- 100 µg/L

EPA Health Advisories and Cancer Risk Levels (1):

- 1 day (child):
  - Cis- 4 mg/L
  - Trans- 20 mg/L
- 10 day (child):
  - Cis- 1 mg/L
  - Trans- 2 mg/L
- Longer term (child):
  - Cis- 1 mg/L
  - Trans- 2 mg/L
- Longer term (adult):
  - Cis- 1 mg/L
  - Trans- 6 mg/L
- Lifetime (adult):
  - Cis- 0.07 mg/L
  - Trans- 0.1 mg/L

SUMMARY OF CRITERIA

EPA Carcinogenic Classification (7):	Group D-not classifiable as to human carcinogenicity
Oral RfD (trans-) (7):	2.0×10^{-2} mg/kg/day
Oral RfD (cis-) (6):	1.0×10^{-2} mg/kg/day
Oral RfD (mixture) (6):	9.0x10-3 mg/kg/day
NOAEL:	17 mg/kg/day
LOAEL:	175 mg/kg/day

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Revisor: Rich Hoff

DDT SERIES PESTICIDES

INTRODUCTION

Technical DDT is a mixture containing 65-80% p,p'-DDT, 15-20% o,-p' DDT and up to 4% p,p'-DDD plus other trace materials. The metabolites of DDT include p,p'-DDE and o,p'-DDD. DDT isomers and their metabolites are usually found together and generally have the similar properties, therefore DDT has been referenced as the total combination of isomers and metabolites. However, specific differences between isomers are identified.

DDT, along with it's metabolites, are organochlorine pesticides which are very persistent in the environment. Historically, DDT was released to the environment during its formulation and extensive use as a pesticide in agricultural and vector control applications. It was banned from use in the United States in 1972, but is still used in other areas of the world. These compounds have been shown to be carcinogenic in mice and chronic exposure primarily cause liver and lung tumors and lymphomas. In addition, the nervous system appears to be one of the primary target organs for DDT toxicity in humans (6). In general, organochlorine pesticides are highly toxic to aquatic organisms, in addition to being responsible for a decreased population of many bird species (1).

Chemical Name:

p,p'-DDT:	p,p'-1,1,1-Trichloro-2,2-bis(4-chlorophenyl)ethane
p,p'-DDD:	p,p'-1,1-Dichloro-2,2-bis(4-chlorophenyl)-ethane
p,p'-DDE:	p,p'-1,1-Dichloro-2,2-bis(4-chlorophenyl)-ethene

CAS Number (1):

p,p'-DDT:	50-29-3
p,p'-DDD:	72-54-8
p,p'-DDE:	72-55-9

Molecular Formula (1):

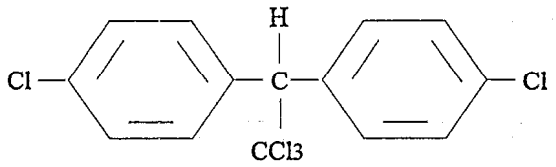
p,p'-DDT:	$C_{14}H_9Cl_5$
p,p'-DDD:	$C_{13}H_{10}Cl_4$
p,p'-DDE:	$C_{14}H_8Cl_4$

Molecular Weight (1):

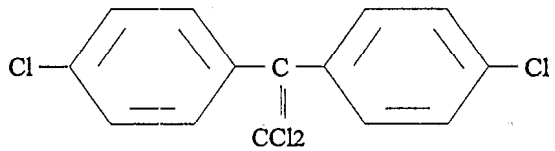
p,p'-DDT:	354.5 g/mole
p,p'-DDD:	320 g/mole
p,p'-DDE:	318 g/mole

Chemical Structure (6):

DDT:



DDE:



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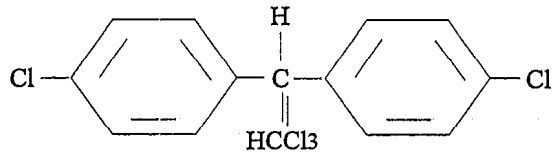

FATE AND TRANSPORT

Chemical and Physical Properties

Log K_{ow} (6):

p,p'-DDT:	6.19
p,p'-DDD:	6.20
p,p'-DDE:	7.00

Log K_{oc} (6):

p,p'-DDT:	5.4
p,p'-DDD:	5.9
p,p'-DDE:	6.6

Boiling Point (1):

p,p-DDT:	260^{0}C

Melting Point:

DDT:	109^{0}C
DDD:	112^{0}C
DDE:	90^{0}C

Solubility in Water @ 25^0C (6):
p,p'-DDT: 3.4 μg/L
p,p'-DDD: 160 μg/L
p,p'-DDE: 120 μg/L

Solubility in Organics (1):
p,p'-DDT: Soluble in acetone, benzene, cyclohexane, morpholine, pyridine, and dioxane

Vapor Pressure (1):
p,p'-DDT: $1.9E^{-7}$ mm Hg at 25^0C
p,p'-DDD: $1.0E^{-6}$ mm Hg at 30^0C
p,p'-DDE: $6.5E^{-6}$ mm Hg at 20^0C

TRANSPORT AND PARTITIONING

DDT and its metabolites may be transported from one medium to another by the processes of solubilization, adsorption, bioaccumulation, or volatilization. Each of these processes will be discussed in the following paragraphs. Studies of DDT transformations in soils predict prolonged persistence. During these extended periods of time, these compounds undergo extensive adsorption to soil particles, as predicted by their organic carbon partition coefficients (K_{oc}) of 2.4 x 10^5, 4.4 x 10^6, and 7.7x10^5, for DDT, DDE and DDD, respectively. DDT, DDE and DDD are only slightly soluble in water (solubilities of 0.0034, 0.12, and 0.160 mg/L at 25°C, respectively). Therefore, loss of these compounds in runoff is primarily due to transport of particulates to which these compounds are bound. Since they are bound strongly to soil, they are not easily displaced from their site of application, nor do they tend to leach to groundwater, and appreciable amounts may remain in the soil for extended periods of time. When DDT is released to water, it quickly adsorbs to particles and is subject to sedimentation, or may bioconcentrate in microorganisms and can become part of the food chain.(6)

Volatilization of DDT and DDE is known to account for considerable losses of these compounds from solids and water. Their tendency to volatilize can be predicted by their Henry's Law Constants, 5.13 x 10^{-4} and 6.8 x 10^{-5} atm-m^3/mol, respectively, and their vapor pressures, 5.5 x 10^{-6} and 6.5 x 10^{-6} Torr, respectively. The tendency of DDD to volatilize is approximately three-fold less than that of DDT or DDE. Estimates of the rate of evaporative losses of DDT from water range from several hours to 50 hours. Laboratory studies of the air/water partition coefficient of DDE indicate that it will volatilize from seawater 10 to 20 times faster than from freshwater. The authors suggest that this process may be related to interaction at the bubble-water surface (6).

Small particles which carry DDT or its degradation products may also be distributed through the atmosphere. Once volatilized or airborne, transport of these compounds into the ambient atmosphere of North America is facilitated by a circulation pattern which brings moisture from the Gulf of Mexico into the Midwest and airflow patterns across the eastern seaboard. Residues are removed from the atmosphere by precipitation, diffusion into large bodies of water, and chemical transformation. Precipitation is believed to account for the greatest rate of removal (6).

DDT, DDE, and DDD are highly lipid soluble, as reflected by their log octanol-water partition coefficients (log K_{ow}) of 6.19, 7.00, and 6.20, respectively. This lipophilic property, combined with an extremely long half-life, has resulted in bioaccumulation (levels in organisms exceed those levels

occurring in the surrounding environment). When they are present in ambient water, DDT and its metabolites are concentrated in freshwater and marine plankton, insects, mollusks, other invertebrates, and fish. As these organisms become part of the food chain, a progressive accumulation of residues may result in high levels of residues in organisms at the top of the food chain. In some cases, man may be the ultimate consumer of these contaminated organisms. The bioconcentration factor (BCF) is defined as the ratio at equilibrium between the concentration of the chemical in the organism or tissue of the organism, and the concentration of the chemical in the surrounding media. There are numerous measurements and estimates of BCFs in fish. Oliver and Niimi estimated the steady-state BCF in rainbow trout as 12000.

TRANSFORMATION AND DEGRADATION

The dominant mechanism for removal of DDT from the atmosphere is believed to be via precipitation and secondarily, particulate fallout. Under simulated atmospheric conditions, both DDT and DDE decompose to form carbon dioxide and hydrochloric acid (6).

AIR

In air and sunlight, DDT is subject to direct photooxidation and reaction with photochemically produced hydroxyl radicals. The latter process has an estimated half-life of two days. Since DDT residues are ubiquitous in the atmosphere, it seems likely that photodegradation must occur at a slow rate. DDT which reaches the photochemically active ionosphere may be rapidly destroyed by solar irradiation as indicated under laboratory conditions (6).

WATER

DDT present in water may be partitioned, transported, or converted in several ways: adsorption to sediments, bioconcentration in aquatic organisms, volatilization, photodegradation, and biodegradation. DDT in water is photodegradable by wavelengths of light which are present in the troposphere. DDT in excess of water solubility limits is adsorbed onto sediments which act as the primary reservoir for excess quantities of DDT. There it is available for ingestion by organisms. Biodegradation is reported to be a minor source of transformation (6).

SOIL

Four mechanisms have been suggested to account for most losses of DDT residues from soils: (1) volatilization, (2) removal by harvest of organic matter, (3) water runoff and (4) chemical transformation. DDT is subject to volatilization, with an estimated half-life of 100 days. Photoxidation of DDT is known to occur on soil surfaces; however, DDT is not known to hydrolyze. Biodegradation may occur under both aerobic and anaerobic conditions in the presence of certain microorganisms. Under aerobic conditions, slow conversion to DDE normally occurs, whereas, under anaerobic conditions, conversion to DDD results and is much more rapid than the aerobic conversion to DDE. Under aerobic conditions, dehydrochlorination is the dominant reaction, while under anaerobic conditions, reductive dechlorination occurs. Both metabolites are very resistant to further transformation. Due to the prolonged half-life of DDE, average levels of DDT are expected to decline slowly while the ratio of DDE to DDT is expected to increase. Estimates of the half-life for DDT biodegraded in soil range from 2 to >15 years (6).

PHARMACOKINETICS

Absorption of DDT by the lung is considered to be a minor route of entry. Due to the large particle size, it does not enter into the smaller spaces of the lung, but is deposited in the upper respiratory tract, and due to the action of the mucociliary apparatus it is eventually swallowed. However, some particles may be small enough to enter the tracheal system. No studies have been located that quantify the rate or extent of absorption of DDT, DDD or DDE (6).

Absorption following ingestion of DDT, DDE and DDD is evident in humans from measurements of serum and adipose tissue concentrations of these chemicals after occupational exposure. Gastrointestinal absorption is evidenced by the presence of DDT and metabolites measured in bile and the induction of tumors in animals following oral administration of the compounds. Approximately 70–90% of the dose has been absorbed following oral exposure to DDT in vegetable oils (6).

Dermal absorption of DDT in humans is considered to be limited, but can be inferred by observation of toxicity following dermal application of DDT. Acute toxicity studies have shown that toxicity is less when DDT is applied dermally than when given by gavage or injection (6).

The distribution and storage of DDT in humans and animals has been extensively studied. DDT and its metabolites are lipid soluble compounds, and once absorbed readily distribute to all body tissues in proportion to respective tissue lipid content (6).

HUMAN HEALTH EFFECTS

Carcinogenic Effects

DDT has been classified as a B2 carcinogen, a probable human carcinogen, however, the existing epidemiological human health research data for carcinogenicity are inadequate . The basis for the B2 classification is the observation of tumors (generally liver tumors) in various mouse strains and in three studies of rats. In addition, DDT is structurally similar to other probable carcinogens such as DDD and DDE. Lung tumors have also been observed in chronic studies of mice, while increased tumor incidence in hamsters have also been observed (2).

DDT has produced both negative and positive responses in tests for genotoxicity (2).

An oral slope factor (CSF) for DDT has been set at 3.4×10^{-1} $(mg/kg/day)^{-1}$ by the United States Environmental Protection Agency (USEPA). An inhalation unit risk number has also been set at $9.7E^{-5}$ $\mu g/m^3$, or an inhalation slope factor of 3.4×10^{-1} $(mg/kg/day)^{-1}$ (2).

For DDD the USEPA has set an oral slope factor of 2.4×10^{-1} $(mg/kg/day)^{-1}$, and for DDE the USEPA has set an oral slope factor of 3.4×10^{-1} $(mg/kg/day)^{-1}$ (2).

Noncarcinogenic Effects

Increased hepatocellular hypertrophy, cytoplasmic oxyphilia and peripheral basophilic cytoplasmic granules were observed in chronic rat studies where rats were fed commercial DDT. Liver lesions were also seen in one 2 year rat study. In addition, DDT-induced liver effects were observed in mice, hamsters and dogs. One three generation rat reproduction study showed offspring mortality at all administered doses. Three other rat studies showed no reproductive effects at much higher dose levels (2).

An oral reference dose (RfD) of $5x10^{-4}$ mg/kg/day has been set by the USEPA. The Lowest Observed Adverse Effects Level for the study in which the RfD was derived was 0.25 mg/kg-d. The study from which the RfD was derived was chosen for the following reasons; male rats were used in the study (male rats are more sensitive than female rats to DDT), the study was of sufficient length to observe toxic effects and several doses were administered in the diet over the range of the dose-response curve (2).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

DDT has been extensively studied in freshwater invertebrates and fish and is quite toxic to most species. Saltwater species are even more sensitive than freshwater species (1).

Ambient Water Quality Criteria (AWQC) have been set for DDT, however, criteria have not been set for DDD and DDE, the Lowest Observed Adverse Effects Level (LOAEL) have been reported as a guideline for aquatic adverse effects for these two compounds (3).

DDT: (AWQC)

Freshwater Acute:	1.1 μg/L
Freshwater Chronic:	0.001 μg/L
Marine Acute:	0.13 μg/L
Marine Chronic:	0.001 μg/L

DDD: (LOAEL)

Freshwater Acute:	0.6 μg/L
Marine Acute:	3.6 μg/L

DDE: (LOAEL)

Freshwater Acute:	1,050 μg/L
Marine Acute:	14 μg/L

Terrestrial and Avian

DDT, DDD and DDE and other persistent organochlorine pesticides are primarily responsible for the great decrease in the reproductive capabilities and consequently in the populations of fish-eating birds, such as the bald eagle, brown pelican, and osprey. DDT has also been shown to decrease the populations of numerous other species of waterbirds, raptors, and passerines significantly (1).

OCCUPATIONAL REGULATORY LEVELS AND CRITERIA

Occupational Safety and Health Administration (OSHA) Permissible Exposure Limit (PEL), Time-weighted average for DDT: 1 mg/m^3, skin (5).

American Conference of Governmental Industrial Hygienist (ACGIH) Time-Weighted Average Threshold Limit Value (TLV) for DDT: 1 mg/m^3 (4).

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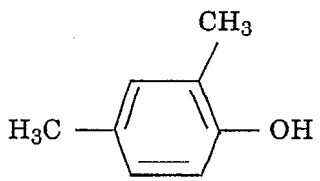
Date of Last Revision: 11/10/94
Revisor: Patrick B. Moroney

2,4-DIMETHYLPHENOL

INTRODUCTION

Chemical Name: 2,4-Dimethylphenol
CAS Number: 105-67-9
Common Names: 2,4-Xylenol; 4,6-Dimethylphenol
Molecular Formula: $C_8H_{10}O$
Molecular Weight: 122.16 g/mole
Chemical Structure:



2,4-Dimethylphenol is a white crystalline solid. It is a naturally-occurring compound. 2,4-Dimethylphenol is a substituted phenol derived from the cresol fraction of petroleum or coal tars and is one of five isomers of dimethylphenol. Presently, there are no direct commercial applications for 2,4-dimethylphenol. However, it is used in the manufacture of a wide range of industrial and agricultural products including phenolic antioxidants, disinfectants, solvents, pharmaceuticals, insecticides, plasticizers, rubber chemicals, polyphenylene oxide, wetting agents and dyestuffs (1).

FATE AND TRANSPORT

Log Koc: 1.98 (1)
Log Kow: 2.3 (1,2); 2.5 (3)
Half-life: Half-hour to days in water (2)

Henry's Law Constant: 6.7×10^{-6} atm-m^3/mole @ 20°C (1)
BCF: 9.5 (estimated), 150 (bluegill) (1)
Degradation Products: Not available
Solubility:
- Water: 1.48×10^3 mg/L @20°C (1); 6.2×10^3 mg/L @25°C; 1.7×10^4 mg/L (3)
- Organic Solvents: Freely soluble in alcohol, chloroform, ether and benzene (3)

Vapor Pressure: 6.7×10^{-2} mm Hg @ 20°C (1); 0.098 mm Hg @25°C (2); 0.06 mm Hg @ 20°C (3)
Density: 0.965 mg/L @20°C(1)

When dissolved in water, sorbed on soil or present as a separate organic phase as a result of a large spill, 2,4-dimethylphenol will migrate easily through soil and water systems. 2,4-Dimethylphenol is fairly mobile in soil-water systems, particularly in the aqueous and air phases. This compound can migrate through soil via bulk transport (i.e, downward movement of infiltrating water), dispersion and diffusion. Its mobility is affected by the extent of its

sorption on soil. Based on the low soil sorption coefficient (Koc), this compound sorbs weakly to soils. However, this is based on the assumption that 2,4-dimethylphenol acts as a neutral species. Under most conditions, it will remain in a neutral, nonionized state. However, the 2,4-isomer of dimethylphenol is a weak acid. It will slightly disocciate in natural waters with elevated pHs. Additionally, the phenolic group can form complexes with dissolved metal cations. This can affect the sorption properties of the chemical in unpredictable ways (1).

Although the persistence of this compound has not been studied, it is assumed that it will persist for a time perios of months to years (1).

A small portion of 2,4-dimethylphenol in the gaseous phase of the soil sytem can diffuse through the soil-air pores up to the ground surface and subsequently be removed by wind dispersion.

However, volatilization through air-filled pores is not considered to be significant. This is because of the chemical's low vapor pressure and relatively high water solubility, which allows it to migrate via infiltrating water (1).

Under normal environmental conditions, this compound is resistant to hydrolysis. However, 2,4-dimethylphenol may be easily biodegraded in wastewater treatment plants. As previously mentioned, there is also the possibility of aqueous phase oxidation via metal catalysis. 2,4-Dimethylphenol does not biodegrade at significant rates in natural environments such as rivers. This is because the concentrations of microorganisms capable of biodegrading 2,4-dimethylphenol in water are very low and drop off substantially with increasing depth (1).

In summary, the volatility of 2,4-dimethylphenol from aqueous solutions and the ability of this compound to be adsorbed by soil is low. 2,4-Dimethylphenol also has a low potential for bioaccumulation. The primary exposure pathway for this compound would be leaching to groundwater. Inhalation is not a major route of exposure.

PHARMACOKINETICS

No information pertaining to human toxicity as a result of exposure to 2,4-dimethyphenol currently exist. It is unlikely that a segment of the population is exposed to this compound alone. Many industrial workers have been exposed via inhalation to commerical degreasing agents that contain dimethylphenols. However, no adverse health effects have been reported. Since this compound has been identified in cigarette and marijuana smoke, smokers and nonsmokers may be at risk (1).

HUMAN HEALTH EFFECTS

Nominally, 2,4-dimethylphenol is a cocarcinogen. Skin-painting and tumor-promotion studies have been conducted using 2,4-dimethylphenol. However, benzene was used as the vehicle for compound administration. Hence, it is difficult to determine the health impact made by the sole contribution of 2,4-dimethylphenol. In addition, there is little information on the genotoxic, reproductive and long-term effects of exposure to 2,4-dimethylphenol (1).

At high doses, other dimethyphenols have been shown to cause pathological changes in the liver, kidneys, and heart. 2,4-Dimethylphenol has been shown to be an ATP blocking agent (3).

Noncarcinogenic Effects

The USEPA oral reference dose for 2,4-dimethylphenol is $2x10^{-2}$ mg/kg/day. There is no inhalation reference concentration value.

Carcinogenic Effects

There is no oral or inhalation cancer potency factor for 2,4-dimethylphenol.

ENVIRONMENTAL HEALTH EFFECTS

No signs of acute toxicity attributable to this compound were seen in freshwater specied exposed to levels less than 2,000 ug/L. Chronic toxicity studies indicate that the acute to chronic ratio is probably between 5 and 10. Although the bioconcentration factor in bluegills exposed to 2,4-dimethylphenol is 150, residues are probably not a significant hazard for freshwater species. No information on the toxicity of 2,4-dimethylphenol to other wildlife or domestic animals was available (3).

REGULATORY LEVELS AND CRITERIA

National 2,4-dimethylphenol regulatory levels and criteria relative to a risk assessment are established under the Safe Drinking Water Act (SDWA).

SUMMARY OF CRITERIA

USEPA Reference Dose from Oral Exposure (4)	$2.0x10^{-2}$ mg/kg/day
USEPA Reference Concentration from Inhalation Exposure (4)	Not available
USEPA Oral Cancer Potency Factor (4)	Not available
USEPA Inhalation Cancer Potency Factor (4)	Not available
USEPA Weight-of-Evidence (4)	Not available
MCLG (1)	Not available
MCL (1)	Not available

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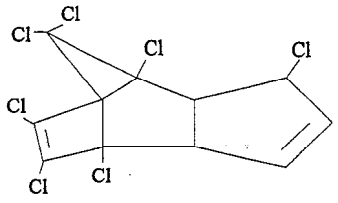
Date of Last Revision: 9/21/93
Revisor: Joy Marshall

HEPTACHLOR

INTRODUCTION

Chemical Name:	Heptachlor
CAS Number:	156-10-5
Synonyms:	2-Chlorochlordene
Molecular Formula:	$C_{10} H_5 Cl_7$
Molecular Weight:	373.35 g/mole
Chemical Structure:	



Heptachlor is a pure white crystalline solid (technical grade is tan). It was used extensively until the 1970s as a broad based insecticide on a wide variety of agricultural crops, especially corn. In addition, it had nonagricultural uses including seed treatment, home and garden uses, and termite control. In 1988, the sale, distribution, and shipment of existing stocks of all heptachlor products was prohibited in the United States. The only commercial use of heptachlor still permitted in the United States is fire ant control in power transformers.

FATE AND TRANSPORT

Log K_{oc} (1):	4.34
Log K_{ow} (1):	5.44
$t_{1/2}$ in fish:	less than 1 day
Henry's Law Constant (1):	1.48×10^{-3} atm-m^3/mole
BCF (1):	217
Solubility:	
In Water (1):	.05 mg/L at 25° C
In Organics (1):	Soluble in most organic solvents
Vapor Pressure (1):	$3E^{-4}$ mm Hg at 20° C

Heptachlor has a low vapor pressure ($3E^{-4}$ mm Hg at 20° C) and a low water solubility (.05 mg/L at 25° C). Its high Henry's Law Constant indicates that it partitions to the atmosphere rapidly, and that volatilization is significant. The soil sorption coefficient (K_{oc} 4.34) is indicative of a very high sorption tendency, suggesting it will adsorb strongly to soil and is not likely to leach into groundwater.

If released into water it adsorbs strongly to suspended and bottom sediment. Volatilization from soil particles to the atmosphere is possible and is an important mechanism of transport from land surfaces.

In the aquatic environment, heptachlor partitions from the water column to sediments and suspended particulate organic matter and is subject to photolysis and biodegradation. Log K_{ow} of the n-octanol/water partition, is an indicator of potential bioaccumulation of a chemical. Due to heptachlors high Log K_{ow}, 5.44, biomagnification in the aquatic food chain is considered to be a major environmental fate process, since it has a high potential for bioaccumulation in aquatic organisms. Heptachlor has also been reported to be taken up by plants via translocation into plants by absorption through the roots.

Heptachlor may undergo direct photolysis and is also susceptible to photosensitized reactions in the atmosphere.

The major environmental fate process for heptachlor in surface water is hydrolyzation. In addition, heptachlor is metabolized by the freshwater microcrustacean, Daphnia magna, to heptachlor epoxide or 1-hydroxychlordene. 1-Hydroxychlordene is then converted to 1-ketochlordene, 1-hyroxy-2,3-epoxychlordene, and their glucosides, sulfates, and other conjugates.

In soil, incubations of heptachlor with a mixed culture of soil microorganisms for 12 weeks, showed the conversion of heptachlor to chlordene, 1-exohydroxychlordene, heptachlor epoxide, and chlordene epoxide. Conversion was about 1% per week during the 12 week test period.

TOXICOKINETICS

Absorption, Distribution, Metabolism, and Excretion

No studies were located regarding the absorption of heptachlor in humans or animals following inhalation exposure.

The health effects of humans known to have consumed dairy products contaminated with heptachlor epoxide were evaluated, and compared to an urban control group. There was no increase in prevalence of abnormal liver function tests in the dairy farm families compared to the urban population. However, this study was limited because exposure level, duration, and frequency of exposure were unknown. Therefore, due to this study and the lack of other research studies, there is insufficient data to make a quantitative estimate for absorption of heptachlor in humans following exposure. In animals, however, heptachlor has been reported to have been absorbed from the gastrointestinal tract of rats and cattle. This has been indicated by the presence of heptachlor or its metabolites in serum, fat, liver, kidney, and muscle of rats and cattle; and by its oral toxicity in rats (LD_{50}, 100-162 mg/kg).

No studies were located regarding the absorption of heptachlor in humans or animals following dermal exposure.

No studies were located regarding the distribution of heptachlor in humans or animals following the inhalation or dermal exposure pathways. However, there is an abundance of information reporting heptachlor in various tissues sampled at autopsy or during surgery, and in serum and milk from

humans after exposure via unknown routes. Autopsies performed of 77 Hawaiian individuals detected high levels of heptachlor epoxide in bone marrow and liver. Other autopsies detected heptachlor epoxide in fat, liver and brain tissue. Heptachlor epoxide has also been detected in human milk at concentrations up to 0.46 parts per million. Unchanged heptachlor has not been detected in human adipose tissue, however, heptachlor epoxide has been detected at levels ranging from 0.0001 to 1.12 part per million.

No studies have been located regarding the metabolism of heptachlor or heptachlor epoxide in humans. However, animal studies have shown that heptachlor undergoes epoxidation to produce heptachlor epoxide, which is more toxic than its parent compound. Heptachlor epoxide is further metabolized into 1-hydroxychlordene, and 1,2-dihydroxychlordene and other unidentified products and excreted in the urine, feces, or through milk production. Heptachlor is formed through the metabolism of chlordane.

No studies were located regarding the excretion of heptachlor in humans or animals following the inhalation or dermal exposure pathways. However, after the oral administration of heptachlor to rats, one study reported that one day after dosing, 36% of the dose had been eliminated in the feces, and by 10 days 62% had been eliminated. Approximately 26% of the parent compound was detected in the feces, the remainder was in the form of metabolites. Elimination of heptachlor epoxide via milk production was found to maximize within 3-7 days in cows that had grazed on pastures immediately following treatment of grasses with heptachlor.

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

There is evidence to suggest that heptachlor can cause adverse cardiovascular effects. Heptachlor has been shown to affect the atherosclerotic process that are involved in both cardiovascular and cerebrovascular diseases. Intermediate and chronic inhalation exposure of humans to mixtures of heptachlor and chlordane has been associated with leukemia and aplstic and hemolytic anemias. Based on animals studies alone, hepatoxicity may be the most sensitive systemic end point for heptachlor and heptachlor epoxide. Animal toxicity included histologic liver damage, increased liver weight, increased levels of serum enzymes, and decreased body weight. In addition, animal studies have indicated that both carbohydrate metabolism and lipid metabolism may be affected by heptachlor and heptachlor epoxide. Affected renal function in humans have been reported after exposure to chlordane and heptachlor. Neurological effects have been reported in human case studies where irritability, salivation, lethargy, dizziness, labored respiration, muscle tremors and convulsions were reported following exposure to chlordane that was from 6-30% heptachlor. Heptachlor was developed to be an insect neurotoxicant, and it is not surprising that the central nervous system would be a primary target for this toxicant.

There is no conclusive evidence that developmental, reproductive and genotoxic effects occur in humans, however, limited animal studies indicate that there may be adverse effects may occurring these areas.

Other systemic effects include, adrenal fibrosis with lipid accumulation in mice; and body weight decrease possibly due to a decrease in food consumption probably due to taste aversion.

The USEPA has developed an oral reference dose (RfD) of $5E^{-4}$ mg/kg-d. However, an inhalation RfD has not been established (2).

Carcinogenic Effects

The USEPA has classified heptachlor as a Group B2 carcinogen - probable human carcinogen, human carcinogenicity data is inadequate. The B2 classifiable status is due to the incidence of benign and malignant liver tumors in mice, in addition to several structurally related compounds are liver tumors. Therefore, the USEPA has established an oral cancer slope factor (CSF) of 4.5 $(mg/kg/day)^{-1}$ for heptachlor(2). An inhalation CSF has not been established for heptachlor (2).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

No data are available for life forms.

Terrestrial and Avian

No data are available for terrestrial and avian life forms.

REGULATORY LEVELS AND CRITERIA

AWQC (3)

Ingestion of water and organisms:	$2.8E^{-4}$ µg/L
Ingestion of organisms only:	$2.9E^{-4}$ µg/L
Maximum Contaminant Level (4):	0.4 µg/L

SUMMARY OF TOXICOLOGICAL INDICES

EPA Carcinogenic Classification (3):

Group B2-classifiable as to probable human carcinogenicity

Oral CSF:	4.5 $(mg/kg-day)^{-1}$
Inhalation CSF:	none

Noncarcinogenic Effects (3):

Oral RfD:	$5E^{-4}$ mg/kg-d
Inhalation RfC:	none

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Date of Last Revision: 11/10/94
Revisor: Rich Hoff

HEXACHLOROBUTADIENE

INTRODUCTION

Hexachlorobutadiene caused an increased incidence of kidney tumors in rats and was found to be mutagenic using the Ames assay. There is equivocal evidence that hexachlorobutadiene increases neonatal mortality. Chronic exposure to low levels of hexachlorobutadiene caused renal toxicity in rats and other studies have shown that exposure can affect the central nervous system and liver. Hexachlorobutadiene is also quite toxic to aquatic organisms.

CAS Number:	87-68-3
Chemical Formula:	$Cl_2C{:}CCl{-}CCl{:}CCl_2$
IUPAC Name:	Hexachloro-1,3-butadiene

Important Synonyms and Trade Names: Dolen, GP-40-66:120, HCBD, perchlorobutadiene, C46

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight:	260.74
Boiling Point:	210 to 220°C
Melting Point:	-19 to -22°C
Specific Gravity:	1.675 at 15.5°C
Solubility in Water:	2 mg/liter at 20°C
Solubility in Organics:	Compatible with numerous resins; soluble in alcohol and ether
Log K_{ow}:	4.8
Vapor Pressure:	0.15 mm Hg at 20°C

FATE AND TRANSPORT

Hexachlorobutadiene (HCBD) is probably rather persistent in the environment. Volatilization and adsorption to organic particulates are apparently important transport processes for HCBD. In soil and sediments, HCBD is bound to organic material. This process acts as a sink for HCBD in the environment. There was no information on the ultimate fate of HCBD in nature in the sources searched.

HUMAN HEALTH EFFECTS

The International Agency for Research on Cancer (IARC 1979) notes that there is limited evidence that hexachlorobutadiene is a carcinogen. Their conclusion is based on one oral feeding study in rats in which the incidence of kidney tumors increased in the animals of both sexes given the highest doses (Kociba, et al., 1977). The results of a spot test of HCBD using the Ames assay were positive. The data on the reproductive toxicity of HCBD are equivocal. One study indicates that neonatal mortality rose following a single, subcutaneous injection of 20 mg/kg body weight to the dam just prior to mating. Another, more recent experiment exposed male and female rats to doses of 0.2, 2, and 20 mg/kg/day for 90 days prior to mating and 15 days during gestation; no toxic effects were noted in the offspring. However, male and female rats given 2 or 20 mg/kg/day of HCBD showed

signs of renal toxicity. The results of a 2-year feeding study in rats confirmed that renal tubular hyperplasia was caused by doses larger than 2 mg/kg/day. Other studies have indicated that HCBD also affects the central nervous system and the liver (Harleman and Seinen 1979). HCBD is a cumulative toxin and is therefore more toxic after chronic exposures. The oral LD_{50} for adult rats is 250 mg/kg, and the LD_{50} for neonatal rats is one-quarter that for the adult animals.

Toxicity to Wildlife and Domestic Animals

Hexachlorobutadiene is very toxic to aquatic organisms, with 96-hour LC_{50} values for goldfish, rainbow trout, fathead minnow, and bluegill ranging from 90 to 330 μg/liter. Its chronic toxicity, as measured in an embryo-larval test in fathead minnows, is 9.3 μg/liter. Invertebrates and saltwater fish were affected at similar levels.

The ingestion of up to 30 ppm of HCBD in their diets (approximately 5-6 mg/kg) had no effect on Japanese quail.

No studies on the toxicity on HCBD to domestic animals were discussed in the literature reviewed.

REGULATORY LEVELS AND CRITERIA

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

EPA Carcinogenic Classification:	C-possible human carcinogen
Oral Cancer Slope Factor (6):	7.8×10^{-2} (mg/kg/day)$^{-1}$
Inhalation Cancer Slope Factor (6):	7.7×10^{-2} (mg/kg/day)$^{-1}$
Oral Reference Dose (4):	2×10^{-4} (mg/kg/day)
ACGIH TLV-TWA:	0.21 mg/m^3 (suspected carcinogen)

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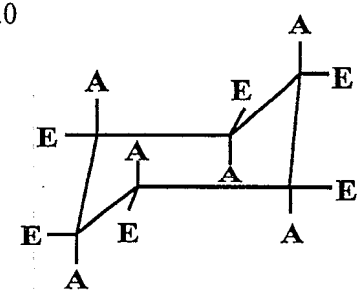
Date of Last Revision: 12/14/94
Revisor: Rich Hoff

alpha-, beta-, delta- and gamma-BHC (HEXACHLOROCYCLOHEXANE)

INTRODUCTION

Chemical Name: alpha, beta, delta, gamma-BHC (hexachlorocyclohexane)
CAS Numbers: 319-84-6, 319-85-7, 319-86-8, 58-89-9
Molecular Formula[1]: $C_6H_6Cl_6$
Molecular Weight[1]: 291.0
Chemical Structure[1]:



Orientation of Cl^- on ring	BHC Isomer
AAEEEE	α (alpha)
EEEEEE	β (beta)
AEEEEE	δ (delta)
AAAEEE	γ (gamma)

Alpha, beta, delta, and gamma-BHC are aliphatic chlorinated hydrocarbon pesticides representing the four major isomers of technical grade hexachlorocyclohexane (HCH). The gamma isomer (lindane) is generally the most toxicologically active[2]. The BHCs are fairly persistent in environmental media. The primary environmental fate of BHCs appears to be adsorption to soils and sediments and subsequent biodegradation.

FATE AND TRANSPORT

Log K_{oc}:	3.58, 3.58, 3.82, 3.58
Log K_{ow}:	3.89, 3.89, 4.15, 3.89
Henry's Law Constant (@ 20°C):	6×10^{-6}, 4.5×10^{-7}, 1.7×10^{-5}, 7.8×10^{-6} (@ 25°C)
Vapor Pressure (@ 20°C)[3]:	2.5×10^{-5}, 2.8×10^{-7}, 1.7×10^{-5}, 1.6×10^{-4} (@ 25°C)
Water Solubility (mg/L @ 25°C)[3]:	1.63, 0.24, 31.4, 7.8
BCF[4]:	100-500

BHCs, by virtue of their K_{ow} and K_{oc} values tend to adsorb to soils and sediments with subsequent biodegradation. Biodegradation of BHCs yields chemicals such as pentachlorocyclohexane, tetrachlorobenzene, trichlorophenol, and, therefore, may not result in substantial detoxification. Gamma-BHC (lindane) has been shown to be rather persistent when applied to soil with as much as 10 percent remaining after 10 years[4].

PHARMACOKINETICS

Workers exposed to γ-HCH via inhalation of workplace air have exhibited adverse health effects such as hematological abnormalities and neurological effects. From this it can be inferred that humans absorb γ-HCH vapor or dusts via inhalation. The α-, β-, γ- and δ-isomers have been detected in the blood serum, adipose tissue and semen of individuals who have been occupationally and environmentally exposed to these chemicals via inhalation, demonstrating that absorption takes place. HCH is absorbed in humans upon oral exposure, as demonstrated by acute accidental poisoning cases wherein high blood levels have been detected. Gastrointestinal absorption has also been demonstrated in animals. Eight minutes after the administration of radiolabelled γ-HCH by stomach tube in fasting mice, a maximum of 17 percent of the administered dose was found in the gastrointestinal tract. In rats, absorption of technical-grade HCH has been quantified. Four days after the oral administration of a single dose, 95.8 percent of technical-grade HCH was absorbed in rats. Absorption of technical-grade HCH administered in the diet for 14 days was 94.9 percent in rats; the average absorption of these α-, β-, γ- and δ-isomers were 97.4 percent, 90.7 percent, 99.4 percent and 91.9 percent, respectively. Gamma-HCH is readily absorbed through the skin. Following whole body application of an anti-scabies lotion (γ-HCH is used to treat scabies) to scabies patients and volunteers, maximal blood levels in both scabies patients and volunteers were reported within 4-6 hours following application. When γ-HCH is applied topically to the forearm, 9 percent of the applied dose is absorbed rapidly, with maximum absorption occurring 2-3 days after application [5].

Alpha, β-, γ-HCH are distributed to the blood and adipose tissue upon inhalation exposure in humans. Gamma-HCH is distributed to the central nervous system (CNS) and the cerebral spinal fluid in humans orally exposed to γ-HCH. In laboratory rats exposed for 5, 10 or 15 days, γ-HCH and β-HCH are primarily stored in the fat. The overall distribution of γ-HCH was in the following order: fat>brain>kidney>muscle>lungs>heart>spleen>liver>blood. The overall distribution for the β-isomer was: fat>kidney>lungs>liver>muscle>heart>spleen>brain>blood. The distribution of α-HCH in rats dosed by gavage was fat>kidney>liver>brain>blood. Upon dermal exposure in humans γ-HCH is distributed to the brain as indicated in a case report of an autopsy of an infant treated with a whole body application of a 1 percent γ-HCH lotion.

Additionally, γ-HCH can be transferred to the fetus from pregnant woman through the placenta and to neonates through the milk. Males environmentally exposed to HCH accumulate γ-HCH and HCH isomers in adipose tissue and to a lesser extent in the testes or semen [5].

The metabolism of γ-HCH in humans is similar to that in mice and rats. Chlorophenols and chlorobenzenes are the primary metabolites of γ-HCH. The primary urinary metabolites of workers involved in γ-HCH production were chlorophenols. Almost 57.7 percent of the γ-HCH metabolites identified in the urine of workers during the last two hours of their shifts were 2, 3, 5-, 2, 4, 6- and 2, 4, 5 trichlorophenols. Other urinary metabolites detected were trichlorophenols, dichlorophenols, tetrachlorophenols and dihydroxychlorobenzenes. Following occupational exposure, pentachlorophenol has also been identified as a metabolite. Gamma-HCH is rapidly transformed by hepatic enzymes to form chlorophenols, chlorobenzene, chlorocyclohexanes, chlorocyclohexanols and conjugates of mercapturic acid, glucuronide and sulfate in animals. In rats, the major urinary metabolites formed following intermediate oral exposure to α- or β-HCH, included tri- and tetrachlorophenols. In kidney tissue pentachlorocyclohexane was also identified. The P450 oxidative system appears to be responsible for the detoxification of γ-HCH [5].

Humans excrete γ-HCH and its metabolites in urine, milk and semen upon inhalation exposure. In the urine from humans occupationally exposed to HCH, chromatographic analyses revealed the presence of chlorinated phenols and all isomers of di-, tri- and tetrachlorophenol. In mice, the major route of elimination is via the urine following intermediate and chronic feeding of γ-HCH. Very little γ-HCH is eliminated in expired air and in the feces following oral administration in rodents. Very little HCH is excreted unaltered. In humans, nonmetabolized γ-HCH was excreted by healthy volunteers and scabies patients upon an acute, whole body application of a 0.3 percent γ-HCH emulsion [5].

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

BHCs can cause convulsions in humans exposed occupationally even by the topical (dermal) route. Nervousness, irritability, insomnia, vertigo, amblyopia, stupor and coma have been observed following excessive cutaneous application. BHCs can sensitize the heart to arrhythmias. Technical grade BHC is an irritant to the skin, eyes, and mucosa. Fatal cases of aplastic anemia have resulted from prolonged exposure to vaporized gamma-BHC[6].

The alpha and gamma isomers are CNS stimulants, but the beta and delta isomers are depressants.

Twenty male and female Wistar rats were administered 0, 0.2, 0.8, 4, 20, or 100 ppm gamma-BHC (lindane) (99.85 percent) in the diet. After 12 weeks, 15 animals were sacrificed. The remaining rats were fed the control diet for an additional 6 weeks before sacrifice. No treatment-related effects were noted on mortality, hematology, clinical chemistry, or urinalysis. Rats receiving 20 and 100 ppm lindane were observed to have greater-than-control incidence of the following: liver hypertrophy, kidney tubular degeneration, hyaline droplets, tubular distension, interstitial nephritis, and basophilic tubules. Since these effects were mild or rare in animals receiving 4 ppm, this represents a NOAEL. The reviewers of the study calculated the dose to be 0.29 mg/kg/day for males and 0.33 mg/kg/day for females, based on measured food intake[7].

In a 2-year feeding study, 10 Wistar rats/sex/group were exposed to 5, 10, 50, 100, 400, 800, or 1,600 ppm lindane. Slight liver and kidney damage and increased liver weights were noted at the 100 ppm level. If a food intake equal to 5 percent body weight is assumed, a NOAEL of 2.5 mg/kg bw/day (50 ppm) can be determined from this assay. In a two-year bioassay, four beagle dogs/sex/group were administered 0, 25, 50, or 100 ppm gamma-BHC in the diet. Treatment-related effects noted in the animals of the 100 ppm group were increased serum alkaline phosphatase and enlarged dark friable livers. A NOAEL was determined to be 50 ppm for gamma-BHC (1.6 mg/kg bw/day)[7].

Carcinogenic Effects

Dietary alpha-BHC has been shown to cause increased incidences of liver tumors in five mouse strains and in Wistar rats.

Groups of 20 to 40 male dd mice were treated with 100, 250, or 500 ppm alpha-HCH in the diet for 24 weeks. Liver nodules and hepatocelluar carcinomas were observed in the two upper dose groups. In a subsequent study, male DDY mice were maintained on a diet containing 500 ppm alpha-HCH for 16, 24, or 36 weeks, respectively. Incidence of liver tumors increased with continuous alpha-BHC administration. Incidence decreased, however, with recovery time. At 24 weeks most lesions observed were nodules, but by 60 or 72 weeks the tumors were primarily hepatocelluar carcinomas[7].

Schulte-Hermann and Parzefall noted an increased incidence of hepatic nodules and hepatocellular carcinomas in female Wistar rats treated with approximately 20 mg/kg/day alpha-BHC for their lifetime. Male Wistar rats were fed alpha-BHC in the diet at 500, 1000, or 1500 ppm for 24, 48, or 72 weeks. Liver nodules and carcinomas were observed in rats fed the two highest doses for 72 weeks. Both males and females of the dd strain responded in a dose-dependent fashion with liver nodules and hepatomas when fed 100, 300, or 600 ppm dietary alpha-BHC for 32 weeks, followed by 5 to 6 weeks basal diet. In a feeding study using male ICR-JCL mice, alpha-BHC produced hepatomas in 100 percent of the animals. Liver tumors have been observed as early as 24 to 26 weeks[7].

Dietary beta-BHC produced positive or marginally positive tumorigenic responses, characterized as benign hepatomas or hepatocellular carcinomas, have been observed in two strains of mice. The studies are limited in that small numbers of animals were used, no dose-response data are available, not all of the animals were examined histologically, or the duration of exposure was less than lifetime.

Thorpe and Walker fed 30 each male and female CF1 mice dietary beta-BHC at 200 ppm for 110 weeks. This resulted in 12 percent mortality of males and 25 percent of females during the first 3 months. A significantly increased incidence of liver tumors was observed in treated males and females.

No statistically significant evidence of increased tumor incidence as a consequence of beta-BHC feeding was seen in several small (5 to 20/group) studies with male and female dd mice fed 0 to 600 ppm for 24 to 32 weeks or in male Wistar rats fed 0 to 1000 ppm for >72 weeks.

Goto et al. maintained male ICR-JCL mice on a diet containing 600 ppm beta-BHC for 26 weeks. Relative liver weight was increased in the treated animals, and there was histologic evidence of benign neoplasms at an unspecified incidence.

USEPA has classified alpha-BHC as a B2; probable human carcinogen on the strength of laboratory dietary mouse studies. USEPA has also classified beta-BHC as a C; possible human carcinogen as a result of dietary mouse studies[7].

Ecological Effects

Gamma-BHC (lindane) is responsible for the effectiveness of technical hexachlorocyclohexane as an insecticide and is generally more toxic than the other isomers or technical BHC. In fact, the presence of the other BHC isomers decreases the toxicity of lindane to aquatic organisms, either by an antagonistic effect or by decreasing the chemical's solubility. Therefore, the toxicity of lindane and other BHCs are considered separately.

Lindane is acutely toxic to freshwater fish with LC_{50} values ranging from 2 μg/L to 141 μg/L; and to saltwater fish at levels of 7.3 to 104 μg/L. Lindane was acutely toxic to the pink shrimp at 0.17 μg/L. Acute-chronic ratios for lindane ranged from 7.5 to 63, and, therefore, the Final Chronic Value for the protection of freshwater species was determined to be 0.08 μg/L. Aquatic organisms appear to bioconcentrate between 100 and 500 times the steady-state concentration of lindane in the water.

Technical BHC was much less toxic than lindane, with acute toxicity ranging from 100 μg/L to 15,000 μg;/L for freshwater fish. Data on saltwater species also indicated that the technical compound was less acutely toxic. No information was available on the chronic toxicity of BHC. A bioconcentration factor was not reported but is probably similar to that for lindane[8].

No studies on the toxicity of the BHC isomers to terrestrial or domestic animals was found in the literature reviewed. However, voles at Love Canal that had decreased lifespans and reproductive ability had high levels of lindane in their livers[9].

REGULATORY LEVELS AND CRITERIA

ACGIH TLV-TWA:	0.5 mg/m^3 (lindane)
OSHA PEL-TWA[10]:	0.5 mg/m^3 (lindane)
Maximum Contaminant Level (MCL)[11]:	0.0002 mg/L (lindane)
MCLG[11]:	0.0002 mg/L (lindane)
Health Advisories[12,13]:	

Lindane	
10 day child	1.0 mg/L
1 day child	1.0 mg/L
Longer term child:	0.033 mg/L
Longer term adult:	0.12 mg/L
Lifetime:	0.0002 mg/L

Ambient Water Quality Criteria[13]

Water and Fish Consumption:	0.0092 μg/L (alpha), 0.0163 μg/L (beta), 0.0186 μg/L gamma
Fish Consumption Only:	0.031 μg/L (alpha), 0.0547 μg/L (beta), 0.0625 μg/L gamma
Acute Freshwater LEC:	100 μg/L (alpha), 100 μg/L (beta), 200 μg/L gamma
Acute Marine LEC:	0.031 μg/L (alpha), 0.0547 μg/L (beta), 0.0625 μg/L gamma

Chronic Freshwater LEC:	0.08 μg/L (gamma)
Chronic Marine LEC:	None

SUMMARY OF TOXICOLOGICAL INDICES

USEPA Carcinogenic Classification:	B2 (alpha), C (beta), D (delta), C (gamma)
Oral RfD[7]:	$3x10^{-4}$ mg/kg/day (gamma)
Inhalation RfC[7]:	Pending
Oral Cancer Slope Factor[7,14]:	6.3 mg/kg/day^{-1} (alpha)
	1.8 mg/kg/day^{-1} (beta)
	1.3 mg/kg/day^{-1} (gamma)
Inhalation Cancer Slope Factor[7]:	1.79 mg/kg/day^{-1} (beta)
Inhalation Cancer Slope Factor[7]:	6.3 mg/kg/day^{-1} (alpha)
Drinking Water Unit Risk[7]:	1.8 x 10^{-4} per (μg/L)

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LEAD

INTRODUCTION

Chemical Name:	Lead
CAS Number:	7439-92-1
Molecular Formula:	Pb
Molecular Weight:	207 g/mole
Chemical Structure:	Pb

Elemental lead is a heavy, ductile, bluish-gray solid at ambient conditions. It is used widely in industry because of its softness, resistance to corrosion and radiation, and high density. Lead is also used as a paint pigment, in solder, and in storage batteries.

FATE AND TRANSPORT

BCF:	49 (1)
Degradation Products:	None
Solubility:	Lead is insoluble in water and organic solvents. It does dissolve in dilute nitric acid (2), and hot, concentrated sulfuric acid (3).
Specific Gravity (2):	11.35 @ 20°C

Lead and lead compounds can be present in air, water, and soil and are extremely persistent in water and soil (4). Metallic lead and common lead minerals are insoluble in water while manufactured alkyl lead compounds are water soluble. A major transport process for inorganic and organic lead compounds is atmospheric dispersion as particulate matter. Lead is removed from air by either wet or dry deposition. Photolysis of atmospheric organic lead compounds occurs rapidly (3). The average residence time of atmospheric lead is 7 to 30 days (4).

Natural lead compounds are not mobile in normal surface and groundwater because lead leached from ore is adsorbed by ferric hydroxide. It also readily combines with hydroxide, carbonate, and sulfate ions to form insoluble compounds. These compounds precipitate and settle in the bed sediment. Lead is not volatile, therefore, volatilization is not an important transport process from aquatic environments (3).

Sorption is a dominant effect on the distribution of lead in soil. Lead readily adsorbs to inorganic solids, organic material and hydrous iron and manganese oxides. Because of its affinity for other materials and its solubility characteristics, the mobility of lead in soil is low. (3). Most lead is retained in soil and not transported via leaching or runoff to surface water (4).

Lead is not readily taken up by plants. Consequently, its availability to terrestrial life forms is limited (3). Lead does not appear to significantly bioaccumulate in most fish (4). Microcosm studies indicate that lead is not biomagnified through the food chain (3).

PHARMACOKINETICS

Lead primarily enters the body by inhalation and ingestion. For all practical purposes, it will not enter the body by dermal contact. Approximately 30-50 percent of all inhaled lead particulate is deposited in the respiratory tract. Almost all of the lead present will be absorbed by the lungs. The primary site of absorption by ingestion in children is the gastrointestinal tract. Fifty percent of all dietary lead ingested by children is absorbed, whereas only 8-15 percent is absorbed by adults. Studies have also demonstrated that transplacental transfer of lead is possible.

Lead is not homogeneously distributed upon entering the body. It concentrates in the following three organs: bone, blood, and soft tissue. The lead in each of these compartments has a different rate of intercompartmental movement and residence time (4).

Inorganic lead is not metabolized in the body; rather it is absorbed, distributed and excreted. However, organic alkyl lead is metabolized in the liver via an oxidative dealkylation reaction which is catalyzed by cytochrome P-450 in animals (4).

Lead that is not absorbed by the respiratory or gastrointestinal tract is excreted in the feces. Blood lead not retained by the body is eliminated by the kidney or bile (4).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Numerous studies have been conducted on the toxic effects of lead. The data present dose-effect relationships and are expressed in terms of internal exposure measured in blood lead levels (4).

Lead affects various systems of the body. The endpoints elicited at low level exposure are neurobehavioral impairment, growth retardation in children, and hypertension in middle-aged men. High exposure levels produce encephalopathy, gastrointestinal effects, anemia, nephropathy, and electrocardiographic abnormalities (4).

Lead affects the hematopoietic system by interfering with heme biosynthesis and ferrochelatase. This interference results in a reduction of the hemoglobin concentration in blood and an increase in erythrocyte (red blood cell) destruction. The combination of these two effects produce hypochromic, normocytic anemia with associated reticulocytosis. The impairment of heme synthesis has a far ranging impact that is not limited to the hematopoietic system.

Lead exposure also affects the central nervous system. Overt neurological signs have been documented in adults with blood lead levels ranging as low as 40 to 60 μg/dl. Encephalopathy can occur at blood lead levels of 100 to 200 μg/dl and 80 to 100 μg/dl for adults and children, respectively. As indicated by these levels, children are much more sensitive to neurological effects of lead. Death or irreversible health effects may occur as a result of central nervous system impairment (4).

The cardiovascular system is also impacted by lead exposure. Common effects from high level exposure include cardiac lesions and electrocardiographic abnormalities. Hypertension has been

clearly related to lead exposure. Studies suggest that high blood pressure resulting from lead exposure may be mediated through effects on the kidney (4).

A direct relationship has been drawn between blood lead level and children's height, weight, and chest circumference. The strongest relationship was drawn with height. As the blood lead level increases the child's growth is retarded (4).

Exposure to lead has significantly effected the human reproductive system. Because lead accumulates in the bones and is sporadically released into the blood, exposure prior to pregnancy may affect the fetus. Blood lead reaches the fetus by crossing through the placental barrier. It was observed in animals that exposure to lead by non-natural routes (e.g., intravenous or intraperitoneal injection) resulted in malformations of the fetus (4).

In the body, lead can interact with other chemicals. Calcium and phosphorus reduce the amount of lead taken up by the body while zinc helps to reduce the toxic effects of lead. Cadmium increases the toxic effects of lead; and lead increases the toxic effects of mercury. Due to the effect of lead on the hemapoietic system, iron deficiency increases as blood lead increases (4).

Derivation of Oral Reference Dose for Lead

In a review of available toxicological data on the potentially adverse effects associated with various blood lead (PbB) levels in adults and children, Marcus (1986) has concluded that the effects on the enzymes of heme synthesis necessary for red blood cell formation occur at low PbB levels of about 10 micrograms per decaliter (μg/dL). Further, the author noted that neurotoxicity in children begins to appear at 15 to 20 μg/L and at 25 to 30 μg/dL in adults. According to the author, the data suggests, "that PbB values of 15 μg/dL should not be exceeded in children and values of 25 μg/dL should not be exceeded in adults".

In order to protect the fetus, it is necessary to set the PbB level in adults at 15 μg/dL since studies have indicated that the ratio of fetal/maternal PbB values can be approximated at a ratio of one to one (Marcus, 1986). It should be noted that this position is consistent with current EPA's conclusions that "PbB levels of 10 to 15 μg/dL constitute an appropriate range of concern for health effects that warrant avoidance" (Federal Register 50, No. 10, 26460-26550, June 7, 1991).

The acceptable daily intake (ADI) of lead via oral ingestion by adults has been determined to be 48 μg/day (Marcus, 1986). This value is equivalent to a PbB level (15 μg/dL) at which no adverse effects are observed to occur in humans.

Carcinogenic Effects

The EPA has stated that "little can be concluded from available epidemiological studies" concerning the carcinogenic potential of lead (4). However, the carcinogenicity of lead salts (primarily phosphates and acetates) administered via injection or the oral route has been demonstrated in rats and mice in several studies. In most of the investigations, the carcinogenic response has been demonstrated only at the highest dose. Although the EPA has stated that animal data are sufficient to conclude carcinogenicity in animals, the Agency has further stated that available toxicity data on metallic lead and lead compounds are inadequate for quantitative risk assessment (6).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Freshwater species are more sensitive to lead contamination in soft water than hard water for both acute and chronic exposure. The ambient water quality criteria for aquatic life are as follows (5):

Four-day average concentration that should not be exceeded more than one time per year.

Freshwater	Salt Water
$e^{(1.273 (\ln (\text{hardness}))-4.705)}$ µg/L or 3.2 µg/L	8.5 µg/L

One-hour average concentration that should not be exceeded more than one time per year.

Freshwater	Salt Water
$e^{(1.273 (\ln (\text{hardness}))-4.460)}$ µg/L or 82 µg/L	220 µg/L

The BCF for saltwater species ranges from 17.5 to 2570 µg/L (3).

Terrestrial

Lead occurs in the tissues of many wildlife species. Lead poisoning has also been reported for a variety of domestic animals including cattle, horses, dogs, and cats. Cattle appear to experience lead poisoning more often because of their indiscriminate eating habits (3).

REGULATORY LEVELS AND CRITERIA

Ambient Air Quality Standard (4):	1.5 µg/m^3
OSHA PEL-TWA (4):	50 µg/m^3
MCL (4):	0.015 mg/L (Action Level)
MCLG (4):	0 mg/L
Reportable Quantity (metallic) (4):	1 lb
ACGIH TLV-TWA (4):	0.15 mg/m^3 (inorganic lead, dust, and fumes)
	0.15 mg/m^3 (lead arsenate)
	0.05 mg/m^3 (lead chromate)
Ambient Water Quality Criterion:	50 µg/L

SUMMARY OF CRITERIA

EPA Carcinogenic Classification (4):	Group B2-probable human carcinogen
RfD (oral) (1)	Not Available
Cancer Slope Factor (oral, inhalation)	Not Available

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MANGANESE

INTRODUCTION

Chemical Name:	Manganese
CAS Number:	7439-96-5
Molecular Formula:	Mn
Molecular Weight:	54.94 g/mole

Manganese is a brittle silvery metal which usually occurs as a complex with other metals such as iron. Manganese and its compounds are used in the making of steel alloys, dry-cell batteries, electrical coils, and other metallic fabrication applications. Other manganese uses include use as an oxidizing agent and as a food additive (3,4).

FATE AND TRANSPORT

Solubility:	
In Water:	Decomposes to ionic forms (1)
In Organics:	Readily dissolves in dilute mineral acids (4)
Specific Gravity:	7.20 (1)

Manganese can occur in soil, water, or air. Because it is an element, manganese cannot be degraded by environmental processes. However, it may transform from one manganese compound to another. While manganese can be transported in dusts or in water, the main source of routine manganese exposure is through ingestion of food. Vegetables, the germinal portions of grains, fruits, nuts, tea, and some spices are rich in manganese (3).

In the soil, the concentration and chemical form in which manganese can occur is affected by pH, cation exchange capacity, drainage, and other factors. Lowered pH and reducing conditions tend to favor solubility and hence, the mobility of manganese. Manganese often occurs at higher concentrations in the bottom of stratified lakes as a result of its release from bottom sediments as manganous ion under reducing conditions (1).

The presence of high concentrations of chlorides, nitrates, and sulfates may also increase manganese solubility. Under these conditions, manganese is more easily taken up by plants. Also, soils with limited cation exchange capacity have a poor ability to bind and retain manganese(1).

Atmospheric transport of manganese fumes or dusts is also possible. These materials can be returned to the earth by wet or dry deposition (1).

PHARMACOKINETICS

Manganese can enter the body through the ingestion of manganese-bearing food or through the inhalation of air containing manganese fumes or dusts. It is considered to be an essential element and cofactor in a number of enzymatic reactions with daily intakes ranging from two to nine

milligrams. Gastrointestinal absorption of the metal is less than five percent. Manganese is transported by a plasma-bound β1-globulin protein (probably transferrin). It is widely distributed throughout the body, concentrating in cells rich in mitochondria. Consequently, tissues such as the pancreas, liver, kidney, and intestines, which contain high numbers of these organelles, tend to be deposits for manganese (3).

The biological half-life for manganese in the body is 37 days. Manganese readily crosses the blood-brain barrier, with its half-life in the brain being somewhat longer than in the body as a whole (3).

Manganese is eliminated in the bile and is resorbed through an enterohepatic pathway through the intestine. The principal means of elimination is through the feces (3).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Noncarcinogenic effects in humans are most pronounced following inhalation exposure. The types of effects observed fall into two categories, depending upon the severity and duration of the exposure (3). The first type of effect observed, manganese pneunmonitis, is the result of acute exposure. Individuals acutely exposed exhibit a lung condition characterized by pathologic changes including epithelial necrosis, followed by mononuclear proliferation (3). The second, and more serious effect observed following chronic manganese exposure, is characterized by neurologic symptoms. Chronic manganese poisoning is characterized by psychiatric disorders manifested as irritability, difficulty walking, speech disturbances and compulsive behaviors. Long-term etiology includes development of a mask-like face and a Parkinsonian-like syndrome (3).

An inhalation reference concentration (RfC) of 5×10^{-5} mg/m^3 (1.43×10^{-5} mg/kg/day) for manganese has been developed (2). The basis for this value is increased prevalence of respiratory symptoms and psychomotor disturbances. Potential limitations to this RfC include the inability to calculate a true absorbed dose as a result of variable particle size and dosimetry effects.

Manganese is considered to be one of the least toxic of the trace elements via the ingestion exposure route. Certain sub-populations, such as the elderly, children, pregnant women, and iron-deficient individuals, may have in increased potential to accumulate excess manganese. However, toxicity from ingested manganese is rarely observed (2).

EPA has established an oral Reference Dose (RfD) for manganese of 5.0×10^{-3} mg/kg/day, based on central nervous system effects (2). This oral RfD is based on total dietary intake, and may not be valid if the intake is to occur from drinking water alone, as manganese in drinking water is more bioavailable than manganese from food.

Carcinogenic Effects

There are no epidemiological studies to suggest that manganese or its compounds are carcinogenic. Manganese is classified as a Group "D" carcinogen, indicating that it is not classifiable as a human carcinogen. The basis for this determination is the lack of existing studies to assess this material (2).

The evidence for rating manganese as a carcinogen in animals is considered to be inadequate. Most studies that have shown some evidence of carcinogenicity have failed to demonstrate a dose-response relationship (2).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Adequate data to develop ambient water quality criteria are not available at this time. A 48-hour LC_{50} value of 16 mg/L of manganese is reported for embryos of the oyster Crassostrea virginica. For the softshell clam, Mya arenaria, a 168-hour LC_{50} value of 300 mg/L is reported (1).

Terrestrial and Avian

Adequate data for characterization of the toxicity of manganese to wildlife or domestic animals are not available (1).

REGULATORY LEVELS AND CRITERIA

OSHA Ceiling Level (1):	5 mg/m^3
ACGIH TLV-TWA (1):	1 mg/m^3 (Fume)
STEL (1):	3 mg/m^3 (Fume)
TLV-TWA (1):	5 mg/m^3 (Dust and Compounds)

SUMMARY OF CRITERIA

EPA Carcinogenic Classification (2)	Group D-Not Classifiable
Oral RfD (water) (2)	5.00×10^{-3} mg/kg/day
Inhalation RfD (2)	1.43×10^{-5} mg/kg/day
Oral (food) RfD (2):	1.4×10^{-1} mg/kg/day
Secondary Maximum Containment Level (2)	0.05 mg/L
NOAEL (oral diet) (2):	0.14 mg/kg/day
NOAEL (oral drinking water) (2):	0.005 mg/kg/day
LOAEL (inhalation) (2):	0.05 mg/cu m
AWQC (5):	
Water and Organisms:	50 μg/L
Organisms Only:	100 μg/L

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Revisor: Rich Hoff

MERCURY

INTRODUCTION

Chemical Name:	Mercury
CAS Number:	7439-97-6
Molecular Formula:	Hg
Molecular Weight:	200.59 g/mole
Chemical Structure:	Hg

Mercury is a silvery, heavy liquid with valences of +1, and +2. Mercury exists as insoluble elemental mercury, organic species, and inorganic species. Solubility depends upon the reduction-oxidation potential and the pH of the environment (2).

Common uses for mercury are amalgams, catalysts, electrical apparati, instruments such as thermometers and barometers, and neutron absorbers in nuclear power plants (2).

FATE AND TRANSPORT

Solubility (4):	81.3
In Water:	µg/L at 30° C; some salts and organic compounds are soluble (1).
Vapor Pressure(4):	0.0012mm Hg at 20° C (1)
Specific Gravity(4):	13.5939 at 20° C (1)

Mercury appears in ambient air, water, soil, and foodstuffs. Because mercury volatilizes from aquatic and terrestrial sources, the atmosphere becomes a reservoir for the element and its compounds. Wet and dry deposition processes return mercury to the land and surface, where it is absorbed in the soil and water surfaces. The sorption rate is related to the organic content of the soil. Since inorganic mercury is sorbed to particulate matter more readily than it is desorbed, sediments become repositories for inorganic forms of the compound. Although leaching is an insignificant transport process, mobilization from particulates can occur through biotic and abiotic oxidation and reduction to elemental mercury and bioconversion to volatile organic forms (1).

The transformation processes of mercury in the atmosphere, water, and soil are biotransformation and bioaccumulation. The primary transformation process is photolysis of organomercurials (1).

PHARMACOKINETICS

Inorganic mercury is absorbed rapidly by the lungs in humans. One study showed that 80 percent of inhaled elemental mercury vapor is retained in human tissues (4). Oral absorption of inorganic mercury is estimated to be about 10 percent while organic mercury is readily absorbed in humans (approximately 95 percent) (4). There are few dermal absorption studies with inorganic and organic mercury, but dermal absorption is known to occur with both types of mercury (4).

Inorganic mercury in body fluids is highly diffusible and lipophilic and, therefore, distributes to all tissues in the body. The highest levels of inorganic mercury occur in the kidney, while lower levels are found in the chest area and lung. Animal inhalation studies have shown mercury to concentrate in the kidney, intestinal mucosa, epithelial layers of the skin, salivary and sweat glands, pancreas, testes and prostate (4).

Organic mercury (i.e., methyl mercury) distributes readily to all tissues in the body following gastrointestinal tract absorption. It can cross diffusion barriers and penetrate membranes easily and, therefore, distributes uniformly in the body; the highest levels being found in the kidney. Organic mercury can easily cross over the placental barrier, and is secreted in breast milk in the inorganic form. Methyl mercury also tends to accumulate in the hair (4).

Inhaled inorganic mercury undergoes rapid oxidation to its divalent form primarily in the red blood cells, and in the lung. Rat studies indicate that inorganic mercury is oxidized in the liver, while other studies indicate that divalent mercury is reduced by mammalian tissue to elemental mercury after its oxidation (4).

Organic mercury (i.e., methyl mercury) is converted to an inorganic form before it enters the oxidation-reduction cycle. Phenyl mercury also metabolizes into inorganic mercury (4).

The urine and feces are the main excretory pathways of organic and inorganic mercury in humans. The fecal pathway is the primary excretory route for organic mercury. In humans, all mercury in the feces is in its inorganic form after organic mercury exposure. Minor amounts of mercury are excreted through saliva, bile and sweat (4).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Irreversible damage to the brain, kidneys, or developing fetus are effects of long-term exposure to mercury (1). In humans, inorganic mercury which is inhaled causes damage to the brain. If inorganic mercury is consumed through water or food, the effect will be focused in the kidneys. Adverse affects of inorganic mercury are characterized by tremors, memory loss, and kidney disease (1).

The EPA is currently reviewing the inhalation and oral RfDs for mercury.

Carcinogenic Effects

The EPA has classified mercury as a Group D carcinogen - not classifiable as to human carcinogenicity. No human data are available and animal studies are inadequate to determine carcinogenicity.

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Freshwater plants exhibit a wide range of sensitivity to mercury, however, the most sensitive plant is less sensitive than the most sensitive freshwater animal.

Fish tend to be more resistant to mercury than mollusks and crustaceans. Aquatic organisms should not be adversely affected if the following average concentrations are not exceeded more than once every three years on the average.

Freshwater organisms:

4-day average: 0.012 µg/L (5)
1-hour average: 2.4 µg/L (5)

Marine organisms:

4-day average: 0.025 µg/L (5)
1-hour average: 2.1 µg/L (5)

Terrestrial and Avian

No data are available pertaining to the effects of mercury on terrestrial and avian life forms (4).

SUMMARY OF REGULATORY LEVELS AND CRITERIA

Oral RfD (6)	3.0×10^{-4} mg/kg/day (inorganic)
Inhalation RfD (6)	8.57×10^{-5} mg/kg/day (inorganic)
EPA Carcinogen Classification (3)	Group-D Not classified as a human carcinogen
AWQC (human)(4)	0.144 µg/L
MCL (4)	2 µg/L
OSHA Ceiling Level(7)	1 mg/10 m^3 (inorganic and vapor)

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Date of Last Revision: 11/10/94
Revisor: Rich Hoff

METHYL CHLORIDE

INTRODUCTION

Methyl chloride is carcinogenic in male mice, causing tumors of the kidney and liver. It was found to be mutagenic using the Ames assay. methyl chloride has also been shown to be teratogenic; it produces heart defects in the offspring of exposed mice. Exposure to high concentrations adversely affects the central nervous system, kidney, and liver in humans.

CAS Number: 74-87-3
Chemical Formula: CH_3Cl
IUPAC Name: Chloromethane
Important Synonyms and Trade Names: Chloromethane, monochloromethane

CHEMICAL AND PHYSICAL PROPERTIES

Molecular Weight: 50.49
Boiling Point: -23.7°C
Melting Point: 97°C
Specific Gravity: 0.9159 at 20°C
Solubility in Water: 6,450 to 7,250 mg/liter at 20°C
Solubility in Organics: Miscible with chloroform, ether, and glacial acetic acid; soluble in alcohol
Log K_{ow}: 0.91

FATE AND TRANSPORT

Methyl chloride is a gas at normal environmental temperatures and therefore is unlikely to remain in soil or water. Experimental studies have found the half-life of methyl chloride in agitated water to be 27 minutes. Although this finding may not be directly applicable to natural waters, it does suggest rapid loss of the compound from water. Sorption of methyl chloride to soil or sediment has not been studied; however, its relatively low log octanol/water partition coefficient suggests (log K_{ow}) that partition occurs primarily into air or water.

The major route of environmental degradation of methyl chloride is probably through oxidation in the troposphere. At this level of the atmosphere, the methyl chloride molecule is attacked by hydroxyl radicals via the mechanism of hydrogen abstraction. The primary product is formyl chloride.

HUMAN HEALTH EFFECTS

Methyl chloride was found to be carcinogenic in male mice exposed to the compound via inhalation for a 2-year period. A significantly increased incidence of benign and malignant kidney tumors was found in animals exposed to 2,100 mg/m^3. An increased incidence of hepatocellular carcinomas that was marginally significant was also found using an actuarial analysis of the data. Negative results for carcinogenicity for female mice and male and female rats were obtained in the same study.

Methyl chloride has been found to be mutagenic using the Ames assay, with and without a metabolic activating system. Methyl chloride has also been shown to be teratogenic in mice, causing heart defects in fetuses exposed in utero at an airborne concentration of 1,050 mg/m^3 on gestation days 6 to 17.

Methyl chloride is not considered to be highly toxic. Repeated or prolonged human exposure to sufficient concentrations (greater than 100 mg/m^3) can result in central nervous system (CNS) effects including blurred vision, headache, nausea, loss of coordination, and personality changes. Renal and hepatic toxicity have also been reported in humans. Animal studies show CNS effects and binding to sulfhydryl-containing cellular macromolecules. This latter effect interferes with metabolism and is probably responsible for the observed tissue toxicity.

Toxicity to Wildlife and Domestic Animals

The only information available on the effects of methyl chloride in wildlife is an acute study on the bluegill that reported an LC_{50} value of 500 mg/liter for this species. Data on the other chlorinated methanes indicate that aquatic toxicity declines with decreased chlorination. Thus, methyl chloride should be less toxic than chloroform or carbon tetrachloride, neither of which had any effect on Daphnia magna or the fathead minnow, respectively, during chronic exposure to 3,400 μg/liter. No information on the toxicity of methyl chloride to terrestrial wildlife or domestic animals was found in the literature reviewed.

REGULATORY LEVELS AND CRITERIA

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

EPA Carcinogenic Classification:	C-possible human carcinogen
Oral Cancer Slope Factor (4):	1.3×10^{-2} (mg/kg/day)$^{-1}$
Inhalation Cancer Slope Factor (4):	6.3×10^{-3} (mg/kg/day)$^{-1}$
OSHA PEL-TWA:	100 ppm 200 ppm (ceiling level)
ACGIH TLV-TWA:	50 ppm 100 ppm (STEL)

REFERENCES

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9. U.S. Environmental Protection Agency (USEPA). 1980. Ambient Water Quality Criteria for halomethanes. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-051.

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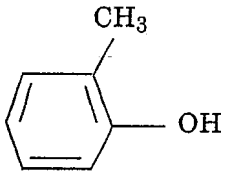
Date of Last Revision: 11/10/94
Revisor: Patrick B. Moroney

2-METHYLPHENOL

INTRODUCTION

Chemical Name: 2-Methylphenol
Synonyms: ortho-Cresol (o-Cresol), 2-Cresol
CAS Number: 95-48-7
Molecular Formula: C_7H_8O
Molecular Weight: 108.15 g/mole
Chemical Structure:



2-Methylphenol (o-cresol) can assume the following forms: a colorless liquid, a colorless crystalline compound or white crystals. It emits a phenolic odor. o-Cresol is an intermediate in the manufacture of phenolic and epoxy resins, sulfur chromium dyes, herbicides, magnet wire coatings, and pharmaceuticals. o-Cresol is also used as a disinfectant, solvent, fiber treatment agent, tanning agent and a metal degreaser (1).

Natural sources of o-cresol are found in small amounts in petroleum, coal and wood. o-Cresol is released to the environment as emissions from the industrial processes involved in the manufacture and use of this compound. Additionally,
o-cresol is present in the wastewater streams from these processes as well as from municipal wastestreams (2).

FATE AND TRANSPORT

Log Koc: 22 (as measured on Brookstone clay loam soil, pH=5.7) (2)
18 (based on water solubility) (2)
1.03 (4)
Log Kow: 1.95 (calculated) (2,4); 2 (calculated) (3)
Half-life: 9.6 hr in air during day; 2 min in air at night (2)
Fully biodegrades (100%) in soil in 8 days (2)
Henry's Law Constant: 1.6×10^{-6} atm-m^3/mole (2)
1.2×10^{-6} atm-m^3/mole @25°C (4)
BCF: 18 (estimated based on log Kow=1.95) (2); log BCF=1.25 (4)
Degradation Products: When heated to decomposition, 2-methylphenol emits highly toxic fumes (1) Nitrocresols (upon reaction with nitrate radicals) (2)
Solubility:
Water: Soluble in 40 parts water (1); 30.8 mg/L @ 40°C (2) 31,000 mg/L @ 40°C (3); 25,950 ppm @25°C (4)
Organic Solvents: Miscible with alcohol, benzene, ether and glycerol (3); soluble with acetone, chloroform, and alkali hydroxides (4)
Vapor Pressure: 1.0 mm Hg @ 38.2°C (1)
0.31 mm Hg @ 25°C (2)
0.299 mm Hg @ 25°C (4)

Specific Gravity: 1.047 @ 20°C (1)
1.030-1.038 @ 25°C (3)

o-Cresol is released to the atmosphere as a result of the following processes: auto and diesel exhaust; coal tar and petroleum refining; wood pulping; and metal manufacture and refinement. Upon atmospheric release, o-cresol will react with hydroxyl radicals produced photochemically. At night, it will react with nitrate radicals generating nitrocresols. In addition, it can be oxidized by metal cations present in rainwater and other atmospheric moisture (2).

Wastewater from these industrial processes as well as from municipal wastestreams contain o-cresol. When released to water, o-cresol will dissolve readily and biodegrade quickly. It has no hydrolyzable groups. Therefore, hydrolysis as a loss process will be insignificant. In the surface layers of oligotrophic waters, photolysis is significant. The low Henry's Law constant for o-cresol indicates that this compound will partition into the water phase, and volatilization/evaporation will not be significant from surface water (2).

Although its fate in soil has not been fully characterized, it is relatively mobile in most soils. Soils which contain high levels of iron oxide and pH are the exception. Also, evidence indicates that o-cresol will biodegrade in soil, but the rate had not been determined (2).

Based on the estimated bioconcentration factor, o-cresol is not expected to significantly bioaccumulate in fish (2).

PHARMACOKINETICS

Little information regarding the rate and extent of absorption of cresols via the oral and inhalation routes in human receptors is currently available. The occurrence of coma, death and systemic effects in two humans dermally exposed to cresols indicates that cresols can be absorbed through the skin. No studies were available that quantify the rate or extent of absorption in humans. An in vitro study of the permeability of human skin to cresols found that these substances had permeability coefficients greater than that for phenol, which is known to readily absorb in human skin. In terms of the distribution of and removal of cresols from the human body upon oral, dermal and inhalation exposure, no studies are currently available (4).

In general, cresols are major skin and eye irritants. Cresols can aggravate the mucous membranes, impair liver and kidney function, and damage the central nervous and cardiovascular systems. Occupational exposure to cresols has caused severe burns and eczema. Cresol released to the air have caused headaches, vomiting and digestive disorders (3).

The most probable route of human exposure is inhalation. Dermal contact is a primary occupational exposure route (2).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

The USEPA reference dose for chronic oral exposure to o-cresol is $5x10^{-2}$ mg/kg/day. The reference concentration for chronic inhalation exposure (RfC) has not been verified by USEPA. As stated previously, o-cresol is very irritating to the skin, mucuous membranes and eyes. Cresols can cause disturbances in the central nervous and cardiovascular systems.

Occupational dermal exposure to cresols has caused severe burns and eczema. Inhalation of o-cresol released to the air have caused headaches, vomiting and digestive disorders (1,2,3).

Carcinogenic Effects

o-Cresol has been classified by the USEPA as possible human carcinogen (USEPA Weight-of-Evidence = C). This is primarily based on an initiation-promotion study where, upon dermal application, an increased occurrence of skin papillomas in mice occurred. In addition, the three cresol isomers (o-,m- and p-cresol) acting alone and in combination generated positive results in genetic toxicity studies (1).

In terms of human carcinogenicity data, inadequate information is currently available. Two cases of multifocal transitional cell carcinoma of the bladder following chronic occupational exposure to cresol and creosote were documented. In addition, a carcinoma of the vocal cords was noted for a petroleum refinery worker with a long history of exposure to cresol, dichlorooctane and chromic acid (1).

ENVIRONMENTAL HEALTH EFFECTS

Cresols present in surface water are toxic to fish and other aquatic life. Trout embryos are especially susceptible. There currently is no evidence that cresols bioaccumulate in the tissues of wildlife species. No changes in reproductive capabilities in wildlife species were attributed to these compounds (3).

REGULATORY LEVELS AND CRITERIA

National o-cresol regulatory levels and criteria relative to a risk assessment are established under the Safe Drinking Water Act (SDWA), and the Ambient Water Quality Criteria (WQC).

SUMMARY OF CRITERIA

Reference Dose for Chronic Oral Exposure (RfD)	5.0×10^{-2} mg/kg/day (1)
NOAEL	50 mg/kg/day (1)
LOAEL	50 mg/kg/day (1)
Chronic Inhalation (RfC)	Information inadequate to verify an RfC (1)
EPA Weight-of-Evidence	C-Possible human carcinogen (1)
SDWA MCLG	Not Available (NA)
SDWA MCL	NA
OSHA PEL-TWA (skin):	5 ppm (3)
ACGIH TLV-TWA:	5 ppm (3)

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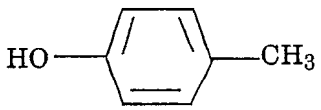
Date of Last Revision: 11/10/94
Revisor: Patrick B. Moroney

4-METHYLPHENOL

INTRODUCTION

Chemical Name: 4-Methylphenol
Synonyms: para-Cresol (p-Cresol), 4-Cresol
CAS Number: 106-44-5
Molecular Formula: C_7H_8O
Molecular Weight: 108.13 g/mole
Chemical Structure:



4-Methylphenol (p-Cresol) is a plant volatile. Also in nature, it is found in petroleum. p-Cresol is released to the environment as emissions and in wastewater as a result of its production in coal tar refining and use as a disinfectant, in metal refining, and chemical manufacturing. p-Cresol is also produced in auto and diesel exhaust, wood pulping, brewing, glass fiber manufacture, and in tobacco smoke (2).

FATE AND TRANSPORT

Log Koc: 49 (as measured on Brookstone clay loam soil) (2)
0.9 (based on water solubility) (2)
1.69 (4)
Log Kow: 1.94 (calculated) (2,4); 2 (calculated) (3)
Half-life: 10 hr in air during day; 4 min in air at night (2)
Fully biodegrades (100%) in soil in 7 days (2)
0.55 hr in a river; 12 hr in a pond or eutrophic lake;
2400 hr in a oligotrophic lake (computer model); 1.4 and 290 yrs in a typical river or lake (based on evaporation rate and laboratory data (2)

Henry's Law Constant: 9.6×10^{-7} atm-m^3/mole (2); 7.92×10^{-7} atm-m^3/mole @25°C (4)
BCF: 18 (estimated based on log Kow = 1.94) (2); log BCF = 1.25 (4)
Degradation Products: Nitrocresols (upon reaction with nitrate radicals) (2)
Solubility:
Water: 22.6 mg/L @ 40°C (2); 21,520 ppm @25°C (4)
Organic Solvents: Miscible with alcohol, benzene, ether and glycerol (3); Soluble with acetone, chloroform, and alkali hydroxides (4)
Vapor Pressure: 0.13 mm Hg @ 25°C (2); 0.11 mmHg @ 25°C (4)
Specific Gravity: 1.030-1.038 @ 25°C (3)

As stated previously, p-cresol is released to the atmosphere as a result of the following processes: auto and diesel exhaust; coal tar and petroleum refining; wood pulping; and metal manufacture and refinement. Upon atmospheric release, p-cresol will react with hydroxyl radicals produced photochemically. At night, it will react with nitrate radicals generating

nitrocresols. In addition, it can be oxidized by metal cations present in rainwater and other atmospheric moisture (2).

Wastewater from these industrial processes as well as from municipal wastestreams contain p-cresol. When released to water, p-cresol will dissolve readily and biodegrade quickly. It has no hydrolyzable groups. Therefore, hydrolysis as a loss process will be insignificant. In the surface layers of oligotrophic waters, photolysis is significant, especially in the prescence of humic acids. In rivers, dilution effects are more significant than biodegradation. The low Henry's Law constant for p-cresol indicates that this compound will partition into the water phase, and volatilization, evaporation and sorption will not be significant in any water bodies (2).

Although its fate in soil has not been fully characterized, it is relatively mobile in most soils and may leach into groundwater. Also, evidence indicates that p-cresol will rapidly biodegrade in soil (2).

Based on the estimated bioconcentration factor, p-cresol is not expected to significantly bioaccumulate in fish (2).

PHARMACOKINETICS

Little information regarding the rate and extent of absorption of cresols via the oral and inhalation routes in human receptors is currently available. The occurrence of coma, death and systemic effects in two humans dermally exposed to cresols indicates that cresols can be absorbed through the skin. No studies were available that quantify the rate or extent of absorption in humans. An in vitro study of the permeability of human skin to cresols found that these substances had permeability coefficients greater than that for phenol, which is known to readily absorb in human skin. In terms of the distribution of and removal of cresols from the human body upon oral, dermal and inhalation exposure, no studies are currently available (4).

In general, cresols are major skin and eye irritants. Cresols can aggravate the mucous membranes, impair liver and kidney function, and damage the central nervous and cardiovascular systems. Occupational exposure to cresols has caused severe burns and eczema. Cresol released to the air have caused headaches, vomiting and digestive disorders (3).

The most probable route of human exposure is inhalation. Dermal contact is a primary occupational exposure route (2).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

The USEPA reference dose for chronic oral exposure to p-cresol was withdrawn on 8/1/91 as a result of further review. A new RfD summary is in preparation; however, HEAST has published an oral RfD of $5x10^{-3}$ (5). The reference concentration for chronic inhalation exposure (RfC) has not been verified by USEPA. As stated previously, p-cresol is very irritating to the skin, mucuous membranes and eyes. Cresols can cause disturbances in the central nervous and cardiovascular systems. Occupational dermal exposure to cresols has caused severe burns and eczema. Inhalation of p-cresol released to the air have caused headaches, vomiting and digestive disorders (1,2,3).

Carcinogenic Effects

p-Cresol has been classified by the USEPA as possible human carcinogen (USEPA Weight-of-Evidence = C). This is primarily based on an iniatiation-promotion study where, upon dermal application, an increased occurrence of skin papillomas in mice occurred. In addition, the three cresol isomers (o-,m- and p-cresol) acting alone and in combination generated positive results in genetic toxicity studies (1).

In terms of human carcinogenicity data, inadequate information is currently available. Two cases of multifocal transitional cell carcinoma of the bladder following chronic occupational exposure to cresol and creosote were documented. In addition, a carcinoma of the vocal cords was noted for a petroleum refinery worker with a long history of exposure to cresol, dichlorooctane and chromic acid (1).

ENVIRONMENTAL HEALTH EFFECTS

Cresols present in surface water are toxic to fish and other aquatic life. Trout embryos are especially susceptible. There currently is no evidence that cresols bioaccumulate in the tissues of wildlife species. No changes in reproductive capabilities in wildlife species were attributed to these compounds (3).

REGULATORY LEVELS AND CRITERIA

National p-cresol regulatory levels and criteria relative to a risk assessment are established under the Safe Drinking Water Act (SDWA), and the Ambient Water Quality Criteria (WQC).

SUMMARY OF CRITERIA

Chronic Oral (RfD):	$5x10^{-3}$ (5)
Chronic Inhalation (RfC):	Information inadequate to verify an RfC (1)
EPA Weight-of-Evidence:	C-Possible human carcinogen (1)
SDWA MCLG:	Not Available (NA)
SDWA MCL:	NA
OSHA PEL-TWA (skin):	5 ppm
ACGIH TLV-TWA:	5 ppm

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Revisor: Rich Hoff

NICKEL

INTRODUCTION

Chemical Name:	Nickel
CAS Number:	7440-02-0
Molecular Formula:	Ni
Molecular Weight:	58.7 g/mole
Chemical Structure:	Ni

Nickel is a malleable silver-white metal that occurs in valences 0,+1, +2, +3, and +4. It is almost always found in the divalent oxidation state in aquatic systems. Nickel is commonly used in alloys, electroplated protective coatings, and fuel cell electrodes. The main source of nickel in the atmosphere is due to the burning of fuel oil. Nickel-containing sewage sludge, the use of certain fertilizers and the deposition of aerosol particles are primarily responsible for nickel contaminated soil (1,2).

FATE AND TRANSPORT

Solubility (1):	Insoluble in water; some salts are soluble
Specific Gravity (1):	8.902 at 25°C

Nickel is found in the air, water, and soil. The average residence time for nickel in the atmosphere is seven days, the residence time in water is between 19 and 23,000 years, and the average time in soil is estimated to be 2400-3500 years (4).

The bioconcentration factors for most aquatic organisms suggests that bioaccumulation would not be significant. However, the BCFs for marine phytoplankton range from 20-8000. Nickel is reasonably mobile in soils with a low pH and cation exchange capacity. It is less mobile in soils with a high organic content. Uptake of nickel in plants from the soil is common (4).

There are no data referencing the significance of biodegradation, photolysis, or volatilization of nickel as environmental fate processes (1,4).

PHARMACOKINETICS

Quantitative data concerning the uptake of nickel by the respiratory tract are not available, however, studies have shown that nickel accumulates in the soft tissue of the lungs. Dietary nickel which has been ingested is absorbed at a level of 1-10%. Following ingestion of nickel, it was found in serum as ultrafilterable nickel, albumin-bound nickel, and in a metalloprotein. Oral doses of nickel chloride have been found to localize in the kidneys, lungs and central nervous system of animals (4).

Dermal penetration of nickel on human skin was found to occur at a level of 55 to 77% in one study. Studies concerning the distribution of nickel which is dermally absorbed are not available (4).

Once nickel is absorbed in the body it binds to a number of serum macromolecular components, (e.g. in humans, nickel binds to albumin, 1 - histidine and alpha-2-macroglobulin).

Most of the nickel that is introduced into the body by inhalation, oral, and dermal absorption is excreted in the urine. Unabsorbed nickel resulting from oral exposure is excreted in the feces (4).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

In both humans and animals, the lung has been the organ targeted for nickel toxicity. Inhalation of nickel is associated with lung cancer, allergenic response, asthma, and pulmonary infections. No human or animal studies regarding dermal absorption are available. No studies concerning oral effects on humans have been identified. However, pulmonary effects on animals were observed when nickel was administered orally (4).

No immunological, renal, or hematological effects have been observed in humans. Injection studies have shown immunological effects in mice. These mice showed significant immunosuppression. Renal effects in mice consisted of aminoaciduria and proteinuria, which are indicative of renal dysfunction. Hematological effects in animals were exhibited by increased white blood cell counts, decreased hemoglobin concentration, decreased hematocrit, and histological lesions in the bone marrow (4).

Teratogenic and reproductive effects of nickel in humans and animals are inadequate. However, mammalian cell transformation data indicates that specific nickel compounds are mutagenic and cause chromosomal alterations (4).

The most common effect of nickel exposure is a sensitization reaction exhibited by dermatitis (4).

The oral RfD is 0.02 mg/kg/day (soluable salts) and is based on decreased organ and body weights in rats. A NOAEL of 5 mg/kg/day was identified. An uncertainty factor of 100 was applied based on a factor of 10 for interspecies extrapolation and a factor of 10 to protect sensitive populations; the modifying factor is 3 due to inadequacies in the reproductive studies (4). The oral RfD supporting study consisted of a 2-year feeding study using rats administered nickel sulfate hexahydrate in concentrations of 0, 100, 1000, or 2500 ppm in the diet. Body weight was significantly decreased at 1000 and 2500 ppm. No significant effects were reported at 100 ppm (3).

Carcinogenic Effects

Although the EPA has not assigned a carcinogen classification for soluble salts of nickel, it has assigned a Group A classification for nickel refinery dust, nickel carbonyl and nickel subsulfide. This classification indicates that there is sufficient evidence from epidemiologic studies to support a causal association between nickel exposure and cancer in humans (4). EPA has also established an inhalation slope factor of 8.4×10^{-1} $(mg/kg/day)^{-1}$ (3).

Epidemiological studies found that nickel-exposed workers developed three types of respiratory cancer: epidermoid, anaplastic, and pleomorphic carcinomas (4).

Experimental animal studies indicate that upon injection, tumors appear on animals at the injection site (4).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

The Ambient Water Quality Criteria for the level of nickel that will not adversely affect aquatic organisms and their uses are as follows (5):

Freshwater:
- Acute toxicity: 1,400 μg/L*
- Chronic toxicity: 160 μg/L*

Saltwater:
- Acute toxicity: 140 μg/L
- Chronic toxicity: 7.1 μg/L

* Based on a hardness of 100 mg/L as $CaCO_3$ (calcium carbonate)

Terrestrial and Avian

No data are available for terrestrial and avian life forms (4).

REGULATORY LEVELS AND CRITERIA

OSHA PEL-TWA (8):	1.0 mg/m^3 (metal and insoluble compounds) 1.0 mg/m^3 (soluble compounds)
ACGIH TLV-TWA (9):	1.0 mg/m^3 (insoluble compounds, nickel sulfide roasting, fume and dust, and metal) 0.1 mg/m^3 (soluble compounds)
AWQC (4)	
Ingestion of Water and Organisms:	632 μg/L
Ingestion of Only Organisms:	4.77 μg/L (ingesting only organisms)
Reportable quantity (3)	1 lb
Maximum Contaminant Level (MCL) (6)	0.1 mg/L 0.1 mg/L

SUMMARY OF CRITERIA

EPA Carcinogenic Classification(3)	Group A-Sufficient evidence to support the causal association between nickel inhalation (nickel refinery dust, nickel carbonyl, and nickel subsulfide) and cancer Group C - Limited evidence of carcinogenicity to support the causal association between exposure (nickel soluble salts) and cancer.
Nickel Refinery Dust	
Cancer Slope Factor (inhalation) (7)	8.4×10^{-1} $(mg/kg/day)^{-1}$
Nickel (Soluble Salts)	
Oral RfD (3)	0.02 mg/kg/day
Inhalation RfD (3)	Pending

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Revisor: Rich Hoff

POLYCHLORINATED BIPHENYLS (PCBS)

INTRODUCTION

Chemical Name:	Polychlorinated Biphenyls (PCBs)
Synonyms and Trade Names:	Aroclor, Kanechlor, Clophen
CAS Numbers:	Aroclor 1242: 53469-21-9
	Aroclor 1248: 12672-29-6
	Aroclor 1254: 11097-69-1
	Aroclor 1260: 001336-36-3
Molecular Formula:	$C_6H_xCl_xC_6H_xClx$
Molecular Weights:	Aroclor 1242: 266.5 g/mole
	Aroclor 1248: 299.5 g/mole
	Aroclor 1254: 328.4 g/mole
	Aroclor 1260: 377.8 g/mole

The term polychlorinated biphenyls (PCBs) commonly refers to a variety of mixtures of individual biphenyl isomers, each consisting of two joined benzene rings and up to ten chlorine atoms. Mixtures of these isomers are known by their commercial designation of Aroclor. This trade name is followed by a four-digit number; the first two numbers indicate the type of isomer mixture and the last two numbers indicate the approximate weight percent of chlorine in the mixture (3).

PCBs are man-made chemicals that were used widely in transformers, electrical equipment and as lubricants (2). Because of their persistence and toxicity in the environment, their manufacture was discontinued in the United States in 1977 (1). However, PCB equipment manufactured before 1977 is currently still being used in the U.S. and this use is being regulated by the Environmental Protection Agency.

PCBs are very stable chemically and tend to be persistent in the environment. Persistence and bioaccumulation in living organisms also occur due to the high lipophilicity of these compounds (2).

CHEMICAL AND PHYSICAL PROPERTIES

Aroclor	**1242**	**1248**	**1254**	**1260**
Log K_{oc}	3.8	5.75	5.51	6.3
Log K_{ow} (2):	5.6	6.11	6.03	7.15
Henry's Law Constant(2): (atm-m^3/mol at 25° C)	5.7×10^{-4}	3.5×10^{-3}	8.4×10^{-3}	7.1×10^{-3}
Water Solubility (mg/L):	0.24	0.054	0.012	0.0027
Vapor Pressure(2): (mm Hg at 25° C)	4.06×10^{-4}	4.94×10^{-4}	7.71×10^{-5}	4.0×10^{-5}
Density (2):	1.35	1.41	1.50	1.57

FATE AND TRANSPORT

PCBs can be found in the atmosphere, water, and soil. Adsorption to sediments is the major fate process for PCBs in water. Because of lower water solubilities and higher octanol-water partition coefficients, higher chlorinated isomers will adsorb more strongly than the lower chlorinated isomers. This also indicates that significant leaching should not occur in soil under most conditions (2).

For PCBs that exist in the dissolved state in water, volatilization becomes the primary fate process. Therefore, the volatilization process is the major removal mechanism of PCBs from water sources. However, the rate of volatilization is dependent upon PCB adsorption to sediment (2).

In the atmosphere, PCBs exist in the vapor phase and can be removed by wet and dry deposition. A typical range of PCB concentrations in the atmosphere is between 1 and 250 μg/L (2).

Degradation of PCBs in the environment is dependent upon the degree of chlorination. Generally, the more chlorinated the PCB molecule, the more persistent it will be in the environment. Factors which determine biodegradability include the amount of chlorination, concentration, type of microbial population, available nutrients, and temperature (2). The dominant degradation process in the atmosphere is dependent upon the vapor phase reaction of PCBs with hydroxyl radicals (2).

Photolysis is thought to be the only transformation process in the aquatic environment. However, the process is extremely slow. It appears the hydrolysis and oxidation do not degrade PCBs (2).

In the atmosphere, typical airborne concentrations of PCBs are as follows (2):

Location	Concentration Range (mg/m^3)
Urban	0.5 to 30
Rural	0.1 to 2.0
Great Lakes	0.4 to 3.0
Marine	0.05 to 2.0
Remote	0.02 to 0.5

The concentrations of PCBs in the open waters of oceans and lakes are shown below (2):

Location	Concentration Range (μg/L)
North Pacific	0.04 to 0.59
Antarctic	0.035 to 0.069
North Atlantic	0.02 to 0.20
Lake Superior	0.63 to 3.30
Lake Michigan	3.0 to 9.0
Lake Huron	0.49 to 17.15

PCBs are found in the soils from different areas of the world in the following concentrations (2):

Location	Concentration Range (ppb)
Great Britain	2.3 to 444
South Wales/Scotland	4.5 to 47.7
Japan	<10 to 100
United States	
Everglades National Forest, Florida	<1 to 33
U.S. Urban areas	0.02 to 11.94
Rocky Mountain National Park	0.098 to 0.54
Great Lakes	2.5 to 251.7

PHARMACOKINETICS

PCBs are absorbed primarily through inhalation and dermal contact in occupational environments. However, the general public absorbs PCBs primarily through oral exposure, such as the ingestion of PCB contaminated fish (2).

Animal studies have shown that PCBs are readily absorbed, but studies to quantify the rate of absorption are needed. Studies indicate that PCBs are absorbed by the gastrointestinal tract, and have been found in the serum and breast milk of woman orally exposed to PCBs (2).

PCBs accumulate in human plasma and adipose tissue with the extent of accumulation dependent on the positions of chlorines on the PCB congeners. Congeners with chlorines in both 4 positions as opposed to the 3 ,4 positions were found in greater concentrations (2). Also, PCBs have been shown to accumulate in human breast milk. The extent of accumulation is approximately 4 to 10 times less than the concentration in maternal blood (2).

Animal studies have indicated maximum concentrations in the liver, brain, and adipose tissue. Studies show that distribution occurs in a biphasic manner. First, PCBs accumulate in the liver and muscle from the blood stream. Following this accumulation, PCBs are either stored in the adipose tissue or metabolized by the liver. It has been suggested that PCBs concentrate in the adipose tissue regardless of the route of exposure (2).

The metabolism of PCBs depends on chlorine content and on the site of chlorination. The major metabolic products are phenolic in nature. Other identified end products are sulfur-containing compounds, trans-dehydrodiols, polyhydroxylated PCBs and methyl ether derivatives (2).

Data regarding the excretion of PCBs following inhalation or dermal exposure are not available. When oral exposure occurs, excretion is dependent upon the metabolism of PCBs to more polar compounds (2).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

The evaluation of the toxicity of PCBs is complicated by a number of factors including differences in isomer/congener/mixture composition, differences in species susceptibility, quantitatively inconsistent data, and varying degrees of contamination from other chemicals such as chlorinated dibenzofurans. Also, it should be noted that because of changes in congener and impurity composition resulting from environmental and/or biological transformations, PCBs currently in the environment may differ from the original PCB mixture (2).

Inhalation Exposure

There are no human data available regarding the lethality/decreased longevity of humans due to acute or chronic inhalation exposure. However, the primary target organs associated with PCB inhalation are the liver and cutaneous tissue. Occupational exposure has been associated with elevated serum levels in the liver and enzyme and dermatologic effects such as chloracne and skin rashes (2).

Human developmental studies have proved inconclusive and lack monitoring data. However, there were suggestions that mothers occupationally exposed to PCBs exhibited a slight decrease in birth weight and gestational age of offspring. No animal studies were available concerning developmental toxicity (2).

In animals, the liver and skin are unequivocal targets of PCB toxicity, especially in terms of chronic toxicity. The range of toxicity for dermal and hepatic effects is from 0.007 to 11.0 mg/m^3 (2).

Oral Exposure

There are no studies which address oral PCB exposure in humans. However, animal studies have established a single dose LD50s for rats and mice. The levels are 1,010 mg/kg for Aroclor 1254 and 750 mg/kg for Aroclor 1221, respectively (2).

Systemic effects in animals include perturbations of the liver and cutaneous tissues. Rats fed 0, 4, 8, and 16 ppm of Aroclor 1254 for 4 days resulted in an increase in liver weight at concentrations greater than 8 ppm and an increase of serum HDL cholesterol levels at 16 ppm. A lowest observed adverse effect level (LOAEL) of 5 ppm was identified in rats based on hepatic effects. At this level, hepatic microsomal enzyme activities increased, production of liver lipid content increased, and frank degenerative liver alterations were observed (2).

Developmental effects in humans from oral exposure to PCB contaminated fish include effects on birth weight, head circumference, gestational age and/or neonatal behavior. For animals, a LOAEL of 50 ppm in female rats has been identified based on fetotoxicity. At this level, effects such as reduced litter size, ultrastructural lesions in the thyroid follicular cells of neonates and weanlings and reduced serum levels of thyroid hormone were observed (2).

The only study relating PCBs to reproduction demonstrated that doses of >2 ppm Aroclor 1254 administered to mink for 4 months prior to mating and during gestation were lethal to fetuses and caused reproductive failure (2).

Dermal Exposure

Dermal exposure is a major route of PCB absorption. However, the current data does not allow for the quantification of dermal absorption to the total body burden of PCBs (2).

A study involving capacitor workers does not show clear evidence of liver disease. However, a correlation can be made between the PCB exposure and liver enzyme induction in the workers. It is not clear to what extent the dermal absorption affected the hepatic changes since inhalation exposure also occurred (2).

A study involving dermal exposure of Aroclor 1260 to female New Zealand rabbits for 5 days/week at a dose of 118 mg/day for 38 days produced degenerative lesions of the liver and kidneys, increased fetal porphyrin elimination and hyperplasia and hyperkeratosis of the follicular and epidermal epithelium (2). Other studies indicate that the median lethal dose for single dermal exposure for rabbits was >1269 mg/kg for Aroclor 1242 and 1248 to <3,169 for Aroclor 1221 (2).

No studies have been located which address immunological, neurological, developmental or reproductive effects of PCBs on humans or animals (2).

Carcinogenic Effects

The EPA has classified PCBs as a Group B2 carcinogen - a probable human carcinogen. This classification is based on the evidence of hepatocellular carcinomas in three strains of rats and two strains of mice. There is suggestive evidence that links PCBs to liver cancer in humans by the ingestion, inhalation, or dermal pathways. However, this evidence is inadequate due to confounding factors and lack of exposure quantification (4).

There have been several studies attempting to associate PCB exposure with carcinogenicity. In New Jersey, a petrochemical plant reported a statistically significant increase in malignant melanomas among 31 research and development employees and 41 refinery workers. Because the study failed to report quantified exposure levels and to identify the presence of other potential or known carcinogens, it was discredited (4).

Two outbreaks of poisoning following accidental consumption of PCB-contaminated rice oil (also containing polychlorinated dibenzofurans and polychlorinated quinones) occurred in Japan in 1968 (Yusho) and in Taiwan in 1979 (Yu-Cheng). A 16-year mortality study was completed which identified an increase in liver cancer in both males and females. There is strong evidence indicating the health effects were attributable to the polychlorinated dibenzofurans in the oil as opposed to the PCBs. Therefore, this study only suggests carcinogenicity of PCBs (4).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

PCBs have the capability to bioaccumulate and biomagnify. For rainbow trout, bluegills and channel catfish, the 96-hour LC50 values were approximately 20 mg/liter. When the exposure was increased to 10 to 20 days, the average LC50 value was 0.1 mg/liter. Studies indicate that juvenile organisms appear to be more susceptible to PCBs than either eggs or adults (3).

A study which experimentally determined the bioconcentration factors of various Aroclors in aquatic species found bioconcentration factors ranging from 26,000 to 660,000 (2).

In a study conducted by the U.S. Fish and Wildlife Service, 315 fish from 107 stations nationwide were analyzed for PCBs. Results showed that 94% of all fish were found to contain PCB residues. The geometric mean concentration of all Aroclors was found to be 0.53 μg/g. It should be noted that this study included the analyses of whole fish samples which include both the edible and nonedible portions of the fish. Therefore, the concentration will not reflect the actual human exposure through oral consumption (2).

Subsequent studies have shown PCB levels in fish collected and analyzed from Lake Huron to contain 600 to 72,000 μg/g PCBs on a lipid basis. Analyses of 62 samples of commercial fish collected from Lake Ontario revealed PCB levels ranging from 0.11 to 4.90 ppm (2).

The Ambient Water Quality Criteria for the protection of aquatic organisms are as follows (3):

Freshwater:

Acute toxicity:	2.0 μg/L
Chronic toxicity:	0.014 μg/L

Marine:

Acute toxicity:	10.0 μg/L
Chronic toxicity:	0.030 μg/L

Terrestrial and Avian

PCBs can affect terrestrial wildlife in three primary ways: mortality, adversely affecting reproduction, and changing behavior. Behavioral effects include increased activity, decreased avoidance response, and decreased nesting (3).

In sensitive bird species, PCB levels of greater than 200 ppm in the diet or 10 mg/kg body weight caused some mortality. When the doses were increased to 1,500 ppm or 100 mg/kg body weight, extensive mortality was exhibited (3).

In studies in which chicken were fed levels of 20 ppm PCBs in the diet, lower egg production, deformities, decreased hatchability, lower growth, and survival were observed (3).

REGULATORY LEVELS AND CRITERIA

OSHA Advisory TWA (2):	Aroclor 1242 - 1.0 mg/m3
	Aroclor 1254 - 0.5 mg/m3
FDA Temporary Tolerances (2):	Foods - 0.2-3.0 ppm
	Packaging - 10.0 ppm
ACGIH (2):	
TLV-TWA for Aroclor 1242:	1.0 mg/m3
TLV-TWA for Aroclor 1254:	0.5 mg/m3
Ambient Water Quality Criteria (2):	0.79 to 0.0079 ng/L for carcinogenicity at 10^{-5} to 10^{-7} risk levels
Drinking Water Criteria (2):	MCLG: 0 μg/L
	MCL: 1 μg/L
Reportable Quantity (2):	10 lbs. (statutory)
	1 lb. (proposed)

SUMMARY OF TOXICOLOGICAL INDICES

EPA Carcinogenic Classification (4) Group: B2-Probable human carcinogen

Carcinogenic Effects:	
Oral CSF(4):	7.7 $(mg/kg/day)^{-1}$
Inhalation CSF (4):	Not Available.
Oral RfD (4) for Aroclor 1254:	2.0×10^{-5} mg/kg/day
NOAEL:	None
LOAEL:	0.005 mg/kg-day

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5. HEAST. Health Effects Assessment Summary Tables. Office of Emergency and Remedial Response, U.S. Environmental Protection Agency. March 1994.

Date of Last Revision: 11/10/94
Revisor: Patrick B. Moroney

SILVER

INTRODUCTION

Chemical Name: Silver
Synonyms (1): Argentum
CAS Number (1): 7440-22-4
Molecular Formula (1): Ag
Molecular Weight (1): 107.87

Silver is a soft, lustrous, white metallic element. It is derived as a by-product of copper, zinc, lead, or gold ores operations. The principal silver ores are argentite and cerargyrite (1). Silver has been used in the past for surgical prostheses and splints, fungicides, and coinage. Presently, approximately 45% of the U.S. consumption is used in photographic materials. An additional 25% of the silver in the U.S. is used in electrical and electronic products, silver paints, and batteries. Silver is also important in brazing alloys and soldiers, electroplating, mirrors, dental amalgam, and purification of drinking water (2).

FATE AND TRANSPORT

Solubility (3):
In Water: Insoluble
In Organics: Soluble in alkali cyanide solutions
Specific Gravity (3): 10.5 @ 20°C

In 1978, the U.S. reported an annual release of silver to land from production processes and consumptive uses of 1.01 million kilograms. Silver is released to the atmosphere, water, and land by natural and man-made sources, wet and dry deposition, and sorption to soil and sediments (2).

In the atmosphere, silver is most likely to occur in the form of metallic silver, silver sulfide, silver sulfate, silver carbonate, or silver halides. The major sources of atmospheric releases are from mining operations, fossil-fuel fired power plants, and solid waste incinerators. It was estimated that approximately 50% of the silver released into the atmosphere from industrial operations will be transported more than 100 km (2).

Silver in surface waters and soils is influenced by the particular form of the compound. Under oxidizing conditions, primary silver compounds are by bromides, chlorides, and iodides. Under reducing conditions, the free metal and silver sulfide predominate. Significant quantities of silver in surface water are sorbed by manganese dioxide whereas pH, and oxidation-reduction conditions affect sorption. Sorption is the dominant process leading to the partitioning of silver in sediments. The concentration in lake sediments were reported to be 1000 times that of the overlying waters (2).

In soil, the mobility of silver is limited by drainage, oxidation-reduction potential, pH, and the presence of organic matter (2).

PHARMACOKINETICS

Silver is absorbed following inhalation exposure. The predominant routes of exposure in the workplace are inhalation and dermal contact. Silver has been reported to be absorbed by humans across mucous membranes in the mouth and nasal passages. The extent of absorption has been found to be associated with clearance time from the gastrointestinal tract. The faster the clearance time, the less silver is absorbed (2).

Human studies have indicated that an estimated 25% of absorbed silver, following inhalation exposure, was distributed to the liver between 2 and 6 days after exposure. The remaining silver was detected in the gall bladder, lungs, liver, blood, intestines, kidneys, and stomach. Following oral exposure, silver was distributed to various body tissues. Ingested silver passes through the liver resulting in excretion into the bile, thus reducing the amount to be distributed to remaining body tissues. Silver has been detected in the muscle, liver, spleen, kidney, heart and bones of humans dermally treated with silver nitrate (2).

The deposition of silver in tissues and organs is the result of the precipitation of insoluble silver salts. Once in the insoluble form, it appears to be transformed into soluble silver sulfide albuminates, resulting in binding or complexation with amino or carboxyl groups in RNA, DNA, and proteins, or to be reduced to metallic silver by ascorbic acid or catecholamines (2).

In an incident which involved the accidental inhalation of radioactive silver metal dust, silver was excreted through the lungs by ciliary action, with ultimate elimination in the feces. Silver was not detected in urine samples. Following both oral and dermal exposure, silver was found to be excreted primarily through the feces, with minor amounts eliminated through the urine (2).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Death from exposure to silver has not been reported. However, a worker was exposed to an unknown form of silver during work with molten silver ingots and experienced respiratory failure the day after exposure. Without treatment, the worker may have died. In terms of laboratory experiments, high levels of colloidal silver were observed to cause death in rats when administered in drinking water for acute and intermediate exposure durations (2).

Upper and lower respiratory tract irritation in humans has been exhibited following inhalation of silver nitrate and/or silver oxide at concentrations of 0.039 and 0.378 mg silver/m3. Ultrastructural damage was seen in the tracheal epithelium of rabbits following inhalation exposure to a silver colloid. Gastric discomfort also accompanied these effects in rabbits. However, these symptoms have resulted from the caustic effects of the silver compounds, and not the presence of silver itself. Silver may also affect renal function. It has been reported that silver is deposited in the glomerular basement membrane of the kidney of animals. However, human studies have not been identified. The predominant effect of exposure to silver in humans is the irreversible pigmentation of the skin. This condition, known as argyria, is a change in the pigmentation of skin to a slate-gray, blue-gray, or gray color, observed in humans exposed to silver. This discoloration is caused by the photo reduction of silver chloride and/or silver phosphate in the skin (2).

Silver has been known to cause a mild allergic response in humans following dermal exposure to various silver compounds. Studies involving woman using nasal drops containing silver nitrate and animals exposed by intraperitoneal injection and through drinking water, have suggested deposition of silver in neurons of the central nervous system. Also, exposure to

silver has affected the volume of hippocampus cell groups within the brain of animals. The implication of the altered volume of these cell groups are not known (2).

Information regarding the developmental effects of silver to humans was not identified. However, there is a possibility of a relationship between the concentration of silver in the tissue of fetuses and the occurrence of developmental abnormalities (2). The EPA has established an oral reference dose of $5x10^{-3}$ mg/kg/day (4).

Carcinogenic Effects

The EPA has assigned silver a carcinogen classification of Group D-not classified as to human carcinogenicity. This is based on the fact that in animals, local sarcomas have been induced after implantation of foils and discs of silver. However, the finding have been questioned due to the phenomenon of solid-state carcinogenesis in which even insoluble solids such as plastic have been shown to result in local fibrosarcomas (4).

Silver is not mutagenic but it may be genotoxic to humans. Studies have shown that silver ions bind with DNA in solution in vitro, and that it can interact with DNA in ways that cause DNA strand breaks and affect the fidelity of DNA replication (2).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Although data are not adequate for establishing criteria, the EPA has reported the lowest values of silver known to be toxic in aquatic organisms. These values are as follows (5):

Freshwater:
- Acute: 4.1* (proposed criteria of 0.92 µg/L)
- Chronic: 1.2x10-01 µg/L

Marine:
- Acute: 2.3 µg/L
- Chronic: 0.92 µg/L (proposed criteria)

*Hardness dependent criteria

Acute toxicity values for freshwater invertebrates range from 0.25 µg/L for Daphnia magna to 4,500 µg/L for Gammarus pseudolimnaeus. Acute values for fish range from 3.9 µg/L for the fathead minnow to 280 µg/L for rainbow trout. The acute toxicity of silver appears to decrease as hardness increases. Also, soluble compounds are generally much more toxic than insoluble compounds (3).

Terrestrial and Avian

Information regarding the toxicity of silver to terrestrial and avian wildlife or domestic animals was not located in the available literature.

REGULATORY LEVELS AND CRITERIA (2)

OSHA PEL-TWA (6):	0.01 mg/m^3
ACGIH TLV-TWA (7):	0.1 mg/m^3 (metal)
	0.01 mg/m^3 (soluble compounds)
FDA Permissible level in bottled water:	0.05 mg/L

Reportable Quantity:	1,000 lb (silver and compounds) 1 lb (silver nitrate)
SMCL:	0.1 mg/L
Recommended Drinking Water Limit:	0.05 mg/L
Ambient Water Quality Criteria:	
Water and Organisms Consumption:	$5.0 \times 10^{+1}$ µg/L
Organisms Consumption Only:	None
Oral RfD (4)	0.005 mg/kg/day
NOEL	None
LOAEL	0.014 mg/kg/day
EPA Carcinogen Classification:	Group D-Not classified as a carcinogen

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Revisor: Rich Hoff

THALLIUM

INTRODUCTION

Acute exposure to soluble thallium compounds has been associated in humans with gastrointestinal irritation; damage of the liver, kidneys, and central and peripheral nervous systems; pulmonary edema; degenerative changes in the adrenals; and ocular effects.

CAS Number:	7440-28-0
Chemical Formula:	Tl
IUPAC Name:	Thallium

CHEMICAL AND PHYSICAL PROPERTIES

Atomic Weight:	204.37
Boiling Point:	1,457°C
Melting Point:	303.5°C
Specific Gravity:	11.85
Solubility in Water:	Insoluble (many compounds are soluble)

FATE AND TRANSPORT

In reducing environments, thallium may be precipitated as the metal or as thallium sulfide. However, much of the thallium present in aquatic systems is likely to remain in solution and be transported to the oceans. Active removal of some dissolved thallium by sorption to clay minerals and hydrous metal oxides present in bed sediments is probably an important environmental fate process. Thallium is readily taken up by aquatic organisms, and bioaccumulation may also be an important fate process. Results of limited studies with algae suggest that thallium may also be available for food chain magnification. There is no evidence to suggest that photolysis or volatilization are important environmental processes. Although there is speculation that thallium can be methylated under aerobic conditions by electrophilic attack, biotransformation does not appear to be an important process in aquatic systems.

HUMAN HEALTH EFFECTS

There is no evidence that thallium is carcinogenic in humans or experimental animals, and it does not appear to have significant mutagenic activity. Exposure to thallium salts during critical developmental stages is reported to produce achondioplasia in chickens and rats. No other significant teratogenic effects are reported.

Thallium, in the form of soluble compounds, is readily absorbed through the skin and gastrointestinal tract. Symptoms associated with acute poisoning in humans include gastrointestinal irritation; liver and kidney damage; pulmonary edema; degenerative change in the adrenals, peripheral nervous system, and central nervous system; and ocular effects, including optic neuritis and, rarely, cataracts. The estimated lethal dose for humans is 8 to 12 mg/kg. In experimental animals, thallium

compounds produce effects similar to those seen in humans. Rats appear to be particularly sensitive to the cataractogenic activity of thallium. Regardless of the specific thallium compound tested, rate of intake, or route of administration, LD_{50} values for a variety of species range from about 3 to 92 mg/kg.

The oral RfD of 8×10^{-5} mg/kg/day applies to thallium carbonate, chloride or sulfate. It is based on a subchronic (90D), gavage study, using thallium sulfate in Sprague-Dawley rats. The NOAEL determined in the study was 0.25 mg/kg/day. Target organs included the liver, blood and hair. Critical effects were increased SGOT, increased serum LDH an alopecia (3).

Toxicity to Wildlife and Domestic Animals

Acute and chronic toxicity of thallium to freshwater aquatic life occurs at concentrations as low as 1,400 and 40 μg/liter, respectively. Acute toxicity to saltwater aquatic life occurs at concentrations as low as 2,130 μg/liter. Toxic effects would be expected to occur at lower concentrations among species more sensitive than those tested. Bioconcentration factors ranged from about 11 for the mussel Mytilus edulis to about 1.5×10^5 for other freshwater and marine invertebrates. Values of about 1×10^5 are reported for marine and freshwater fish.

REGULATORY LEVELS AND CRITERIA

Ambient Water Quality Criteria (USEPA):

Aquatic Life

The available data are not adequate for establishing criteria.

Human Health

EPA Carcinogenic Classification:	D--not classified as a carcinogen
Criterion:	13 μg/liter
OSHA PEL-TWA:	0.1 mg (soluable compounds as Tl)
ACGIH TLV:	0.1 mg/m^3 (elemental and soluble compounds, as Tl)
Oral Reference Dose (RfD):	8×10^{-5} mg/kg/day for thallium carbonate/ chloride/sulfate

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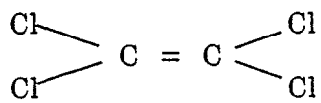
Revisor: Rich Hoff

TETRACHLOROETHENE

INTRODUCTION

Chemical Name: Tetrachloroethene
Synonyms: Perchloroethylene; PCE; perchlor
CAS Number: 127-18-4
Molecular Formula: C_2Cl_4
Molecular Weight: 165.83 g/mole
Chemical Structure:



Tetrachloroethene, also known as perchloroethylene (PCE), is a man-made substance and is not known to occur in nature (1). PCE is an extremely stable, colorless liquid with an ether-like odor. The most common uses of PCE are as a dry cleaning solvent, vapor-degreasing solvent, drying agent for metals, vermifuge, heat-transfer media, and as a raw material for the production of other man-made chemicals (2).

FATE AND TRANSPORT

K_{oc} (3): 364
Log K_{ow}: 2.88 (2)
2.60 (3)
3.40 (4)
Half-life:
Air: 47 days
Surface Water: 1 to 30 days
Henry's Law Constant: 2.59×10^{-2} atm-m^3/mole (3)
1.49×10^{-2} atm-m^3/mole (4)
BCF: 42 (Fish) (3)
38.9 fathead minnow (Pimephales promelas) (4)
49 bluegill sunfish (Lepomis macrochirus) (4)
Solubility:
In Water: 150 mg/L @ 25° C (2)
In Organics: Miscible with ethanol, diethyl ether, and oils in all proportions
Vapor Pressure: 14 mm Hg @ 20°C (2)
18.49 mm Hg @ 25°C (4)
24 mm Hg @ 30°C (2)
Specific Gravity: 1.62 @ 20°C (2)

The majority of the PCE released into the environment is lost by evaporation from industries such as dry cleaners, metal degreasing units, and in the production of fluorocarbons and other chlorohydrocarbons. An estimated 80 to 85% of the PCE used in the U.S. is released to the environment. Other routes of loss include landfill sites and stack effluents from municipal waste incinerators (2).

Volatilization of PCE occurs rapidly. The rate of evaporation is dependent on temperature, water movement and depth, and associated air movement. PCE volatilization half-lives for typical bodies of water are: ponds-7 days; rivers-1.4 days; lakes-5.6 days (2).

The K_{oc} values are indicative of medium to high soil mobility. Studies of groundwater have indicated that leachability is a contributing factor to PCE migration. Also, it has been shown that PCE will not partition significantly from a water column to sediment in natural bodies of water (2).

Because of the detection of PCE in rainwaters collected in the U.S., it is thought that physical removal by means of wet deposition is an important environmental fate mechanism (2).

The dominant degradation process of PCE in the atmosphere is via hydroxyl radicals reactions. The estimated half-life of these radicals is approximately 96 day. This long half-life allows for atmospheric transport which can result in exposures at regions distant from the emission source (2).

In water and soil environments, biodegradation and hydrolysis are the primary transformation processes. The hydrolysis half-life of PCE in water at room temperature is approximately 9 months. Because biodegradation occurs slowly, the most prominent degradation process from surface water occurs through volatilization (2).

PHARMACOKINETICS

The primary route of PCE absorption is through inhalation exposure. The pulmonary uptake is proportional to the rate of ventilation, duration of exposure, and the concentration in inhaled air. PCE is readily absorbed from the lungs into the blood in both humans and animals. The largest percentage of absorption occurs during the first few minutes of exposure (2).

Only one occurrence of oral exposure in humans has been documented. This case reveals that PCE was found in the blood of a person who ingested 12 to 16 g of PCE. In animals, PCE is rapidly and virtually completely absorbed following oral administration (2).

Absorption by dermal exposure is insignificant when compared to absorption by the inhalation route. Animal studies indicate a poor in vivo dermal absorption of 0.24 $mg/cm^2/hour$ and PCE penetration factor of 0.0055 $mg/cm^2/hour$ (2).

There were no available studies on dermal or oral distribution of PCE in humans or animals. Distribution following inhalation has not been studied extensively in humans. In animals, PCE is readily distributed to fat compartments, primarily adipose tissue and perinatal fat. PCE is also known to cross the placenta for distribution to the fetus and amniotic fluid (2).

In general, PCE undergoes metabolism to a form a of glutathione s-conjugate. This metabolic product is formed in the liver and transported to plasma and bile for elimination. PCE is also to metabolized by beta-lyase to an unstable thiol that leads to the formation of toxic products (2). The primary metabolites in the urine and blood of humans exposed to PCE via inhalation

were trichloracetic acids and trichloroethanol. In rats, the primary metabolite was oxalic acid (2).

One case study of a child who ingested 8 to 10 ml of pure PCE reported that trichloroethylene, trichloroethane and unmetabolized PCE were detected in the urine of the child. The overall PCE metabolism is capacity-limited in animals. Oxalic acid is the primary urinary metabolite; however concentrations of N-oxalylaminoethanol, dichloroacetic acid, trichloroethane, N-trichloroacetylaminoethanol, and free and conjugated trichloroethylene have been detected (2). There are no studies available which address the metabolism of PCE via dermal exposure (2).

Regardless of the route of exposure, the primary route of excretion is the exhalation of the unmetabolized parent compound. The approximate amount of PCE excreted from the lungs unchanged is 80 to 100%. Animal studies suggest that PCE metabolism and urinary excretion of metabolites in rodents are rate-limited and dose dependent. The half-lives of PCE via inhalation exposure are: 12 to 16 hours in the vessel-rich group, 30 to 40 hours in the muscle group, and 55 hours in the adipose group (2).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Neurological effects of acute inhalation exposure to high levels of PCE are well documented. Symptoms include eye and upper respiratory irritation, headaches, dizziness, and drowsiness. A human threshold of PCE may be in the range of 100 to 200 ppm. Studies involving animals indicate that alterations in behavior as a result of continuous exposure to PCE concentrations as low as 60 ppm. CNS-depressant effects of PCE may be attributed to the incorporation of lipophilic compound into brain membranes and the subsequent alteration of the fatty acid pattern of brain phospholipids. Histological and biochemical evidence to support neuronal-damage is manifested as hypertrophy and/or proliferation of astroglial cells. Also, another study has demonstrated that the brain taurine content in gerbils was significantly reduced when the gerbils were exposed to 120 ppm PCE for 12 months. The reduction of taurine and other amino acids leads to alterations in nerve impulse transmission (2).

PCE is a known hepatotoxin in humans and animals via oral and inhalation routes. However, exposure and dose are less adequately characterized. It has been known since the 1960s that acute or chronic exposure to PCE via inhalation can cause liver damage. Manifestations include cirrhosis of the liver, toxic hepatitis, liver cell necrosis, hepatomegaly, and altered liver function indices. Animal studies have demonstrated that mice are hypersensitive to PCE. Following short-term exposure, peroxisome proliferation in mouse liver was evident. PCE induced hepatic effects associated with intermediate duration exposure are not totally reversible (2).

Renal effects from inhalation and oral exposure are manifested in rodents by the accumulation of alpha-2u globulin in lysosomes, degeneration and necrosis of tubular cells, formation of granular casts, and regeneration of the tubular epithelium. Human documentation of these effects does not exist. However, because nonproliferative kidney lesions are characteristic of various halogenated aliphatic hydrocarbons in humans, PCE should be regarded as a nephrotoxins (2).

Studies addressing the cardiotoxicity of PCE have been limited. It is hypothesized that PCE exposure in humans may sensitize the myocardium to endogenous epinephrine. Although studies involving dogs failed to support this hypothesis, intravenous administration of PCE enhanced myocardial sensitivity to an exogenous epinephrine challenge (2).

Studies examining the developmental effects of PCE in humans were not available. Animal studies have indicated that PCE is fetotoxic but not teratogenic at maternally toxic concentrations. One study examining high concentrations of TCA (PCE metabolite) concluded that TCA may be transported to the fetus through the fetal amniotic fluid. Long-term retention in the fetus of animals is thought to contribute to embryotoxicity (2).

Data regarding reproductive toxicity in humans were not available, and were limited in animal studies. One study showed a presence of abnormal sperm in mice exposed to 500 ppm PCE. Evidence that PCE or its metabolites reached germinal tissue and damaged DNA was not provided in the study (2).

Based on a gavage study involving Swiss-Cox mice, the EPA has derived an oral RfD for PCE. The mice were exposed to doses of 0, 20, 100, 200, 500, 1500, and 2000 mg/kg, 5 days per week for 6 weeks. Liver toxicity parameters were evaluated in the mice to determine a no observed adverse effect level and (NOAEL) lowest observed adverse effect level (LOAEL). A NOAEL and LOAEL of 14 and 71 mg/kg/day, respectively, were identified from this study. The hepatotoxic effects reported at these dose levels were weight gain and increased liver triglyceride levels. An oral RfD of 1×10^{-2} mg/kg/day was derived by applying an uncertainty factory of 1000 to the LOAEL.

An inhalation reference concentration (RfCs) is not currently available for tetrachloroethene.

<u>Carcinogenic Effects</u>

Studies involving dry-cleaning workers have suggested an increased cancer risk for exposure to PCE. However, because of the presence of possible contributing factors, the studies remain inconclusive (2).

Although the human data are insufficient for determining carcinogenicity, the EPA has classified PCE as a Group B2 (though currently under review) carcinogen-probable human carcinogen. Carcinogenic slope factors (CSFs) have been derived for both the oral and inhalation route of exposure by the Environmental Criteria and Assessment Office (ECAO). An oral CSF of 5.2×10^{-2} $(mg/kg/day)^{-1}$ was derived from a study where mice were exposed via gavage and exhibited a significant increase in hepatocellular carcinomas. The inhalation CSF is 2.03×10^{-3} $(mg/kg/day)^{-1}$ (8).

ENVIRONMENTAL HEALTH EFFECTS

<u>Aquatic</u>

Although ambient water quality criteria for the protection of aquatic organisms were not established, the lowest known exposures to be toxic in aquatic organisms have been reported below (7):

Freshwater:

Acute Toxicity:	5,280 µg/L
Chronic Toxicity:	840 µg/L

Marine:

Acute Toxicity:	10,200 µg/L
Chronic Toxicity:	450 µg/L

As these data are representative of the average toxicity occurrence among freshwater and saltwater aquatic life, it should be noted that toxicity may occur at lower concentrations among species that are more sensitive than those tested (6).

Terrestrial and Avian

No data are available pertaining to the effects of mercury on terrestrial and avian life forms (5).

REGULATORY LEVELS AND CRITERIA

OSHA PEL-TWA:	100 ppm (8-hour TWA) (2)
OSHA Ceiling:	200 ppm (2)
Reportable Quantity (5):	100 lb
ACGIH TLV-TWA (9) :	25 ppm
ACGIH TLV-STEL (9):	100 ppm
Drinking Water Equivalent Level (5):	0.50 mg/L

SUMMARY OF TOXICOLOGICAL INDICES

EPA Carcinogenic Classification (3):	Group B2-Probable human carcinogen
Noncarcinogen Effects:	
Oral RfD (8):	1.0×10^{-2} mg/kg/day
Inhalation RfC (8):	Not Available
Carcinogenic Effects:	
Oral Cancer Slop Factor (8):	5.2×10^{-2} (mg/kg/day)$^{-1}$
Inhalation Cancer Slope Factor (8):	2.03×10^{-3} (mg/kg/day)-1

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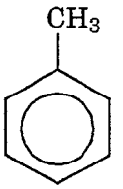
Revisor: Rich Hoff

TOLUENE

INTRODUCTION

Chemical Name: Toluene
CAS Number: 108-88-3
Molecular Formula: $C_6H_5CH_3$
Molecular Weight: 92 g/mole
Chemical Structure:



Toluene is a colorless liquid at standard temperature and pressure. It is primarily used as a component in gasoline but is also used in the synthesis of benzene, methane foams and other organic chemicals (1).

FATE AND TRANSPORT

Log K_{oc} (1): 2.47
Log K_{ow} (2): 2.73
$t_{1/2}$:
- Air: 1.3 days
- Surface Water: 0.17 day

Henry's Law Constant (2): 6.66×10^{-3} atm - m^3/mol
BCF (4): 10.7
Degradation Products (1): Simple hydrocarbons
Solubility:
- In Water (2): 535 mg/L
- In Organics(3,4): Soluble in acetone, ligroin, and carbon disulfide, and miscible with alcohol ether, benzene, chloroform, glacial acetic acid, and other organic solvents

Vapor Pressure (1): 22 mm Hg @ 20°C
Specific Gravity (3): 0.866 @ 20/4°C

Toluene can be present in the air, water, and soil. The low vapor pressure indicates that volatilization is the most probable route of transport among medium.

Photooxidation is the primary fate mechanism for atmospheric toluene. In the atmosphere, it is readily degraded by reacting with hydroxyl radicals resulting in benzaldehyde and cresols. Benzaldehyde is the principal organic product of this atmospheric conversion (1). These rings undergo cleavage to form simple hydrocarbons. Toluene is also oxidized by reacting with nitrogen dioxide, oxygen, and ozone (1).

Volatilization is a primary means of transport from surface water (1). This volatilization from water depends on the turbulence of the water. Toluene in static water has a half-life of one to six days. In turbulent water, the half-life is reduced to five to six hours. In the aquatic environment, toluene can also be oxidized by a reaction similar to that of atmospheric toluene. However, the rate of the reaction is much slower. Degradation of toluene that is not volatilized is primarily through microbial action (1).

The partition coefficients in conjunction with water solubility indicate that toluene would tend to leach into groundwater (1).

The primary route of transport from surface soil is volatilization. The rate of evaporation from soil depends on environmental setting characteristics such as temperature, humidity, and soil type. In general, more than 90 percent of toluene present in soil vaporizes within the first 24 hours. Toluene that has not evaporated from soil is rapidly biodegraded by various bacterial species (1).

Toluene has a low to moderate tendency to bioaccumulate in lipophilic tissues (1). Consequently, bioaccumulation is most likely not a significant environmental fate process (4).

PHARMACOKINETICS

Toluene readily enters the body via inhalation and ingestion and to a lesser degree, by dermal contact. In human volunteers exposed to toluene via inhalation, uptake was found to be rapid. This rapid uptake was demonstrated by a high correlation between the alveolar and arterial concentrations of toluene that were measured during and after exposure (4). Studies of laboratory animals show that absorption following oral exposure to toluene is less rapid than by inhalation. Maximum blood levels following inhalation were reached in fifteen to thirty minutes whereas maximum blood levels after oral exposure were reached in two to three hours after exposure (4).

It has been demonstrated that human dermal absorption of toluene is slow. Two human volunteers soaked areas of their body with toluene for five minutes resulting in a maximum blood concentration of 5.4 µmol/l (4).

Animal studies indicate that once toluene enters the body it tends to accumulate in adipose tissue, bone marrow, body fat, spinal nerves, the spinal cord, and white brain matter. Moderately high concentrations of toluene and its metabolities were found in the liver and kidneys (1).

The majority of toluene absorbed into the body is rapidly metabolized. The metabolic end products include hipparic acid (60-70%); benzoyl glucuronide (10-20%); and o- and, p-cresol (<1%) (1).

Excretion of toluene and its metabolites usually occurs within the first 12 hours after exposure. Metabolic products of toluene are excreted in the urine while unmetabolized toluene is excreted by exhalation (1).

HUMAN HEALTH EFFECTS

Noncarcinogenic

The most profound effect from inhalation exposure to toluene occurs in the central nervous system (CNS). Chronic exposure to moderate and/or high concentrations is associated with

reversible CNS disturbances and impaired neuromuscular function. Irreversible damage has been associated with long-term exposure to high concentrations in solvent users. Human studies indicate that chronic inhalation of toluene can irritate the respiratory tract (1).

Current studies suggest that toluene exposure by inhalation can result in decreased leukocyte counts in the blood. However, this effect in earlier studies was attributable to benzene which is a contaminant in toluene. More recent research indicates that toluene affects the blood but is not the primary target organ (1).

Absorption of toluene can occur through the skin, although this route of exposure is minor compared to inhalation. Direct contact with toluene can moderately irritate skin and eyes (1). No studies were located examining the toxicity of toluene when administered by the oral route (1).

Developmental effects have been observed in animal studies but evidence for human developmental effects is inconclusive. Toluene is not a reproductive toxicant by either inhalation or ingestion (1).

Populations at a greater risk from toluene exposure include those who experience the following: asthma, respiratory difficulties and cardiovascular disease. Additionally, the elderly, cigarette smokers, and chronic alcohol drinkers are more sensitive to toluene exposure (1).

The chronic oral RfD, for toluene, is $2x10^{-1}$ mg/kg/day (5). This value is based on CNS effects, eye and nose irritation, and changes in liver and kidney weights. An uncertainty factor of 1000, based on inter- and intraspecies extrapolations, subchronic to chronic extrapolation, and limited reproductive and developmental toxicity data, was applied to the NOAEL in calculating the RfD.

An inhalation RfD of $1.14x10^{-1}$ mg/kg/day is also available for toluene (5).

Carcinogenic Effects

This chemical is among the substances being evaluated by EPA for evidence of human carcinogenic potential (5).

One animal study showed that dermally administered toluene markedly inhibits skin tumorigenesis in a two stage mouse model utilizing phorbol-12-myristate-13-acetate (PMA) as a promoter. It is speculated from the results of this study that toluene competes for the PMA receptor site; or it may interfere with biochemical processes in cell membranes or in the intercellular cascade between membrane and nucleus (1).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Available data are not adequate to develop ambient water quality criteria. Consequently, EPA has reported the lowest effects level as criteria. These values are as follows:

Freshwater (5,6):

Acute toxicity: 17,500 µg/L
Chronic toxicity: Not available

Marine (5,6):

Acute toxicity: 6,300 µg/L
Chronic toxicity: 5,000 µg/L

Terrestrial and Avian

Information regarding the toxicity of toluene to terrestrial and avian wildlife and domestic animals was not found in the available literature.

REGULATORY LEVELS AND CRITERIA

ACGIH TWA (1):	200 ppm
OSHA TWA (1):	50 ppm
Reportable Quantity (1):	1,000 lbs
MCL:(8):	1 mg/L
MCLG (8):	1 mg/L
Health Advisories:	
1-day:	21.5 mg/L
10 day:	3.46 mg/L
Longer-term (chill):	3.46 mg/L
Lifetime:	2.42 mg/L
Ingestion of Water and Organisms:	14.3 mg/L
Ingestion of Organisms Only:	424 mg/L

SUMMARY OF TOXICOLOGICAL INDICES

EPA Carcinogenic Classification (1):	Group D-Not Classified as to human carcinogenocity
Oral RfD (5):	2×10^{-1} mg/kg/day
Inhalation RfD (5):	1.14×10^{-1} mg/kg/day

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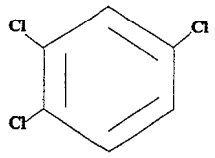
Date of Last Revision: 12/20/94
Revisor: Shirley K. Duffy

1,2,4-TRICHLOROBENZENE

INTRODUCTION

Chemical Name:	1,2,4-Trichlorobenzene
CAS Number (1):	120-82-1
Molecular Formula (2):	$C_6H_3Cl_3$
Molecular Weight (2):	181.45 g/mole
Chemical Structure (2):	



1,2,4-Trichlorobenzene is a colorless liquid with an aromatic odor. The major use of 1,2,4-trichlorobenzene is in the textile industry where it is used as a dye carrier. It is also used in the pesticide industry during the production of dicamba, stirofos and trichlorodinitrobenzene, and in addition, it is used for dielectric and transformer oils. Miscellaneous uses include: degreasing agents, septic tank and drain cleaner formulations, wood preservatives and abrasive formulations used in the manufacture of grinding wheels.

FATE AND TRANSPORT

Absorption Coefficient (log K_{ow}):	3.84 [1]
Octanol/Water Coefficient (log K_{oc}):	4.26 [1]
Solubility:	30 mg/L [1]
Vapor Pressure:	0.42 mm Hg [1]

Transport of 1,2,4-trichlorobenzene through the soil and groundwater system occurs when present at low concentrations, or as a separate organic phase (e.g., resulting from a large quantity spill). The mobility of 1,2,4-trichlorobenzene in the soil/groundwater system (and its eventual migration into aquifers) is strongly affected by the extent of its sorption on soil particles. Sorption on soils is expected to:

- Increase with increasing soil organic matter content
- Increase slightly with decreasing temperature
- Increase moderately with increasing salinity of the soil water, and
- Decrease moderately with increasing dissolved organic matter content of the soil water

Volatilization of 1,2,4-trichlorobenzene tends to occur in near surface soils. Soil porosity, temperature, convection currents, barometric pressure changes, and physiochemical properties (such as Henry's Law Constant) influence the rate of volatilization. In addition, volatilization of 1,2,4-trichlorobenzene from aqueous solutions tends to occur at a relatively rapid rate.

The fate and persistence of 1,2,4-trichlorobenzene has been studied in coastal seawaters. Half-lives calculated for 1,2,4-trichlorobenzene in the marine water column were 22 days in the spring, 11 days in the summer, and 12 days in the winter. Volatilization was identified as the major removal process. The volatilization rates in this environment depend on factors such as depth, turbulence and other environmental conditions.

The persistence of 1,2,4-trichlorobenzene in soil and groundwater systems is not well documented. Once released from the soil into the atmosphere, it will eventually undergo photochemical oxidation; with a half-life in the atmosphere of several days and a residence time of 116 days. Hydrolysis is not expected to be a significant biodegradation pathway.

1,2,4-Trichlorobenzene tends to biodegrade by soil microbes at very slow rates. 1,2,4-Trichlorobenzene was not observed to degrade in groundwater; in addition, biodegradation does not appear to be an important process in seawater.

HUMAN HEALTH EFFECTS

Carcinogenic

The United States Environmental Protection Agency (USEPA), has classified this compound a "D" carcinogen, i.e., it is not classifiable as to human carcinogenicity. No adequate studies are available to assess the possible carcinogenic effects associated with 1,2,4-trichlorobenzene (2).

Noncarcinogenic

The USEPA has developed an oral Reference Dose (RfD) of 1.0×10^{-2} for 1,2,4-trichlorobenzene. The derivation of the oral RfD is based on a multigeneration reproductive rat study. Male and female progeny of the F0 parents were dosed with 0, 25, 100, or 400 parts per million (ppm) of 1,2,4-trichlorobenzene in drinking water. Maternal weights, litter size, neonate sex and weight, and 24-hour food and water intake were recorded. Blood samples and organs were collected on days 27 and 95 of age for chemistry determinations and organ weights. Similar procedures were used with the F1 generation. The study ended when the F2 generation was 32 days old. A Lowest Observed Adverse Effects Level (LOAEL) was derived from a significant increase in adrenal gland weights observed in the 400 ppm groups of males and females of the F0 and F1 generations. The investigators then duplicated the increase in adrenal weights in an acute experiment in which preweanling females were given 3 daily intraperitoneal injections of 1,2,4-trichlorobenzene. The No Observed Adverse Effects Level (NOAEL) was determined to be 100 ppm, and the LOAEL was determined to be 400 ppm on the basis of increased adrenal gland weight and vacuolization of zona fasciculata in the cortex. Additional toxicokinetic studies have shown that initially, adrenal glands have the highest concentration of 1,2,4-trichlorobenzene immediately following oral exposure. Later during the first week following dosing abdominal fat, kidney and liver exhibited higher concentrations than the adrenal gland.

Teratogenic effects were not observed following treatment of rats and mice with 1,2,4-trichlorobenzene, and embryotoxic effects were seen only at concentrations which produced maternal toxicity.

HEAST reports an inhalation reference concentration (RfC) of 2.0×10^{-1} mg/m^3 based on a NOAEL of 104 ppm in a 6 and 26 week study in rats, rabbits, dogs and monkeys. This translates into an inhalation reference dose (RfD) of 5.71×10^{-2} mg/kg/day. The target organ was the liver and the critical effect was non-adverse weight changes (3).

Dermal toxicity studies on rats have shown responses from redness and scaling at a 5% solution of 1,2,4-trichlorobenzene, to severe scaling, encrustation, and desquamation at 25% solutions. No signs of systemic toxicity were observed in this study. Other studies have disputed these findings.

In humans, eye and respiratory tract irritation have been reported following exposure to 3 ppm while a level of 2.4 ppm produced no effects. The only other additional data on human exposure that was available were two individual reports of aplastic anemia. The lethal oral dose has been estimated to be between 50 and 500 milligrams per kilogram for a 70 kilogram person.

ECOLOGICAL HEALTH EFFECTS

Information regarding the toxicity of 1,2,4-trichlorobenzene to aquatic, terrestrial and avian wildlife were not available.

REGULATORY LEVELS AND CRITERIA

Maximum Contaminant Level (MCL):	0.07 mg/L(4)
ACGIH STEL/Ceiling:	C5 ppm/C37 mg/m^3(5)

SUMMARY OF CRITERIA

EPA Carcinogenic Classification (2):	Group D - not classifiable as to human carcinogenicity
Oral RfD (2):	1.0×10^{-2} mg/kg/day
Inhalation RfD (3):	5.71×10^{-2} mg/kg/day

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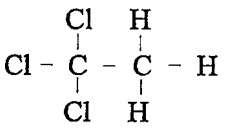
Date of Last Revision: 3/19/93
Revisor: Rich Hoff

1,1,1-TRICHLOROETHANE

INTRODUCTION

Chemical Name:	1,1,1-Trichloroethane
Synonyms:	Methyl chloroform, chloroethene
CAS Number:	71-55-6
Molecular Formula:	$C_2H_3Cl_3$
Molecular Weight:	133.40 g/mole
Chemical Structure:	



1,1,1-Trichloroethane (1,1,1-TCA) exists as a colorless liquid and is a man-made chemical which does not occur naturally. It is insoluble in water but soluble in alcohol and ether. Common uses for 1,1,1-TCA are as a solvent for cleaning precision instruments, metal degreasing, pesticide, and textile processing (1). Approximately 700 million pounds of 1,1,1-TCA were produced in 1987 (2).

FATE AND TRANSPORT

Log K_{oc} (2):	2.03
Log K_{ow} (2):	2.49
$t_{1/2}$:	2-6 years in the atmosphere
Henry's Law Constant (2):	6.3×10^{-3} to 17.2×10^{-3} atm-m^3/mol at 25°C
BCF (2):	9 for bluegill and sunfish
Solubility (1):	
In Water:	Insoluble
In Organics:	alcohol and ether
Vapor Pressure (3):	123 mm Hg at 20°C
Vapor Density (3):	4.63
Specific Gravity (3):	1.34 at 20°C (liquid)

In the environment, 1,1,1-TCA can be found as a liquid, vapor, or dissolved in water and other chemicals (3).

Volatilization into the atmosphere is the dominant environmental fate of 1,1,1-TCA. Once in the air, it reacts slowly with photochemically produced hydroxyl radicals. Because of its long atmospheric half-life, a proportion of 1,1,1-TCA is capable of reaching the stratosphere where degradation can occur by reaction with ozone. The long atmospheric half-life also allows 1,1,1-TCA to be transported long distances from its original point of release. Because 1,1,1-TCA is moderately soluble, precipitation may wash the compound out of the atmosphere. However, the 1,1,1-TCA would be expected to re-volatilize after reaching the land surface (2).

In soil, liquid 1,1,1-TCA has the capability to be highly mobile which results in a potential for groundwater contamination. An important primary fate process is exhibited as volatilization; however, information on biodegradation in the soil is lacking. 1,1,1-TCA does not undergo aerobic biodegradation (2).

Volatilization of 1,1,1-TCA occurs upon its release to surface water. Neither adsorption to sediment nor bioconcentration in aquatic organisms is recognized as an important environmental fate (2).

PHARMACOKINETICS

1,1,1-TCA can enter the body through inhalation of contaminated air, or by drinking water or ingestion of food containing 1,1,1-TCA (2).

If liquid 1,1,1-TCA contacts the skin, rapid evaporation into the atmosphere limits the amount capable of penetrating the skin. Upon inhalation, 60 to 80% 1,1,1-TCA will be eliminated from the body via exchange of air within 2 hours, and 90 to 99% will be eliminated within 50 hours (2).

Upon entering the systemic circulation, 1,1,1-TCA, is metabolized by the body and excreted within a few days either through the urine or exhalation. The major metabolites of 1,1,1-TCA are water soluble trichloroethanol, a trichloroethanol glucuronide conjugate, trichloroacetic acid, and carbon dioxide. If chronic exposure occurs, it is possible for the metabolites to accumulate in the body (2).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Acute exposure to high concentrations of 1,1,1-TCA via inhalation has been found to have lethal effects on humans and animals. Incidences of accidental exposure in humans have indicated that 6,000 to 20,000 ppm 1,1,1-TCA are lethal. Nausea, vomiting, and diarrhea are more common manifestations following acute exposure to 1,1,1-TCA. Mild eye irritation has also been reported from 1,1,1-TCA exposure (2).

The systemic effects on humans consist of central nervous system depression, respiratory arrest or severe cardiac arrhythmia. There is some indication of irreversible toxic effects on the heart following long-term exposure to 1,1,1-TCA. Inhalation may also produce mild hepatic effects (2).

Very high dermal dose levels have killed rabbits. Dermal effects on humans consist of mild irritation to chemical burns, mild erythema, and fine scaling (2).

There have been either no studies conducted or no effects exhibited regarding the effects of 1,1,1-TCA on the respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, or renal systems via oral or dermal exposure. Also, studies involving immunological, neurological, developmental, reproductive, and genotoxic effects have not been conclusive or have produced insignificant results (2).

The cardiovascular, gastrointestinal, hepatic, renal, and ocular systems are affected when inhalation exposure occurs. In addition, neurological effects including mild motor impairments, euphoria, anesthesia, and death are exhibited upon varied levels of inhalation exposure (2).

The EPA has derived both an oral and inhalation RfD for exposure to 1,1,1-TCA. Both the oral and inhalation RfDs were calculated from an inhalation study with male and female guinea pigs at concentrations of 500, 1,000, 2,000, and 10,000 ppm for 7 hours per day, 5 days per week for 6 months. At the three highest concentrations, there was evidence of hepatotoxicity in the exposed animals. A no observed adverse affect level (NOAEL) of 90 and 304 mg/kg/day was identified for the oral and inhalation route, respectively. The oral NOAEL was determined based on a route to route extrapolation. An uncertainty factor of 1,000 was applied to both of the NOAEL values derive the oral RfD dose of 9×10^{-1} mg/kg/day.

U.S. EPA Region III has established an inhalation RfD of 2.86×10^{-1} mg/kg/day for 1,1,1-TCA.

Carcinogenic Effects

The EPA has classified 1,1,1-TCA as a Group D carcinogen - not classifiable as to human carcinogenicity. The basis for this decision is lack of reported human data and in conclusive carcinogenicity data in animal studies (4).

Short-term bioassays have shown evidence of 1,1,1-TCA mutagenicity in Salmonella. A positive result in mammalian cell transformation tests and occasional positive results in other mammalian cell assays suggest that 1,1,1-TCA may be genotoxic in animals (2, 4).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Although data are not adequate for establishing criteria, the EPA has reported the lowest values of 1,1,1-TCA known to be toxic in aquatic organisms. These values are as follows (3):

Freshwater (4):
- Acute toxicity: 18 mg/liter
- Chronic toxicity: 8.4 mg/liter

Marine (4):
- Acute toxicity: 31.2 mg/liter
- Chronic toxicity: No available data

Terrestrial and Avian

Information regarding the toxicity of 1,1,1-TCA to terrestrial and avian wildlife or domestic animals was not available (3).

REGULATORY LEVELS AND CRITERIA

The following regulatory levels and criteria have been established for 1,1,1-TCA:

OSHA (2)
- PEL 350 ppm
- TWA 350 ppm
- STEL 450 ppm

NIOSH (2) IDLH 1000 ppm
Ceiling 350 ppm

Ambient water quality criterion (2):	18 mg/liter
Maximum contaminant level (2):	200 µg/liter
Maximum contaminant level goal (2):	20 µg/liter
Reportable quantity (2):	1,000 pounds

SUMMARY OF TOXICOLOGICAL INDICES

EPA Carcinogenic Classification (5): D-Not classifiable as to human carcinogenicity

Noncarcinogenic Effects:

Oral RfD (5):	9.0×10^{-2} mg/kg/day
Inhalation RfD (8):	2.86×10^{-1} mg/kg/day

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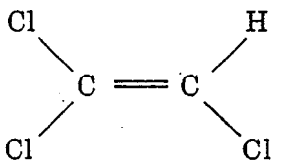
Date of Last Revision: 11/10/94

Revisor: Rich Hoff

TRICHLOROETHENE

INTRODUCTION

Chemical Name: Trichloroethene
CAS Number: 79-01-6
Common Names: Trichloroethene; 1,2,2-Trichloroethylene; TCE
Molecular Formula: C_2HCl_3
Molecular Weight: 131.40 g/mole
Chemical Structure:



Trichloroethene (TCE) is a colorless liquid at standard temperature and pressure. It is a man-made chemical primarily used as a solvent in degreasing operations (1).

FATE AND TRANSPORT

Log K_{oc} (9): 2.10
Log K_{ow}: 2.29 (3)
2.42(1)
$t_{1/2}$ (1): Surface Water: 1-28 days
Henry's Law Constant (1): 0.02 atm-m3/mole @ 20°C
0.01 atm-m3/mole @ 25°C
Fish Bioaccumulation Factor (BCF) (1): 17
Degradation Products:
In Water: Dichloroethylene and vinyl chloride
In Air: Phosgene, dichloroacetyl chloride, and formyl chloride (1); hydrochloric acid, carbon monoxide, carbon dioxide and carboxylic acid (3).
Solubility:
In Water (1): 1.07 g/kg @ 20°C
1,366 mg/L @ 25°C
In Organics (1): Miscible with many common organic solvents such as ether, alcohol and chloroform
Vapor Pressure (1): 59 mm Hg @ 20°C
74 mm Hg @ 25°C
Specific Gravity (1): 1.465 @ 20°C

TCE rapidly volatilizes into the atmosphere. Consequently, the atmosphere is the primary recipient of TCE releases. The average half-life of atmospheric TCE is approximately 3.7 days (1). In the atmosphere, the dominant transformation process is the reaction of TCE with hydroxyl radicals. Various degradation products result from this photooxidation process

including hydrochloric acid, carbon monoxide, carbon dioxide, carboxylic acid, phosgene, dichloroacetyl chloride and formyl chloride (3, 4).

TCE readily volatilizes from surface water into the atmosphere. This is the primary migration pathway of TCE from surface water. Once TCE enters the atmosphere, it degrades by the process described above (1).

Slow degradation of TCE in soil occurs under anaerobic conditions. The Koc value indicates that TCE is quite mobile in soil and will readily leach into groundwater (1).

Once TCE has entered the groundwater, it remains there for years since volatilization is not a viable migration pathway. TCE in groundwater degrades primarily into dichloroethylene. To a lesser degree, it will degrade to vinyl chloride (1). TCE may be present in groundwater as a degradation product of perchloroethylene (4).

TCE, unlike other chlorinated compounds, has a low potential for bioaccumulation in fish, animals, and the food chain (4).

PHARMACOKINETICS

TCE is primarily absorbed into the body via inhalation and ingestion. To a lesser degree, it can be absorbed into the body by direct dermal contact (1).

The primary target organs for inhaled TCE are the central nervous system (CNS), liver, kidney, and hematological system. Principal organs targeted by oral exposure include the liver, kidney, and immunological system (1).

TCE distributes throughout all tissues but tends to concentrate in the fat, kidney, adrenals, vas deferens, epididymis, brain, and liver (4). Once in the body, TCE undergoes extensive metabolism. Numerous metabolites such as trichloroethanol, Trichloroethene-glucuronide, trichloroacetic acid, chloral hydrate (hypnotic agent), Trichloroethene oxide, trichloroacetaldehyde, and chloroacetic acid are generated (1, 4).

There are two primary routes of excretion for TCE. Unmetabolized TCE is excreted by exhalation while TCE metabolites are excreted in the urine (1). Other routes of excretion include sweat, feces, and saliva, but occur to a much lesser extent (4).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Adverse effects to the CNS, kidney, liver, and immune system due to TCE exposure have been documented.

Animal studies show that inhalation of TCE impairs neurological functions such as limb movement control, perception and reaction time. It is expected that larger quantities of TCE by oral exposure are required to create similar effects on the CNS as those experienced by inhalation. This is hypothesized since ingested TCE is subject to first pass elimination by the liver and lungs. It has been demonstrated that the trichloroethanol metabolite is three times as effective as the parent compound in inducing adverse effects on CNS (1). A relationship between exposure and renal effects has also been established in chronic animal studies. Inhalation and/or ingestion of TCE produced histological alterations characterized by renal tubular alterations and/or toxic nephropathy (1). Other chronic studies have identified an association between TCE exposure and deficiencies of the immune system. The effects of TCE

on the immune system are similar to those generated by other chlorinated hydrocarbons (1). A provisional oral reference dose of $6x10^{-3}$ mg/kg/day has been provided by EPA's Environmental Criteria and Assessment Office (ECAO) (7).

It has been documented that acute inhalation exposure to high TCE concentrations have caused severe liver damage. However, it is unlikely that long-term exposure to ambient air concentrations will adversely effect the liver. The most common hepatic effect in TCE-exposed animals is liver enlargement. Histological alterations characterized by cellular hypertrophy were associated with the liver enlargement (1).

Developmental effects from exposure to TCE result from ingestion but not inhalation. Animal studies on the ingestion of TCE indicate that there are reproductive effects such as reduced testis and epididymis weight and sperm mortality (1).

The EPA has not derived an inhalation RfD for TCE.

Carcinogenic Effects

TCE is classified by the EPA as a B2 carcinogen - Probable Human Carcinogen. This means there is sufficient evidence of carcinogenicity in animals; however, evidence of carcinogenicity in humans is inadequate (5).

Animal studies indicate that TCE exposure produced liver and lung tumors in mice and kidney adenocarcinomas, testicular leydig cell tumors, and possibly leukemia in rats (1).

Studies are being conducted to determine if TCE metabolites are involved in cancer development. It is known that trichloroacetic acid and dichloroacetic acid (both metabolites of TCE) are complete carcinogens in the male mouse liver (1). Although the liver appears to be the main site of TCE, there is evidence for extrahepatic TCE metabolism which may be responsible for extrahepatic sites of toxicity.

A provisional inhalation cancer slope factor of 6.0×10^{-3} $(mg/kg/day)^{-1}$ has been provided by the EPA's ECAO (7).

Based on two gavage studies, an oral cancer slope factor of 1.1×10^{-2} $(mg/kg/day)^{-1}$ was derived for TCE (10). B6C3F1 male and female mice were exposed to 0, 1169, or 2339 mg/kg/day and 0, 869 or 1,739 mg/kg/day, respectively, 5 days per week for 8 weeks. The researchers noted a statistically significant increase in hepatocellular carcinomas in male mice at both dose levels and in female mice at the highest dose level. In another study, TCE administration to B6C3F1 male and female mice resulted in an increased incidence of hepatocellular carcinonomas. These results are consistent with those found in the previous B6C3F1 mice cancer bioassay (1).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Although ambient water quality criteria have not been established for TCE, the lowest values known to cause toxicity in aquatic organisms have been reported. These values are as follows (3, 6):

Freshwater:

Acute toxicity:	45,000 µg/L
Chronic toxicity:	Not available

Marine:

Acute toxicity:	2,000 µg/L
Chronic toxicity:	Not available

REGULATORY LEVELS AND CRITERIA

MCLG (1):	0 mg/L
MCL (1):	5 µg/L
OSHA PEL-TWA (8):	100 ppm
ACGIH TLV-TWA (2):	50 ppm
ACGIH TLV-STEL (2):	100 ppm
Reportable Quantity:	1,000 lbs
WQC (6)	
Ingestion of Water and Organisms:	2.7 µg/L
Ingestion of Organisms Only:	8.0 µg/L

SUMMARY OF TOXICOLOGICAL INDICES

EPA Carcinogenic Classification (1):	Group B2-probable human carcinogen
Noncarcinogenic Effects:	
Oral RfD (7):	6.0×10^{-3} mg/kg/day
Inhalation RfC (7):	Not Available
Carcinogenic Effects (7):	
Oral CSF (7):	1.1×10^{-2} (mg/kg/day)$^{-1}$
Inhalation CSF (7):	6.0×10^{-3} (mg/kg/day)$^{-1}$

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Date of Last Revision: 12/15/94
Revisor: Rich Hoff

VANADIUM

INTRODUCTION

Chemical Name: Vanadium
CAS Number (1): 7440-62-2
Molecular Formula (1): V
Molecular Weight (1): 50.94 g/mole

Vanadium is a bright, white, soft ductile metal. It is not found native, but is found in the following ores: patronite, roscoelite, carnotite and vanadinite (1). It can also be found in foods, such as milk, seafoods, cereals, and vegetables (2). Vanadium is used as the target material for x-rays, in the manufacture of alloy steels, and as a catalyst for sulfuric acid and synthetic rubber (1).

FATE AND TRANSPORT

Vanadium is capable of being transported in water systems. The extent of transport is dependant upon the chemical species present and by environmental factors determining its solubility and binding to organic materials. In the atmosphere, vanadium is transported as fumes and particulates. Vanadium does bioaccumulate in humans and animals (3).

PHARMACOKINETICS

Vanadium is moderately absorbed human adipose tissue. Parenteral administration increases levels in the liver and kidney in animals, but these amounts may only be transient. Vanadium can also be found in lung tissue if it is inhaled, but other organs contain negligible amounts. Since vanadium is moderately absorbed, it has been suggested that a homeostatic mechanism maintains the normal levels of vanadium upon excessive exposure. The primary route of excretion is through urine (2).

HUMAN HEALTH EFFECTS

Vanadium has not produced carcinogenic, mutagenic, teratogenic, or reproductive effects in humans or animals. However, industrial exposure to airborne vanadium compounds can cause eye and skin irritation. Also, gastrointestinal distress, nausea, vomiting, abdominal pain, cardiac palpitation, tremor, nervous depression, and kidney damage have been linked with industrial exposure. Oral exposure may produce gastrointestinal disturbances, slight abnormalities of clinical chemistry related to renal function and nervous system effects. Symptoms associated with inhalation exposure to vanadium include acute upper and lower respiratory irritation with mucous discharge and bronchitic, cough, bronchospasm, and chest pain. These effects can occur at concentrations as low as 0.1 mg/m^3 (2). Additionally, studies suggest that the liver, adrenals, and bone marrow may be adversely affected by subacute exposure at high concentrations (2).

The toxicity of vanadium increases as the valence number increases. (Vanadium can be found in the 0, +2, +3, +4, and +5 oxidation states.) A study reported an oral LD_{50} of 130 mg/kg vanadium trioxide (3).

HEAST (4) reports on oral RfD of 7.0 x 10^{-3} mg/kg/day based on a NOAEL of 5 ppm in drinking water administered to rats over a lifetime.

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Ambient water quality criteria have not been established for vanadium. Limited information on the toxicity of vanadium to aquatic organisms has revealed that freshwater fish had 96-hour LC_{50} value ranging from 5,000 to 100,000 µg/L. The majority of the values were approximately 10,000 µg/L. A 96- hour LC_{50} value of less than 0.16 µg/L was reported for daphnids. Chronic toxicity was reported to be around 2,000 µg/L. The lowest value causing an adverse toxic effect was 500 µg/L for a 6-day LC_{50} in a guppy (3).

Terrestrial and Avian

Information regarding the toxicity of vanadium to terrestrial and avian life is very limited. It was reported that calcium vanadate was fatal to chicks fed 200 to 600 ppm for 11 to 32 days (3).

REGULATORY LEVELS AND CRITERIA

OSHA Ceiling Level (5):	0.5 mg/m^3 (respirable dust) 0.1 mg/m^3 (fume)
ACGIH TLV-TWA (6):	0.05 mg/m^3 (vanadium pentoxide, respirable dust or fumes)
Oral RfD (4):	7 x 10^{-3} mg/kg/day

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Date of Last Revision: 11/10/94

Revisor: Patrick B. Moroney

VINYL CHLORIDE

INTRODUCTION

Chemical Name: Vinyl chloride
Synonyms (1): Chloroethane; Ethyl chloride; Hydrochloric ether; Monochloroethane; Muriatic ether
CAS Number (1): 75-00-3
Molecular Formula (1): C_2H_5Cl
Molecular Weight (2): 64.52 g/mole
Chemical Structure (2): $CH_3 - CH_2 - Cl$

Vinyl chloride, a man-made compound, is a colorless gas at room temperature with a sharp odor. However, when placed under pressure, it can exist as a liquid. The largest use of vinyl chloride is to make tetraethyl lead. Because of the phaseout of leaded gasoline, the production of tetraethyl lead has declined. Vinyl chloride is also used as a chemical intermediate in the manufacture of ethyl cellulose plastics, alkyl catalysts, dyes and pharmaceuticals, and as a solvent, aerosol propellant, local anesthetic, and refrigerant (1,2).

FATE AND TRANSPORT

Log K_{ow} (2): 1.43
Log K_{oc} (2): 1.52 (estimated)
$t_{1/2}$: Air (2): 40 days
Surface Water (2): 2.4 hours
Henry's Law Constant (2): 1.11×10^{-2} atm-m^3/mole @ 24.8°C
BCF (1): 1.30 (estimated)
Solubility:
In Water (2): 5,678 mg/L @ 20°C
In Organics (2): Miscible with principal organic solvents
Vapor Pressure (1): 1000 mm Hg @ 20°C
Vapor Density (3): 2.23
Specific Gravity (3): 0.8978 @ 20°C

Vinyl chloride is released into the environment through the following sources: process and fugitive emissions from production; evaporation from wastewater streams, such as solvents, refrigerants, and anesthetics; emissions from plastics, refuse and biomass combustion; and evaporation and leaching from landfills (2).

The dominant removal mechanism for vinyl chloride that is released into the atmosphere is reaction with photochemically-generated hydroxyl radicals. Also, vinyl chloride can be removed from the atmosphere via precipitation. However, the removed vinyl chloride is likely to reenter the atmosphere through volatilization (2).

When released to a water system, vinyl chloride will volatilize. It is not expected to bioconcentrate significantly in aquatic organisms (2).

Releases to the soil will either volatilize rapidly or leach through subsurface soil where it becomes a potential groundwater contaminant. Vinyl chloride would be subject to chemical hydrolysis in moist subsurface soil systems. Studies have indicated that vinyl chloride may be subject to biodegradation in soil under anaerobic conditions (2).

PHARMACOKINETICS

Upon inhalation exposure, vinyl chloride is expected to be readily absorbed through the lungs in humans and animals. This theory is supported by the anesthetic capabilities of vinyl chloride. When dermal contact occurs, vinyl chloride is expected to be easily absorbed. No quantitative studies have been located regarding the absorption of vinyl chloride through inhalation, dermal contact, or ingestion (2).

No studies were located which address the distribution of vinyl chloride in humans following oral, dermal, or inhalation exposure. The only animal study described distribution of vinyl chloride following inhalation exposure. This study reported that the highest concentration of vinyl chloride in the animal body was found in the fatty tissue around the kidney. The lowest amount was found in the cerebrospinal fluid. The brain of the animals accumulated a concentration two times that of the blood (2).

The metabolism of vinyl chloride has not been studied in humans. Limited animal studies have suggested that vinyl chloride does not metabolize to any significant degree in vivo. Slight metabolism of vinyl chloride to ethanol via dechlorination was noted following administration of high anesthetic doses (2).

Vinyl chloride is rapidly excreted through the lungs in humans and animals. It was reported that 30% of the retained dose of vinyl chloride is excreted via exhalation within 1 hour. Studies have reported that excretion in the urine was very slow, approximately 1 hour after inhalation. Studies addressing the excretion of vinyl chloride following oral and dermal exposure in humans and animals were not located in the available literature (2).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Vinyl chloride inhaled at very high concentrations (40,000 ppm) can cause death in humans and animals. Death is caused by effects on the heart, lungs, and nervous system and consist of respiratory paralysis and cardiac depression (2).

Vinyl chloride has had adverse effects on the respiratory, cardiovascular, gastrointestinal, and ocular systems in humans following exposure to very high concentrations for a short time. In humans exposed to 20,000 ppm vinyl chloride, increased respiratory rate, respiratory paralysis, cardiac depression, abdominal cramps and vomiting, and mild eye irritation were reported. In animals, nausea and vomiting, interference with cardiac function, and tissue changes in the heart, lungs, pancreas, intestines, spleen, liver, and kidney were observed. Dermal application may result in frostbite after prolonged exposure due to vinyl chloride's ability to draw heat from the skin (2).

Vinyl chloride does not cause adverse immunological effects in humans or animals following inhalation or oral exposure. However, upon dermal exposure, allergic eczema occurs (2).

When inhaled at high concentrations (33,000 ppm for 15 min), vinyl chloride has a general anesthetic effect. The severity of the anesthetic effect increases with increased concentration

and duration of exposure. This is made possible by vinyl chloride's ability to produce structural changes in proteins or lipoprotein structures. Recovery from effects are rapid.

U.S. EPA has classified vinyl chloride as a Class A - human carcinogen. Water and air unit risks of 5.4×10^{-5} per µg/L and 8.5×10^{-5} per µg/m^3 have been promulgated (5). From these unit risks, oral and inhalation carcinogenic potency estimates of 1.9 and 0.3 $(mg/kg/d)^{-1}$ can be established.

Studies have shown that vinyl chloride produces reversible structural changes in globular proteins in vitro. Some studies suggest transient signs and symptoms of cerebellar dysfunction and possible autonomic nervous system dysfunction in humans (2).

In view of the paucity of data available on the toxicity associated with exposure to vinyl chloride estimates of exposure levels of concern can not be made with any confidence.

Carcinogenic Effects

Vinyl chloride was tested by the National Toxicology Program for carcinogenicity and genetic toxicity in both rats and mice via inhalation (3). The study has shown that 86% of female mice chronically exposed to vinyl chloride vapors developed highly malignant uterine carcinomas. Also, the incidence of hepatocellular carcinomas increased significantly. Male mice had an increased incidence of alveolar and bronchiolar adenomas. This study provides equivocal evidence for the carcinogenicity of vinyl chloride in mice (2).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Specific information addressing the toxicity to aquatic organisms has not been established. However, it should be noted that freshwater and marine data indicates that toxicity increases greatly with increasing chlorination. Therefore, vinyl chloride is probable less toxic than 1,2-divinyl chloride, trivinyl chlorides, tetravinyl chlorides, pentavinyl chloride, and hexavinyl chloride (4).

Terrestrial and Avian

Information regarding the toxicity of vinyl chloride to terrestrial and avian wildlife or domestic animals was not located in the available literature (3).

REGULATORY LEVELS AND CRITERIA

OSHA PEL - TWA (7):	1 ppm
OSHA PEL - C (7):	5 ppm
ACGIH TLV - TWA (6):	5 ppm
Reportable Quantity (2):	100 lbs

SUMMARY OF TOXICOLOGICAL INDICES (5)

Unit Risk (Water):	5.4×10^{-5} per µg/L
Oral Cancer Slope Factor (8):	1.9 $(mg/kg/d)^{-1}$
Unit Risk (Air):	8.5×10^{-5} per µg/m^3
Inhalation Cancer Slope Factor (8):	0.3 $(mg/kg/d)^{-1}$

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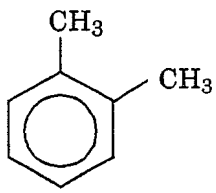
Revisor: Rich Hoff

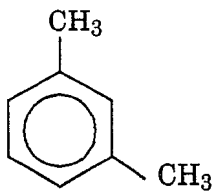
XYLENES (MIXED)

INTRODUCTION

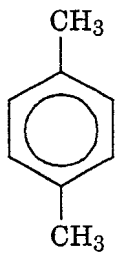
Chemical Name: Xylenes (Mixed)
CAS Number: 1330-20-7
Molecular Formula: $C_6H_4(CH_3)_2$
Molecular Weight: 106.17 g/mole
Chemical Structure: Mixed xylenes are composed of a combination of the following three isomeric forms:



ortho



meta



para

Mixed xylenes (hereinafter "xylenes") consist of a clear liquid mixture of the ortho, meta, and para isomers of the compound. Xylenes are a common organic solvent used especially as an intermediate for dyes and inorganic synthesis. Xylenes also are used in formulating various aviation fuels (2).

FATE AND TRANSPORT

Log K_{ow} (4): 3.12-3.20
K_{oc} (4): 166
$t_{1/2}$ (5): 100% reduction in 192 hrs in incubated conditions
Henry's Law Constant (4): 5.19×10^{-3} to 7.6×10^{-3} atm-m^3/mol
BCF (4): 2.14-23.6
Degradation Products (5): Toluic Acid, Catechol
Solubility:
    In Water (4): 30 ppm @ 25° C
    In Organics (4): Miscible with alcohol, ether and other organics
Vapor Pressure (4): 6.16 mm Hg @ 20° C
Specific Gravity (1): 0.86

Xylenes are volatile compounds which ultimately partition into the atmosphere. Their vapor pressures and Henry's Law Constants are indicative of compounds that will tend to migrate fairly rapidly from both surface waters and soils into the air. Xylenes in the atmosphere tend

to undergo photooxidation. Direct photolysis is not believed to be a significant degradation process (4).

Spilled xylenes in soil tend to be relatively mobile in subsurface environments by virtue of their water solubility. Because of their ability to desiccate clays, xylenes can open macropores in this soil type thereby further enhancing their transport potential (4).

Xylenes in groundwater and sediments tend to undergo biodegradation. Hydrolysis and oxidation are not significant transformation processes for xylenes in the aquatic environment. Xylenes tend to marginally bioaccumulate in various aquatic species with bioconcentration factors ranging from 2.14 to 23.6 (4).

PHARMACOKINETICS

Xylenes can be absorbed into the systemic circulation following exposure via ingestion, inhalation or dermal contact (4).

Xylenes are most readily absorbed following inhalation and are rapidily absorbed into the circulation via this pathway. The majority of inhaled xylenes are distributed, initially, with the serum proteins with the remainder distributed in the other elements of the serum. Xylenes are ultimately distributed to the adipose tissue (4).

Limited information is available regarding the distribution of xylenes following oral exposure. However, metabolites of xylenes have been shown to be distributed throughout various tissues within two hours of ingestion in experimental animals (4).

Dermal absorption of xylenes has not been quantified (4).

The metabolism and elimination of xylenes are primarily mediated through the oxidation of a side-chain methyl group via microsomal enzymes in the liver to yield toluic acid. These acids are subsequently conjugated with glycine to form methylhippuric acids which are excreted in the urine. This metabolic pathway accounts for the elimination of nearly all absorbed xylenes (4).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

Short-term exposure to high levels of xylenes can cause irritation of the skin, eyes, nose and throat, increased reaction time to visual stimuli, impared memory, stomach discomfort, and changes in the liver and kidney. Both short and long-term exposure can result in a number of effects on the nervous system including headaches, dizziness, confusion, and changes in the sense of balance (4).

EPA has identified a no observed adverse effect level (NOAEL) for xylenes of 179 mg/kg/day. An uncertainty factor of 100 was applied to the NOAEL to account for interspecies extrapolation and for the protection of sensitive individuals to derive an oral RfD of 2.0 mg/kg/day. The RfD is based on hyperactivity, decreased body weight and increased mortality. EPA has given the RfD a confidence base of "medium", since it was derived from a well-designed study in which adequately sized groups of two species were tested over a substantial portion of their lifespan, comprehenisve histopathology was performed, and a NOAEL was defined (3). An inhalation RfD value for mixed xylenes has not been verified. An uncertainty factor of 100 was applied to the NOAEL (27 mg/m^3).

Carcinogenic Effects

EPA has classified xylene mixtures as a Group D carcinogen - not classifiable as to human cancinogenicity. Orally administered xylenes did not result in significant increases in tumor responses in rats or mice of both sexes (3). There is no evidence available regarding xylenes as human carcinogens.(3)

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

Xylenes have been shown to adversely affect trout populations at concentrations as low as 3.6 mg/L in a continuous flow-through system. The LC_{50} in trout was determined to be 13.5 mg/L. The LC_{50}s for other freshwater species were approximately 30 mg/L. Saltwater species are generally more susceptible to xylene than are freshwater species (1).

Terrestrial and Avian

No information on the toxicity of xylenes to terrestrial wildlife and domestic animals was documented in readily available literature. However, because of the low acute toxicity of xylenes, it is unlikely that they would be toxic to wild or domestic birds and animals under most environmental exposure situations (1).

REGULATORY LEVELS AND STANDARDS

Reportable Quantity (3)	1,000 lbs
MCLG (total) (7)	10 mg/L
OSHA TWA (4):	100 ppm
ACGIH TVL - TWA (4)	100 ppm
TVL - STEL (4):	150 ppm

SUMMARY OF CRITERIA

EPA Carcinogenic Classification(3)	D-Not classifiable as to human carcinogenicity
Oral RfD (3)	2.0 mg/kg/day
Inhalation RfC (6)	Unverifiable

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Revisor: Patrick B. Moroney

ZINC

INTRODUCTION

Chemical Name: Zinc
Synonyms (1): Zinc dust; Zinc powder
Trade Names (1,2): Asarco; Blue powder; CI 77945; JASAD; PASCO
CAS Number (1): 7440-66-6
Molecular Formula (1): Zn
Molecular Weight (1): 65.38 g/mole
Chemical Structure (1): Zn

Zinc is a shiny white metal with a bluish-gray luster that exhibits a valence of +2 and has five stable isotopes. It is derived from the pyrometallurgical or distillation process, or the hydrometallurgical or electrolytic process. Among zincs uses are as an alloy in brass, bronze, and die-casting alloys; galvanizing iron; as a fungicide; and as a protective coating for other metals (3).

FATE AND TRANSPORT

Density (1): 7.14
Solubility (1):
In Water: Insoluble
In Organics: Soluble in acetic acid and alkali

Zinc occurs in the environment in the +2 oxidation state in both the suspended and dissolved forms. Sorption to other materials is the dominant environmental fate of zinc. The predominant fate of zinc in an aerobic aquatic system is the sorption of the divalent cation by hydrous iron and manganese oxide, clay minerals, and organic material. The efficiency of these materials in removing zinc from solution varies according to their compositions and concentrations, the pH and salinity of the water (1).

Zinc concentrations in air are relatively low and fairly constant except near industrial sources such as smelters (1).

Zinc is strongly bioaccumulated even in the absence of abnormally high ambient concentrations although it does not appear to biomagnify. Although zinc is actively bioaccumulated in aquatic systems, the biota appear to represent a relatively minor sink compared to the sediments (8).

PHARMACOKINETICS

The efficiency with which zinc is absorbed following ingestion has been reported to be approximately 80 percent (4). Information was not available on zinc absorption via other routes of exposure. Zinc is primarily distributed to the prostate gland, liver, bone, muscle, kidney, and pancreas following absorption.

During initial absorption, zinc binds to carrier macromolecules by a process influenced by prostaglandins E2 and F2 and chelated by the tryptophan derivative picolinic acid. Zinc induces metallothionein synthesis in mucosal cells. Zinc also binds to metallothionein in

other tissues including liver, salivary glands, intestinal mucosa, and pancreas. In blood, zinc binds principally to albumin and beta-2-macroglobulin proteins. It also binds to the enzyme acid phosphatase which is most abundant in the prostate gland. Binding with metallothionein may be followed by release into the gastrointestinal tract via the salivary glands, intestinal mucosa, pancreas, and liver (5). Urinary excretion constitutes an important route and is correlated quantitatively with dietary zinc intake (5). However, the major route of elimination is in feces. (6).

HUMAN HEALTH EFFECTS

Noncarcinogenic Effects

In humans, death has been exhibited following acute inhalation of zinc chloride. The concentration of zinc chloride was unknown and other chemicals were detected. Therefore, death can not be exclusively attributed to zinc exposure. Death was reported in ferrets and mice following acute and intermediate oral exposure. However, no animal studies addressing the effects of zinc following inhalation exposure were located (1).

The adverse effects of zinc following acute inhalation exposure were limited to the respiratory tract of both humans and animals. Following oral exposure, zinc has been shown to cause adverse effects on the gastrointestinal system. The hematological and renal systems are also affected following acute, intermediate, and chronic exposures in humans and animals (1).

Zinc has been found to reduce the serum HDL-cholesterol levels in humans following intermediate oral exposure. The decrease in HDL levels may increase risk of coronary artery disease (1).

Zinc is essential to maintain one's health. However, ingestion of high levels of zinc present a potential for gastrointestinal disorders in humans and animals such as pancreatic abnormalities and gastrointestinal irritation. Biochemical changes (increased serum amylase levels and hypocalcemia) were evident in humans following ingestion of a zinc chloride solution. Animal studies have shown adverse effects including pancreatic fibrosis and degeneration and necrosis of acinar cells of the pancreas. Zinc has also caused internal bleeding in humans and animals following ingestion of high levels (1).

Respiratory disorders have been observed in humans and animals following acute inhalation exposure to zinc compounds. Acute exposure to high concentrations of zinc oxide in humans cause metal fume fever, and penetrates the alveoli, damages lung tissue and transiently impairs pulmonary function. The same effects have been exhibited in animals. Zinc compounds such as zinc chloride cause more damage to respiratory tissue following inhalation exposure than other compounds (1).

Hematological effects in humans and animals following high level acute, intermediate or chronic oral exposure to zinc and zinc compounds consist of anemia which can be exacerbated by gastrointestinal bleeding. These effects occur over a wide range of concentrations depending upon the species (1).

No adverse hepatic effects were reported following intermediate oral exposure to zinc in humans. This indicates that the liver is not a primary target organ for zinc toxicity. Hepatic necrosis was reported in sheep following acute and intermediate exposure, and enzymatic changes where exhibited in rats exposed following intermediate exposure to zinc (1).

The only renal effects observed were in laboratory animals following intermediate oral exposure to zinc compounds. No adverse renal effects have occurred in humans (1).

In vitro studies suggest that neurological symptoms and lethargy can occur in humans following oral administration of zinc. These studies indicate that zinc inhibits the entry of calcium ions into the nerve terminals, which influences the release of neurotransmitters. Additionally, zinc has been known to be toxic to neurons and glia cells of the central nervous system (1).

Zinc has retarded fetal growth and altered fetal and maternal concentrations of zinc and copper in rats. Congenital malformations such as exencephaly and rib fusions have been exhibited in offspring of pregnant hamsters injected intravenously with zinc sulfate. There is no evidence of these effects occurring in humans (1).

The EPA has established an oral RfD value of 3.0×10^{-1} mg/kg/day for zinc. This value is based on a LOAEL of 1 mg/kg/day in human females. Zinc exposure affected the blood as manifested by decreased concentration of the blood enzyme erythrocyte superoxide dismutase (ESOD) (9).

Carcinogenic Effects

Zinc has not been shown to be carcinogenic. Therefore, the EPA has not assigned a carcinogenicity classification of D for zinc (1).

Although studies have failed to provide evidence for mutagenicity of zinc, there are indications that zinc is a weak clastogen. Human studies involving the treatment of human lymphocytes in cultures with zinc have shown a slight increase in chromosomal aberrations (1).

ENVIRONMENTAL HEALTH EFFECTS

Aquatic

The EPA has established Ambient Water Quality Criteria for zinc in order to protect freshwater and marine aquatic life. The criteria are as follows (7):

Freshwater:
- Acute toxicity: 120* µg/L
- Chronic toxicity: 110 µg/L*

Saltwater:
- Acute toxicity: 95 µg/L
- Chronic toxicity: 86 µg/L

*Hardness dependent criteria.

Terrestrial and Avian

Information regarding the toxicity of zinc to terrestrial and avian wildlife or domestic animals was not available.

REGULATORY LEVELS AND CRITERIA

SMCL (8):	5 mg/L
Reportable Quantity (1):	1,000 lbs
Permissible Level in bottled water (1):	5.0 mg/L
OSHA Standard PEL TWA (10):	5 mg/m^3 (zinc oxide fume/zinc oxide dust, respirable fraction) 15 mg/m^3 (zinc oxide, total dust)

ACGIH TLV (11):

Compound	TWA	STEL
Zinc chloride fume:	1 mg/m^3	2 mg/m^3
Zinc oxide fume:	5 mg/m^3	10 mg/m^3
Zinc oxide dust:	10 mg/m^3	--
Zinc chromates:	0.01 mg/m^3	--

SUMMARY OF CRITERIA

Oral RfD (9):	3.0×10^{-1} mg/kg/day
NOAEL (9):	2.14 mg/kg/day
Uncertainty Factor (9):	10

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APPENDIX D - RISK CALCULATIONS

APPENDIX D
RISK CALCULATION SPREADSHEETS

TABLE D-1
LOCAL RESIDENTS - RESIDENTIAL AREA - CURRENT SCENARIO
ACCIDENTAL INGESTION OF GROUNDWATER FROM DOMESTIC WELLS[1]
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

*CDI (mg/kg/d)= (Cw*IR*ET*EF*ED)/(BW*AT)*
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Local Adult Resident	Local Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/d)	0.05	0.05	
ET	Exposure time (hours/event)	2.6	2.6	
EF	Exposure Frequency (events/yr)	28	28	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.038	0.091	NA	2.3E-06	2.1E-07	86.9%	5.4E-06	--	--	2.2E-06	2.0E-07	86.9%	2.5E-05	--	--
Tetrachloroethene	0.0100	0.052	0.01	6.1E-07	3.2E-08	13.1%	1.4E-06	1.4E-04	100.0%	5.7E-07	3.0E-08	13.1%	6.6E-06	6.6E-04	100.0%
				Total ILCR:	2.4E-07	100.0%	HI:	1.4E-04	100.0%	Total ILCR:	2.3E-07	100.0%	HI:	6.6E-04	100.0%

NOTES:
[1] Groundwater drawn from wells are used for non-potable purposes, e.g., swimming.
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-2
LOCAL RESIDENTS - RESIDENTIAL AREA - CURRENT SCENARIO
DERMAL CONTACT WITH GROUNDWATER FROM DOMESTIC WELLS[1]
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [2])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Local Adult Resident	Local Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	20000	8023	
EF	Exposure frequency (days)	28	28	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/event	CS	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	2.6	2.6	

							Adult Residents						Child Residents (0 - 6 Yrs Old)					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.038	5.3E-03	3.9E-03	1.5E-07	0.091	NA	1.4E-06	1.3E-07	31.4%	3.2E-06	--	0.0%	5.2E-07	4.7E-08	31.4%	6.0E-06	--	0.0%
Tetrachloroethene	0.0100	4.8E-02	5.6E-02	5.6E-07	0.052	0.01	5.3E-06	2.7E-07	68.6%	1.2E-05	1.2E-03	100.0%	2.0E-06	1.0E-07	68.6%	2.3E-05	2.3E-03	100.0%
								Total ILCR:	4.0E-07	100.0%	HI:	1.2E-03	100.0%	Total ILCR:	1.5E-07	100.0%	HI:	2.3E-03	100.0%

NOTES:
[1] Groundwater drawn from wells are used for non-potable purposes, e.g., swimming.
[2] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-3
LOCAL RESIDENTS - AREA A- CURRENT TRESPASSING SCENARIO
ACCIDENTAL INGESTION OF SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*EF*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Local Adult Resident	Local Child Resident (6 - 15 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/hour)	0.05	0.05	
ET	Exposure time (hours/event)	2.6	2.6	
EF	Exposure Frequency (events/yr)	7	28	
ED	Exposure Duration (yrs)	30	9	
BW	Body weight (kg)	70	37	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	9125	3285	

				Adult Residents						Child Residents (6 - 15 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Tetrachloroethene	0.0053	0.052	0.01	8.1E-08	4.2E-09	0.5%	2.3E-07	2.3E-05	0.3%	1.8E-07	9.6E-09	0.5%	1.4E-06	8.2E-05	0.3%
Trichloroethene	0.0096	0.011	0.006	1.5E-07	1.6E-09	0.2%	4.1E-07	6.8E-05	0.8%	3.3E-07	3.7E-09	0.2%	2.6E-06	4.9E-05	0.2%
Vinyl chloride	0.0053	1.9	NA	8.1E-08	1.5E-07	17.8%	2.3E-07	--	--	1.8E-07	3.5E-07	17.8%	1.4E-06	--	--
Xylenes(total)	0.003	NA	2	4.6E-08	--	--	1.3E-07	6.4E-08	0.0%	1.0E-07	--	--	8.1E-07	1.6E-02	58.5%
Bis(2-ethylhexyl)phthalate	0.003	0.014	0.02	4.6E-08	6.4E-10	0.1%	1.3E-07	6.4E-06	0.1%	1.0E-07	1.5E-09	0.1%	8.1E-07	1.6E-04	0.6%
4,4'-DDD	0.00013	0.24	NA	2.0E-09	4.7E-10	0.1%	5.5E-09	--	--	4.5E-09	1.1E-09	0.1%	3.5E-08	--	--
4,4'-DDE	0.000056	0.34	NA	8.6E-10	2.9E-10	0.0%	2.4E-09	--	--	1.9E-09	6.6E-10	0.0%	1.5E-08	--	--
alpha-Chlordane	0.000015	1.3	0.00006	2.3E-10	3.0E-10	0.0%	6.4E-10	1.1E-05	0.1%	5.2E-10	6.8E-10	0.0%	4.0E-09	4.9E-07	0.0%
Aroclor-1254	0.00044	7.7	0.00002	6.7E-09	5.2E-08	6.0%	1.9E-08	9.4E-04	11.4%	1.5E-08	1.2E-07	6.0%	1.2E-07	1.6E-07	0.0%
delta-BHC	0.000025	1.8	NA	3.8E-10	6.9E-10	0.1%	1.1E-09	--	--	8.7E-10	1.6E-09	0.1%	6.7E-09	--	--
Dieldrin	0.000027	16	0.00005	4.1E-10	6.6E-09	0.8%	1.2E-09	2.3E-05	0.3%	9.4E-10	1.5E-08	0.8%	7.3E-09	4.1E-07	0.0%
gamma-Chlordane	0.000024	1.3	0.00006	3.7E-10	4.8E-10	0.1%	1.0E-09	1.7E-05	0.2%	8.3E-10	1.1E-09	0.1%	6.5E-09	4.9E-07	0.0%
Heptachlor epoxide	0.000006	9.1	0.000013	9.2E-11	8.3E-10	0.1%	2.6E-10	2.0E-05	0.2%	2.1E-10	1.9E-09	0.1%	1.6E-09	1.1E-07	0.0%
Aluminum	6.85	NA	1	1.0E-04	--	--	2.9E-04	2.9E-04	3.6%	2.4E-04	--	--	1.8E-03	8.2E-03	29.3%
Arsenic	0.024	1.75	0.0003	3.7E-07	6.4E-07	74.4%	1.0E-06	3.4E-03	41.8%	8.4E-07	1.5E-06	74.4%	6.5E-06	2.5E-06	0.0%
Barium	0.165	NA	0.07	2.5E-06	--	--	7.1E-06	1.0E-04	1.2%	5.7E-06	--	--	4.5E-05	5.8E-04	2.0%
Lead	0.261747	NA	NA	4.0E-06	--	--	1.1E-05	--	--	9.1E-06	--	--	7.1E-05	--	--
Manganese	0.371	NA	0.005	5.7E-06	--	--	1.6E-05	3.2E-03	38.5%	1.3E-05	--	--	1.0E-04	4.1E-05	0.1%
Mercury	0.0013	0.0003	NA	2.1E-08	6.2E-12	0.0%	5.8E-08	--	--	4.7E-08	1.4E-11	0.0%	3.6E-07	--	--
Silver	0.00459	NA	0.005	7.0E-08	--	--	2.0E-07	3.9E-05	0.5%	1.6E-07	--	--	1.2E-06	4.1E-05	0.1%
Zinc	0.620	NA	0.3	9.5E-06	--	--	2.6E-05	8.8E-05	1.1%	2.1E-05	--	--	1.7E-04	2.5E-03	8.8%
				Total ILCR:	8.7E-07	100.0%		HI:	8.2E-03	100.0%	Total ILCR:	2.0E-06	100.0%	HI:	2.8E-02	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-4
LOCAL RESIDENTS - AREA A- CURRENT TRESPASSING SCENARIO
DERMAL CONTACT WITH SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Local Adult Resident	Local Child Resident (6 - 15 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	5300	3683	
EF	Exposure frequency (d/yr)	7	28	
ED	Exposure duration (yrs)	30	9	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	37	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/event)	CS	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	2.6	2.6	

							Adult Residents						Child Residents (6 - 15 Yrs Old)					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Tetrachloroethene	0.0053	0.052	2.0E-01	1.1E-06	0.052	0.01	6.7E-07	3.5E-08	0.1%	1.6E-06	1.6E-04	6.7%	1.1E-06	5.5E-08	0.1%	8.3E-06	8.3E-04	6.7%
Trichloroethene	0.0096	0.011	5.9E-02	5.7E-07	0.011	0.006	3.5E-07	3.9E-09	0.0%	8.2E-07	1.4E-04	5.9%	5.8E-07	6.1E-09	0.0%	4.3E-06	7.2E-04	5.9%
Vinyl chloride	0.0053	1.9	2.2E-02	1.2E-07	1.9	NA	7.3E-08	1.4E-07	0.5%	1.7E-07	--	--	1.2E-07	2.2E-07	0.5%	9.0E-07	--	--
Xylenes(total)	0.003	NA	2.0E-01	6.1E-07	NA	2	3.8E-07	--	--	8.8E-07	4.4E-07	0.0%	6.0E-07	--	--	4.6E-06	2.3E-06	0.0%
Bis(2-ethylhexyl)phthalate	0.003	3.3E-02	6.7E-01	2.0E-06	0.014	0.02	1.3E-06	1.8E-08	0.1%	2.9E-06	1.5E-04	6.3%	2.0E-06	2.8E-08	0.1%	1.5E-05	7.7E-04	6.3%
4,4'-DDD	0.00013	2.8E-01	3.5E+00	4.5E-07	0.24	NA	2.8E-07	6.7E-08	0.3%	6.5E-07	--	--	4.4E-07	1.1E-07	0.3%	3.4E-06	--	--
4,4'-DDE	0.000058	2.4E-01	2.9E+00	1.7E-07	0.34	NA	1.0E-07	3.5E-08	0.1%	2.4E-07	--	--	1.6E-07	5.5E-08	0.1%	1.3E-06	--	--
alpha-Chlordane	0.000015	4.6E-02	1.1E+00	1.6E-08	1.3	0.00006	1.0E-08	1.3E-08	0.1%	2.4E-08	3.9E-04	16.8%	1.6E-08	2.1E-08	0.1%	1.2E-07	2.1E-03	16.8%
Aroclor-1254	0.00044	3.7E-01	1.2E+01	5.3E-06	7.7	NA	3.3E-06	2.5E-05	98.1%	7.6E-06	--	--	5.2E-06	4.0E-05	98.1%	4.0E-05	--	--
delta-BHC	0.000025	2.6E-02	2.6E-01	6.6E-09	1.8	NA	4.1E-09	7.4E-09	0.0%	9.6E-09	--	--	6.5E-09	1.2E-08	0.0%	5.1E-08	--	--
Dieldrin	0.000027	1.6E-02	3.0E-01	8.2E-09	16	0.00005	5.1E-09	8.1E-08	0.3%	1.2E-08	2.4E-04	10.1%	8.0E-09	1.3E-07	0.3%	6.2E-08	1.2E-03	10.1%
gamma-Chlordane	0.000024	4.6E-02	1.1E+00	2.6E-08	1.3	0.00008	1.6E-08	2.1E-08	0.1%	3.8E-08	6.3E-04	26.9%	2.6E-08	3.3E-08	0.1%	2.0E-07	3.3E-03	26.9%
Heptachlor epoxide	0.000008	6.7E-04	1.4E-02	8.1E-11	9.1	0.000013	5.0E-11	4.6E-10	0.0%	1.2E-10	9.1E-06	0.4%	8.0E-11	7.2E-10	0.0%	6.2E-10	4.8E-05	0.4%
Aluminum	6.85	1.0E-03	NA	1.8E-05	NA	1	1.1E-05	--	--	2.6E-05	2.6E-05	1.1%	1.7E-05	--	--	1.4E-04	1.4E-04	1.1%
Arsenic	0.024	1.0E-03	NA	6.3E-09	1.75	0.0003	3.9E-08	6.8E-08	0.3%	9.1E-08	3.0E-04	13.0%	6.2E-08	1.1E-07	0.3%	4.8E-07	1.6E-03	13.0%
Barium	0.165	1.0E-03	NA	4.3E-07	NA	0.07	2.7E-07	--	--	6.2E-07	8.9E-06	0.4%	4.2E-07	--	--	3.3E-06	4.7E-05	0.4%
Lead	0.261747	1.0E-03	NA	6.8E-07	NA	NA	4.2E-07	--	--	9.9E-07	--	--	6.7E-07	--	--	5.2E-06	--	--
Manganese	0.371	1.0E-03	NA	9.6E-07	NA	0.005	6.0E-07	--	--	1.4E-06	2.8E-04	12.0%	9.5E-07	--	--	7.4E-06	1.5E-03	12.0%
Mercury	0.0013	1.0E-03	NA	3.5E-09	0.0003	NA	2.2E-09	6.6E-13	0.0%	5.1E-09	--	--	3.4E-09	1.0E-12	0.0%	2.7E-08	--	--
Silver	0.00459	1.0E-03	NA	1.2E-08	NA	0.005	7.4E-09	--	--	1.7E-08	3.5E-06	0.1%	1.2E-08	--	--	9.1E-08	1.8E-05	0.1%
Zinc	0.620	1.0E-03	NA	1.6E-06	NA	0.3	1.0E-06	--	--	2.3E-06	7.8E-06	0.3%	1.6E-06	--	--	1.2E-05	4.1E-05	0.3%
							Total ILCR:	2.6E-05	100.0%	HI:	2.3E-03	100.0%	Total ILCR:	4.0E-05	100.0%	HI:	1.2E-02	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from *Dermal Exposure Assessment: Principles and Applications, Interim Report*, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-5
LOCAL RESIDENTS - AREA A- CURRENT TRESPASSING SCENARIO
ACCIDENTAL INGESTION OF SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d) (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Local Adult Resident	Local Child Resident (6 - 15 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	100	200	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
FI	Fraction of sediment ingested from site	1	1	
EF	Exposure Frequency (d/yr)	7	28	
ED	Exposure Duration (yrs)	30	9	
BW	Body weight (kg)	70	37	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	

				Adult Residents						Child Residents (6 - 15 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.955	7.7	NA	1.1E-08	8.6E-08	3.4%	2.6E-08	--	--	5.1E-08	3.9E-07	3.4%	4.0E-07	--	--
4,4'-DDE	0.103	0.34	NA	1.2E-09	4.1E-10	0.0%	2.8E-09	--	--	5.5E-09	1.9E-09	0.0%	4.3E-08	--	--
4,4'-DDT	0.049	0.34	NA	5.7E-10	1.9E-10	0.0%	1.3E-09	--	--	2.6E-09	8.8E-10	0.0%	2.0E-08	--	--
Arsenic	118	1.75	0.0003	1.4E-06	2.4E-06	96.5%	3.2E-06	1.1E-02	60.4%	6.3E-06	1.1E-05	96.5%	4.9E-05	1.6E-01	60.4%
Cadmium	49.6	NA	0.0005	5.7E-07	--	--	1.3E-06	2.7E-03	14.9%	2.6E-06	--	--	2.0E-05	4.0E-02	14.9%
Chromium	568	NA	0.005	6.9E-06	--	--	1.6E-05	3.2E-03	18.1%	3.1E-05	--	--	2.4E-04	4.9E-02	18.1%
Copper	553	NA	0.0371	6.5E-06	--	--	1.5E-05	4.1E-04	2.3%	2.9E-05	--	--	2.3E-04	6.2E-03	2.3%
Lead	299	NA	NA	3.5E-06	--	--	8.2E-06	--	--	1.6E-05	--	--	1.2E-04	--	--
Manganese	51.2	NA	0.005	6.0E-07	--	--	1.4E-06	2.8E-04	1.6%	2.7E-06	--	--	2.1E-05	4.2E-03	1.6%
Mercury	0.770	NA	0.0003	9.0E-09	--	--	2.1E-08	7.0E-05	0.4%	4.1E-08	--	--	3.2E-07	1.1E-03	0.4%
Silver	19.5	NA	0.005	2.3E-07	--	--	5.3E-07	1.1E-04	0.6%	1.0E-06	--	--	8.1E-06	1.6E-03	0.6%
Vanadium	77.1	NA	0.007	9.0E-07	--	--	2.1E-06	3.0E-04	1.7%	4.1E-06	--	--	3.2E-05	4.6E-03	1.7%
				Total ILCR:	2.5E-06	100.0%	HI:	1.8E-02	100.0%	Total ILCR:	1.1E-05	100.0%	HI:	2.7E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-6
LOCAL RESIDENTS - AREA A- CURRENT TRESPASSING SCENARIO
DERMAL CONTACT WITH SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Local Adult Resident	Local Child Resident (6 - 15 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	1	
ABS	Absorption fraction --			
	Cadmium and default organics:	0.01	0.01	
	Default inorganics:	0.001	0.001	
	PCBs:	0.06	0.06	
A	Skin surface area available for contact (cm2)	5300	3683	
EF	Exposure Frequency (d/yr)	7	28	
ED	Exposure Duration (yrs)	30	9	
BW	Body weight (kg)	70	37	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	

					Adult Residents						Child Residents (6 - 15 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.955	0.06	7.7	NA	3.6E-08	2.7E-07	6.6%	8.3E-08	--	--	5.6E-08	4.3E-07	6.6%	4.4E-07	--	--
4,4'-DDE	0.103	0.1	0.34	NA	6.4E-09	2.2E-09	0.1%	1.5E-08	--	--	1.0E-08	3.5E-09	0.1%	7.9E-08	--	--
4,4'-DDT	0.049	0.1	0.34	NA	3.0E-09	1.0E-09	0.0%	7.1E-09	--	--	4.8E-09	1.6E-09	0.0%	3.7E-08	--	--
Arsenic	118	0.03	1.75	0.0003	2.2E-06	3.9E-06	93.3%	5.1E-06	1.7E-02	81.8%	3.5E-06	6.1E-06	93.3%	2.7E-05	9.0E-02	81.8%
Cadmium	48.6	0.01	NA	0.0005	3.0E-07	--	--	7.1E-07	1.4E-03	6.7%	4.8E-07	--	--	3.7E-06	7.4E-03	6.7%
Chromium	588	0.01	NA	0.005	3.7E-06	--	--	8.5E-06	1.7E-03	8.2%	5.8E-06	--	--	4.5E-05	9.0E-03	8.2%
Copper	553	0.01	NA	0.0371	3.4E-06	--	--	8.0E-06	2.2E-04	1.0%	5.4E-06	--	--	4.2E-05	1.1E-03	1.0%
Lead	299	0.01	NA	NA	1.8E-06	--	--	4.3E-06	--	--	2.9E-06	--	--	2.3E-05	--	--
Manganese	51.2	0.01	NA	0.005	3.2E-07	--	--	7.4E-07	1.5E-04	0.7%	5.0E-07	--	--	3.9E-06	7.8E-04	0.7%
Mercury	0.770	0.01	NA	0.0003	4.8E-09	--	--	1.1E-08	3.7E-05	0.2%	7.6E-09	--	--	5.9E-08	2.0E-04	0.2%
Silver	19.5	0.01	NA	0.005	1.2E-07	--	--	2.9E-07	5.7E-05	0.3%	1.9E-07	--	--	1.5E-06	3.0E-04	0.3%
Vanadium	77.1	0.01	NA	0.005	4.8E-07	--	--	1.1E-06	2.2E-04	1.1%	7.6E-07	--	--	5.9E-06	1.2E-03	1.1%
					Total ILCR:	4.1E-06	100.0%	HI:	2.1E-02	100.0%	Total ILCR:	6.5E-06	100.0%	HI:	1.1E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-7
LOCAL RESIDENTS - AREA A- CURRENT TRESPASSING SCENARIO
INHALATION OF OUTDOOR AMBIENT AIR
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	Local Adult Resident	Local Child Resident (6 - 15 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFi	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDi	Oral reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of chemical in air (mg/m3)	CS	CS	
RR	Respiration rate (m3/hr)	0.83	0.83	
ET	Exposure time (hrs/d)	1	2	
EF	Exposure Frequency (d/yr)	150	150	
ED	Exposure Duration (yrs)	30	9	
BW	Body weight (kg)	70	37	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	

				Adult Residents						Child Residents (6 - 15 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFi 1/(mg/kg/d)	RfDi (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2,4-Trimethylbenzene	0.0003	NA	NA	6.3E-07	--	--	1.5E-06	--	--	7.1E-07	--	--	5.5E-06	--	--
Benzene	0.0007	0.029	0.00171	1.5E-06	4.2E-08	46.8%	3.4E-06	2.0E-03	26.1%	1.7E-06	4.9E-08	46.8%	1.3E-05	7.5E-03	26.1%
Bromomethane	0.0008	NA	0.00143	1.7E-06	--	--	3.9E-06	2.7E-03	35.7%	1.9E-06	--	--	1.5E-05	1.0E-02	35.7%
Hexachlorobutadiene	0.0003	0.077	0.0005	6.3E-07	4.8E-08	53.2%	1.5E-06	2.9E-03	38.2%	7.1E-07	5.5E-08	53.2%	5.5E-06	1.1E-02	38.2%
				Total ILCR:	9.1E-08	100.0%		HI:	7.6E-03	100.0%	Total ILCR:	1.0E-07	100.0%	HI:	2.9E-02	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-8
BRIG PRISONERS - AREA A- CURRENT SCENARIO
ACCIDENTAL INGESTION OF SURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Brig Prisoner	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
IR	Ingestion Rate (mg/d)	50	
CF	Conversion factor (kg/mg)	1E-06	
FI	Fraction of soil ingested from site	1	
EF	Exposure Frequency (d/yr)	350	
ED	Exposure Duration (yrs)	2	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	730	

				Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.353	7.7	NA	6.9E-09	5.3E-08	2.4%	2.4E-07	–	–
Aluminum	9766	NA	1	1.9E-04	–	–	6.7E-03	6.7E-03	2.1%
Arsenic	63.6	1.75	0.0003	1.2E-06	2.2E-06	97.6%	4.4E-05	1.5E-01	46.0%
Barium	880	NA	0.07	1.7E-05	–	–	6.0E-04	8.6E-03	2.7%
Cadmium	74.5	NA	0.0005	1.5E-06	–	–	5.1E-05	1.0E-01	32.3%
Chromium	101	NA	0.005	2.0E-06	–	–	6.9E-05	1.4E-02	4.4%
Copper	394	NA	0.0371	7.7E-06	–	–	2.7E-04	7.3E-03	2.3%
Lead	577	NA	NA	1.1E-05	–	–	4.0E-04	–	–
Manganese	124	NA	0.005	2.4E-06	–	–	8.5E-05	1.7E-02	5.4%
Thallium	0.920	NA	8E-05	1.8E-08	–	–	6.3E-07	7.9E-03	2.5%
Vanadium	74.0	NA	0.007	1.4E-06	–	–	5.1E-05	7.2E-03	2.3%
				Total ILCR:	2.2E-06	100.0%	HI:	3.2E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
– Not applicable.

TABLE D-9
BRIG PRISONERS - AREA A- CURRENT SCENARIO
DERMAL CONTACT WITH SURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/ (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Brig Prisoner	
DAD	Dermally absorbed dose (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
CF	Conversion factor (kg/mg)	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	
ABS	Absorption fraction --		
	Cadmium and default organics:	0.01	
	Default inorganics:	0.001	
	PCBs	0.06	
A	Skin surface area available for contact (cm	4100	
EF	Exposure Frequency (d/yr)	350	
ED	Exposure Duration (yrs)	2	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	730	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.353	0.06	7.7	NA	3.4E-08	2.6E-07	4.7%	1.2E-06	--	--
Aluminum	9766	0.01	NA	1	1.6E-04	--	--	5.5E-03	5.5E-03	1.1%
Arsenic	63.6	0.03	1.75	0.0003	3.1E-06	5.4E-06	95.3%	1.1E-04	3.6E-01	71.9%
Barium	880	0.01	NA	0.07	1.4E-05	--	--	4.9E-04	7.1E-03	1.4%
Cadmium	74.5	0.01	NA	0.0005	1.2E-06	--	--	4.2E-05	8.4E-02	16.8%
Chromium	101	0.01	NA	0.005	1.6E-06	--	--	5.7E-05	1.1E-02	2.3%
Copper	394	0.01	NA	0.0371	6.3E-06	--	--	2.2E-04	6.0E-03	1.2%
Lead	577	0.01	NA	NA	9.3E-06	--	--	3.2E-04	--	--
Manganese	124	0.01	NA	0.005	2.0E-06	--	--	7.0E-05	1.4E-02	2.8%
Thallium	0.920	0.01	NA	8E-05	1.5E-08	--	--	5.2E-07	6.5E-03	1.3%
Vanadium	74.0	0.01	NA	0.007	1.2E-06	--	--	4.2E-05	5.9E-03	1.2%
					Total ILCR:	5.6E-06	100.0%	HI:	5.0E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-10
BRIG PRISONERS - AREA A- CURRENT SCENARIO
INHALATION OF FUGITIVE DUSTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)
Where: Ca = Cs * (1/PEF)

ILCR = CDI*CSFI
HQ = CDI/RfDI

Parameter	Description	Current Brig Prisoner	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	incremental lifetime cancer risk	CS	
CSFI	Inhalation cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDi	Inhalation reference dose (mg/kg/d)	CS	
Ca	Concentration of chemical in air as fugitive dusts (m/m3)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
PEF	Particulate emission factor (m3/kg)	4.63E+09	
RR	Respiration rate (m3/hr)	0.83	
ET	Exposure time (hrs/d)	2	
EF	Exposure Frequency (d/yr)	350	
ED	Exposure Duration (yrs)	2	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	730	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	Ca (mg/m3)	CSFI 1/(mg/kg/d	RfDI (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.353	7.61E-11	NA	NA	4.9E-14	--	--	1.7E-12	--	--
Aluminum	9766	2.11E-06	NA	NA	1.4E-09	--	--	4.8E-08	--	--
Arsenic	63.6	1.37E-08	15.1	0.0003	8.9E-12	1.3E-10	16.9%	3.1E-10	1.0E-06	1.4%
Barium	880	1.90E-07	NA	0.000143	1.2E-10	--	--	4.3E-09	3.0E-05	40.8%
Cadmium	74.5	1.61E-08	6.3	NA	1.0E-11	6.6E-11	8.3%	3.7E-10	--	--
Chromium	101	2.18E-08	42	NA	1.4E-11	5.9E-10	74.8%	5.0E-10	--	--
Copper	394	8.51E-08	NA	NA	5.5E-11	--	--	1.9E-09	--	--
Lead	577	1.25E-07	NA	NA	8.1E-11	--	--	2.8E-09	--	--
Manganese	124	2.69E-08	NA	0.0000143	1.7E-11	--	--	6.1E-10	4.3E-05	57.7%
Thallium	0.920	1.99E-10	NA	NA	1.3E-13	--	--	4.5E-12	--	--
Vanadium	74.0	1.60E-08	NA	NA	1.0E-11	--	--	3.6E-10	--	--
					Total ILCR:	8.0E-10	100.0%		HI: 7.4E-05	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-11
BRIG PRISONERS - AREA A- CURRENT SCENARIO
INHALATION OF INDOOR AIR
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFI
HQ = CDI/RfDI

Parameter	Description	Current Brig Prisoner	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFI	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDI	Oral reference dose (mg/kg/d)	CS	
Ca	Concentration of chemical in air (mg/m3)	CS	
RR	Respiration rate (m3/hr)	0.83	
ET	Exposure time (hrs/d)	22	
EF	Exposure Frequency (d/yr)	350	
ED	Exposure Duration (yrs)	2	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	730	

				Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFI 1/(mg/kg/d)	RfDI (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2,4-Trimethylbenzene	0.0003	NA	NA	2.1E-06	--	--	7.5E-05	--	--
1,4-Dichlorobenzene	0.0006	NA	0.229	4.3E-06	--	--	1.5E-04	6.6E-04	0.3%
Benzene	0.0007	0.029	0.00171	5.0E-06	1.5E-07	46.8%	1.8E-04	1.0E-01	40.5%
Hexachlorobutadiene	0.0003	0.077	0.0005	2.1E-06	1.7E-07	53.2%	7.5E-05	1.5E-01	59.3%
				Total ILCR:	3.1E-07	100.0%	HI:	2.5E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-12
BRIG PRISONERS - AREA A- CURRENT SCENARIO
INHALATION OF OUTDOOR AMBIENT AIR
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFI
HQ = CDI/RfDI

Parameter	Description	Current Brig Prisoner	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFi	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDI	Oral reference dose (mg/kg/d)	CS	
Ca	Concentration of chemical in air (mg/m3)	CS	
RR	Respiration rate (m3/hr)	0.83	
ET	Exposure time (hrs/d)	2	
EF	Exposure Frequency (d/yr)	350	
ED	Exposure Duration (yrs)	2	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	730	

				Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFI 1/(mg/kg/d)	RfDI (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2,4-Trimethylbenzene	0.0003	NA	NA	1.9E-07	--	--	6.8E-06	--	--
Benzene	0.0007	0.029	0.00171	4.5E-07	1.3E-08	46.8%	1.6E-05	9.3E-03	26.1%
Bromomethane	0.0008	NA	0.00143	5.2E-07	--	--	1.8E-05	1.3E-02	35.7%
Hexachlorobutadiene	0.0003	0.077	0.0005	1.9E-07	1.5E-08	53.2%	6.8E-06	1.4E-02	38.2%
				Total ILCR	2.8E-08	100.0%	HI:	3.6E-02	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-13
BRIG EMPLOYEES - AREA A- CURRENT SCENARIO
ACCIDENTAL INGESTION OF SURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Brig Employee	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
IR	Ingestion Rate (mg/d)	50	
CF	Conversion factor (kg/mg)	1E-06	
FI	Fraction of soil ingested from site	1	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

				Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.353	7.7	NA	6.2E-08	4.7E-07	2.4%	1.7E-07	--	--
Aluminum	9766	NA	1	1.7E-03	--	--	4.8E-03	4.8E-03	2.1%
Arsenic	63.6	1.75	0.0003	1.1E-05	1.9E-05	97.6%	3.1E-05	1.0E-01	46.0%
Barium	880	NA	0.07	1.5E-04	--	--	4.3E-04	6.2E-03	2.7%
Cadmium	74.5	NA	0.0005	1.3E-05	--	--	3.6E-05	7.3E-02	32.3%
Chromium	101	NA	0.005	1.8E-05	--	--	4.9E-05	9.9E-03	4.4%
Copper	394	NA	0.0371	6.9E-05	--	--	1.9E-04	5.2E-03	2.3%
Lead	577	NA	NA	1.0E-04	--	--	2.8E-04	--	--
Manganese	124	NA	0.005	2.2E-05	--	--	6.1E-05	1.2E-02	5.4%
Thallium	0.920	NA	8E-05	1.6E-07	--	--	4.5E-07	5.6E-03	2.5%
Vanadium	74.0	NA	0.007	1.3E-05	--	--	3.6E-05	5.2E-03	2.3%
				Total ILCR:	2.0E-05	100.0%		HI:	2.3E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-14
BRIG EMPLOYEES - AREA A- CURRENT SCENARIO
DERMAL CONTACT WITH SURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Brig Employee	
DAD	Dermally absorbed dose (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
CF	Conversion factor (kg/mg)	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	
ABS	Absorption fraction –		
	Cadmium and default organics:	0.01	
	Default inorganics:	0.001	
	PCBs	0.06	
A	Skin surface area available for contact (cm2)	4100	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.353	0.06	7.7	NA	3.0E-07	2.3E-06	4.7%	8.5E-07	–	–
Aluminum	9766	0.01	NA	1	1.4E-03	–	–	3.9E-03	3.9E-03	1.1%
Arsenic	63.6	0.03	1.75	0.0003	2.7E-05	4.8E-05	95.3%	7.7E-05	2.6E-01	71.9%
Barium	880	0.01	NA	0.07	1.3E-04	–	–	3.5E-04	5.0E-03	1.4%
Cadmium	74.5	0.01	NA	0.0005	1.1E-05	–	–	3.0E-05	6.0E-02	16.9%
Chromium	101	0.01	NA	0.005	1.4E-05	–	–	4.0E-05	8.1E-03	2.3%
Copper	394	0.01	NA	0.0371	5.6E-05	–	–	1.6E-04	4.3E-03	1.2%
Lead	577	0.01	NA	NA	8.3E-05	–	–	2.3E-04	–	–
Manganese	124	0.01	NA	0.005	1.8E-05	–	–	5.0E-05	1.0E-02	2.8%
Thallium	0.920	0.01	NA	8E-05	1.3E-07	–	–	3.7E-07	4.6E-03	1.3%
Vanadium	74.0	0.01	NA	0.007	1.1E-05	–	–	3.0E-05	4.2E-03	1.2%
					Total ILCR:	5.0E-05	100.0%	HI:	3.6E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
– Not applicable.

TABLE D-15
BRIG EMPLOYEES - AREA A- CURRENT SCENARIO
INHALATION OF FUGITIVE DUSTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)
Where: Ca = Cs * (1/PEF)

ILCR = CDI*CSFI
HQ = CDI/RfDi

Parameter	Description	Current Brig Employee	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFI	Inhalation cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDi	Inhalation reference dose (mg/kg/d)	CS	
Ca	Concentration of chemical in air as fugitive dusts (mg/m3)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
PEF	Particulate emission factor (m3/kg)	4.63E+09	
RR	Respiration rate (m3/hr)	0.83	
ET	Exposure time (hrs/d)	10	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	Ca (mg/m3)	CSFI 1/(mg/kg/	RfDI (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.353	7.61E-11	NA	NA	2.2E-12	--	--	6.2E-12	--	--
Aluminum	9766	2.11E-06	NA	NA	6.1E-08	--	--	1.7E-07	--	--
Arsenic	63.8	1.37E-08	15.1	0.0003	4.0E-10	6.0E-09	16.9%	1.1E-09	3.7E-06	1.4%
Barium	880	1.90E-07	NA	0.000143	5.5E-09	--	--	1.5E-08	1.1E-04	40.8%
Cadmium	74.5	1.61E-08	6.3	NA	4.7E-10	2.9E-09	8.3%	1.3E-09	--	--
Chromium	101	2.18E-08	42	NA	6.3E-10	2.7E-08	74.8%	1.8E-09	--	--
Copper	394	8.51E-08	NA	NA	2.5E-09	--	--	6.9E-09	--	--
Lead	577	1.25E-07	NA	NA	3.6E-09	--	--	1.0E-08	--	--
Manganese	124	2.69E-08	NA	0.0000143	7.8E-10	--	--	2.2E-09	1.5E-04	57.7%
Thallium	0.920	1.99E-10	NA	NA	5.8E-12	--	--	1.6E-11	--	--
Vanadium	74.0	1.60E-08	NA	NA	4.6E-10	--	--	1.3E-09	--	--
					Total ILCR:	3.6E-08	100.0%		HI: 2.6E-04	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-16
BRIG EMPLOYEES - AREA A- CURRENT SCENARIO
ACCIDENTAL INGESTION OF SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*ET*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Brig Employee	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cw	Concentration of chemical in water (mg/L)	CS	
IR	Ingestion Rate (L/hour)	0.05	
ET	Exposure time (hours/event)	2.6	
EF	Exposure Frequency (events/yr)	12	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

				Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Tetrachloroethene	0.0053	0.052	0.01	1.2E-07	6.0E-09	0.5%	3.3E-07	3.3E-05	0.3%
Trichloroethene	0.0096	0.011	0.006	2.1E-07	2.3E-09	0.2%	5.9E-07	9.8E-05	0.8%
Vinyl chloride	0.0053	1.9	NA	1.2E-07	2.2E-07	17.8%	3.3E-07	--	--
Xylenes(total)	0.003	NA	2	6.5E-08	--	--	1.8E-07	9.2E-08	0.0%
Bis(2-ethylhexyl)phthalate	0.003	0.014	0.02	6.5E-08	9.2E-10	0.1%	1.8E-07	9.2E-06	0.1%
4,4'-DDD	0.00013	0.24	NA	2.8E-09	6.7E-10	0.1%	7.9E-09	--	--
4,4'-DDE	0.000056	0.34	NA	1.2E-09	4.2E-10	0.0%	3.4E-09	--	--
alpha-Chlordane	0.000015	1.3	0.00006	3.3E-10	4.3E-10	0.0%	9.2E-10	1.5E-05	0.1%
Aroclor-1254	0.00044	7.7	0.00002	9.6E-09	7.4E-08	6.0%	2.7E-08	1.3E-03	11.4%
delta-BHC	0.000025	1.8	NA	5.5E-10	9.8E-10	0.1%	1.5E-09	--	--
Dieldrin	0.000027	16	0.00005	5.9E-10	9.4E-09	0.8%	1.6E-09	3.3E-05	0.3%
gamma-Chlordane	0.000024	1.3	0.00006	5.2E-10	6.8E-10	0.1%	1.5E-09	2.4E-05	0.2%
Heptachlor epoxide	0.000005	9.1	0.000013	1.3E-10	1.2E-09	0.1%	3.7E-10	2.8E-05	0.2%
Aluminum	6.85	NA	1	1.5E-04	--	--	4.2E-04	4.2E-04	3.6%
Arsenic	0.024	1.75	0.0003	5.3E-07	9.2E-07	74.4%	1.5E-06	4.9E-03	41.8%
Barium	0.165	NA	0.07	3.6E-06	--	--	1.0E-05	1.4E-04	1.2%
Lead	0.261747	NA	NA	5.7E-06	--	--	1.6E-05	--	--
Manganese	0.371	NA	0.005	8.1E-06	--	--	2.3E-05	4.5E-03	38.5%
Mercury	0.0013	0.0003	NA	2.9E-08	8.8E-12	0.0%	8.2E-08	--	--
Silver	0.00459	NA	0.005	1.0E-07	--	--	2.8E-07	5.6E-05	0.5%
Zinc	0.620	NA	0.3	1.4E-05	--	--	3.8E-05	1.3E-04	1.1%
				Total ILCR:	1.2E-06	100.0%	HI:	1.2E-02	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-17
BRIG EMPLOYEES - AREA A- CURRENT SCENARIO
DERMAL CONTACT WITH SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Brig Employee	
DAD	Dermally absorbed dose (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	
A	Skin surface area available for contact (cm2)	4100	
EF	Exposure frequency (d/yr)	12	
ED	Exposure duration (yrs)	25	
EV	Event frequency (events/day)	1	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	
Cw	Concentration of chemical in water (mg/L)	CS	
CF	Conversion factor (L/cm3)	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/event)	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	
tevent	Duration of event (hours/event)	2.6	

							Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Tetrachloroethene	0.0053	4.8E-02	2.0E-01	1.1E-06	0.052	0.01	7.4E-07	3.9E-08	0.1%	2.1E-06	2.1E-04	6.7%
Trichloroethene	0.0096	1.6E-02	5.9E-02	5.7E-07	0.011	0.006	3.9E-07	4.3E-09	0.0%	1.1E-06	1.8E-04	5.9%
Vinyl chloride	0.0053	7.3E-03	2.2E-02	1.2E-07	1.9	NA	8.1E-08	1.5E-07	0.5%	2.3E-07	--	--
Xylenes(total)	0.003	4.8E-02	2.0E-01	6.1E-07	NA	2	4.2E-07	--	--	1.2E-06	5.9E-07	0.0%
Bis(2-ethylhexyl)phthalate	0.003	3.3E-02	6.7E-01	2.0E-06	0.014	0.02	1.4E-06	1.9E-08	0.1%	3.9E-06	1.9E-04	6.3%
4,4'-DDD	0.00013	2.8E-01	3.5E+00	4.5E-07	0.24	NA	3.1E-07	7.4E-08	0.3%	8.8E-07	--	--
4,4'-DDE	0.000056	2.4E-01	2.9E+00	1.7E-07	0.34	NA	1.1E-07	3.9E-09	0.1%	3.2E-07	--	--
alpha-Chlordane	0.000015	4.6E-02	1.1E+00	1.6E-08	1.3	0.00006	1.1E-08	1.5E-08	0.1%	3.1E-08	5.2E-04	16.8%
Aroclor-1254	0.00044	3.7E-01	1.2E+01	5.3E-06	7.7	NA	3.6E-06	2.8E-05	98.1%	1.0E-05	--	--
delta-BHC	0.000025	2.6E-02	2.6E-01	6.6E-09	1.8	NA	4.6E-09	8.2E-09	0.0%	1.3E-08	--	--
Dieldrin	0.000027	1.6E-02	3.0E-01	8.2E-09	16	0.00005	5.6E-09	9.0E-09	0.3%	1.6E-08	3.1E-04	10.1%
gamma-Chlordane	0.000024	4.6E-02	1.1E+00	2.6E-08	1.3	0.00006	1.8E-08	2.3E-08	0.1%	5.0E-08	8.4E-04	26.9%
Heptachlor epoxide	0.000006	6.7E-04	1.4E-02	8.1E-11	9.1	0.000013	5.6E-11	5.1E-10	0.0%	1.6E-10	1.2E-05	0.4%
Aluminum	6.85	1.0E-03	NA	1.8E-05	NA	1	1.2E-05	--	--	3.4E-05	3.4E-05	1.1%
Arsenic	0.024	1.0E-03	NA	6.3E-08	1.75	0.0003	4.3E-08	7.6E-08	0.3%	1.2E-07	4.0E-04	13.0%
Barium	0.165	1.0E-03	NA	4.3E-07	NA	0.07	3.0E-07	--	--	8.3E-07	1.2E-05	0.4%
Lead	0.261747	1.0E-03	NA	6.8E-07	NA	NA	4.7E-07	--	--	1.3E-06	--	--
Manganese	0.371	1.0E-03	NA	9.6E-07	NA	0.005	6.6E-07	--	--	1.9E-06	3.7E-04	12.0%
Mercury	0.0013	1.0E-03	NA	3.5E-09	0.0003	NA	2.4E-09	7.2E-13	0.0%	6.9E-09	--	--
Silver	0.00459	1.0E-03	NA	1.2E-08	NA	0.005	8.2E-09	--	--	2.3E-08	4.6E-06	0.1%
Zinc	0.620	1.0E-03	NA	1.6E-06	NA	0.3	1.1E-06	--	--	3.1E-06	1.0E-05	0.3%
							Total ILCR:	2.8E-05	100.0%		HI: 3.1E-03	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-18
BRIG EMPLOYEES - AREA A- CURRENT SCENARIO
ACCIDENTAL INGESTION OF SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d) (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Brig Employee	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	
IR	Ingestion Rate (mg/d)	50	
CF	Conversion factor (kg/mg)	1E-06	
FI	Fraction of sediment ingested from site	1	
EF	Exposure Frequency (d/yr)	12	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

				Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.955	7.7	NA	8.0E-09	6.2E-08	3.4%	2.2E-08	--	--
4,4'-DDE	0.103	0.34	NA	8.7E-10	3.0E-10	0.0%	2.4E-09	--	--
4,4'-DDT	0.049	0.34	NA	4.1E-10	1.4E-10	0.0%	1.1E-09	--	--
Arsenic	118	1.75	0.0003	9.9E-07	1.7E-06	96.5%	2.8E-06	9.2E-03	60.4%
Cadmium	48.6	NA	0.0005	4.1E-07	--	--	1.1E-06	2.3E-03	14.9%
Chromium	588	NA	0.005	4.9E-06	--	--	1.4E-05	2.8E-03	18.1%
Copper	553	NA	0.0371	4.6E-06	--	--	1.3E-05	3.5E-04	2.3%
Lead	289	NA	NA	2.5E-06	--	--	7.0E-06	--	--
Manganese	51.2	NA	0.005	4.3E-07	--	--	1.2E-06	2.4E-04	1.6%
Mercury	0.770	NA	0.0003	6.5E-09	--	--	1.8E-08	6.0E-05	0.4%
Silver	19.5	NA	0.005	1.6E-07	--	--	4.6E-07	9.1E-05	0.6%
Vanadium	77.1	NA	0.007	6.5E-07	--	--	1.8E-06	2.6E-04	1.7%
				Total ILCR:	1.8E-06	100.0%		HI: 1.5E-02	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-19
BRIG EMPLOYEES - AREA A- CURRENT SCENARIO
DERMAL CONTACT WITH SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Brig Employee	
DAD	Dermally absorbed dose (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in sediment (mg/k	CS	
CF	Conversion factor (kg/mg)	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	
ABS	Absorption fraction –		
	Cadmium and default organics:	0.01	
	Default inorganics:	0.001	
	PCBs	0.06	
A	Skin surface area available for contact (cm2)	4100	
EF	Exposure Frequency (d/yr)	12	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.955	0.06	7.7	NA	3.9E-08	3.0E-07	6.6%	1.1E-07	--	--
4,4'-DDE	0.103	0.1	0.34	NA	7.1E-09	2.4E-09	0.1%	2.0E-08	--	--
4,4'-DDT	0.049	0.1	0.34	NA	3.4E-09	1.1E-09	0.0%	9.4E-09	--	--
Arsenic	118	0.03	1.75	0.0003	2.4E-06	4.3E-06	93.3%	6.8E-06	2.3E-02	82.1%
Cadmium	48.6	0.01	NA	0.0005	3.3E-07	--	--	9.4E-07	1.9E-03	6.8%
Chromium	589	0.01	NA	0.005	4.0E-06	--	--	1.1E-05	2.3E-03	8.2%
Copper	553	0.01	NA	0.0371	3.8E-06	--	--	1.1E-05	2.9E-04	1.0%
Lead	299	0.01	NA	NA	2.1E-06	--	--	5.8E-06	--	--
Manganese	51.2	0.01	NA	0.005	3.5E-07	--	--	9.9E-07	2.0E-04	0.7%
Mercury	0.770	0.01	NA	0.0003	5.3E-09	--	--	1.5E-08	4.9E-05	0.2%
Silver	19.5	0.01	NA	0.005	1.3E-07	--	--	3.7E-07	7.5E-05	0.3%
Vanadium	77.1	0.01	NA	0.007	5.3E-07	--	--	1.5E-06	2.1E-04	0.8%
					Total ILCR:	4.6E-06	100.0%		HI: 2.8E-02	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-20
BRIG EMPLOYEES - AREA A- CURRENT SCENARIO
INHALATION OF INDOOR AIR
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFI
HQ = CDI/RfDi

Parameter	Description	Current Brig Employee	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFi	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDi	Oral reference dose (mg/kg/d)	CS	
Ca	Concentration of chemical in air (mg/m3)	CS	
RR	Respiration rate (m3/hr)	0.83	
ET	Exposure time (hrs/d)	10	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

Parameter	Ca (mg/m3)	CSFi 1/(mg/kg/d)	RfDi (mg/kg/d)	Carcinogens CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	Noncarcinogens CDI (mg/kg/d)	HQ	% Contrib. HI
1,2,4-Trimethylbenzene	0.0003	NA	NA	9.7E-06	--	--	2.4E-05	--	--
1,4-Dichlorobenzene	0.0008	NA	0.229	1.7E-05	--	--	4.9E-05	2.1E-04	0.3%
Benzene	0.0007	0.029	0.00171	2.0E-05	5.9E-07	46.8%	5.7E-05	3.3E-02	40.5%
Hexachlorobutadiene	0.0003	0.077	0.0005	8.7E-06	6.7E-07	53.2%	2.4E-05	4.9E-02	59.3%
				Total ILCR:	1.3E-06	100.0%	HI:	8.2E-02	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-21
BRIG EMPLOYEES - AREA A- CURRENT SCENARIO (1)
INHALATION OF OUTDOOR AMBIENT AIR
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	Current Brig Employee	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFi	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDi	Oral reference dose (mg/kg/d)	CS	
Ca	Concentration of chemical in air (mg/m3)	CS	
RR	Respiration rate (m3/hr)	0.83	
ET	Exposure time (hrs/d)	10	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

				Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFi 1/(mg/kg/	RfDi (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2,4-Trimethylbenzene	0.0005	NA	NA	1.5E-05	--	--	4.1E-05	--	--
Benzene	0.0007	0.029	0.00171	2.0E-05	5.9E-07	46.8%	5.7E-05	3.3E-02	5.5%
Bromomethane	0.0008	NA	0.00143	2.3E-05	--	--	6.5E-05	4.5E-02	7.5%
Toluene	0.67	NA	0.114	1.9E-02	--	--	5.4E-02	4.8E-01	79.9%
Hexachlorobutadiene	0.0003	0.077	0.0005	8.7E-06	6.7E-07	53.2%	2.4E-05	4.9E-02	8.1%
				Total ILCR:	1.3E-06	100.0%		HI: 6.0E-01	100.0%

NOTES:
[1] Evaluates potential air exposures to brig employees outside of the brig building and near the gas well.
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-22
ON-SITE RESIDENTS - AREA A - FUTURE SCENARIO
RESIDENTIAL INGESTION OF SHALLOW GROUNDWATER FROM LOCATION B-20W
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d) = (Cw*IR*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/d)	2	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethene	6.1	NA	0.009	7.2E-02	--	--	1.7E-01	1.9E+01	9.9%	3.3E-02	--	--	2.6E-01	2.9E+01	9.9%
2-Butanone	4.3	NA	0.6	5.0E-02	--	--	1.2E-01	2.0E-01	0.1%	2.4E-02	--	--	1.8E-01	3.1E-01	0.1%
4-methyl-2-pentanone	16	NA	0.08	1.9E-01	--	--	4.4E-01	5.5E+00	2.9%	8.8E-02	--	--	6.8E-01	8.5E+00	2.9%
Benzene	0.31	0.029	NA	3.6E-03	1.1E-04	0.1%	8.5E-03	--	--	1.7E-03	4.9E-05	0.1%	1.3E-02	--	--
Methylene chloride	ND	0.0075	0.06	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene	ND	0.052	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Toluene	5.4	NA	0.2	6.3E-02	--	--	1.5E-01	7.4E-01	0.4%	3.0E-02	--	--	2.3E-01	1.2E+00	0.4%
Trichloroethene	ND	0.011	0.006	--	--	--	--	--	--	--	--	--	--	--	--
Vinyl chloride	3.3	1.9	NA	3.9E-02	7.4E-02	93.9%	9.0E-02	--	--	1.8E-02	3.4E-02	93.9%	1.4E-01	--	--
2-Methylphenol	1.8	NA	0.05	2.1E-02	--	--	4.9E-02	9.9E-01	0.5%	9.9E-03	--	--	7.7E-02	1.5E+00	0.5%
2,4-Dimethylphenol	1.4	NA	0.02	1.6E-02	--	--	3.8E-02	1.9E+00	1.0%	7.7E-03	--	--	6.0E-02	3.0E+00	1.0%
4-Methylphenol	21	NA	0.005	2.5E-01	--	--	5.8E-01	1.2E+02	61.5%	1.2E-01	--	--	8.9E-01	1.9E+02	61.5%
Bis(2-ethylhexyl)phthalate	ND	0.014	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Aldrin	ND	17	0.00003	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	0.00014	9.1	0.000013	1.6E-06	1.5E-05	0.0%	3.8E-06	3.0E-01	0.2%	7.7E-07	7.0E-06	0.0%	6.0E-06	4.6E-01	0.2%
Aluminum	80.6	NA	1	9.5E-01	--	--	2.2E+00	2.2E+00	1.2%	4.4E-01	--	--	3.4E+00	3.4E+00	1.2%
Antimony	ND	NA	0.0004	--	--	--	--	--	--	--	--	--	--	--	--
Arsenic	0.2	1.75	0.0003	2.3E-03	4.1E-03	5.2%	5.5E-03	1.8E+01	9.8%	1.1E-03	1.9E-03	5.2%	8.5E-03	2.8E+01	9.8%
Barium	7.27	NA	0.07	8.5E-02	--	--	2.0E-01	2.8E+00	1.5%	4.0E-02	--	--	3.1E-01	4.4E+00	1.5%
Beryllium	0.0106	4.3	0.005	1.2E-04	5.4E-04	0.7%	2.9E-04	5.8E-02	0.0%	5.8E-05	2.5E-04	0.7%	4.6E-04	9.0E-02	0.0%
Cadmium	0.0459	NA	0.0005	5.4E-04	--	--	1.3E-03	2.5E+00	1.3%	2.5E-04	--	--	2.0E-03	3.9E+00	1.3%
Chromium	0.187	NA	0.005	2.2E-03	--	--	5.1E-03	1.0E+00	0.5%	1.0E-03	--	--	8.0E-03	1.6E+00	0.5%
Copper	0.279	NA	0.0371	3.3E-03	--	--	7.6E-03	2.1E-01	0.1%	1.5E-03	--	--	1.2E-02	3.2E-01	0.1%
Lead	0.298	NA	NA	3.5E-03	--	--	8.2E-03	--	--	1.6E-03	--	--	1.3E-02	--	--
Manganese	2.76	NA	0.005	3.2E-02	--	--	7.6E-02	1.5E+01	8.1%	1.5E-02	--	--	1.2E-01	2.4E+01	8.1%
Nickel	0.114	NA	0.02	1.3E-03	--	--	3.1E-03	1.6E-01	0.1%	6.2E-04	--	--	4.9E-03	2.4E-01	0.1%
Vanadium	0.396	NA	0.007	4.6E-03	--	--	1.1E-02	1.5E+00	0.8%	2.2E-03	--	--	1.7E-02	2.4E+00	0.8%
				Total ILCR:	7.8E-02	100.0%	HI:	1.9E+02	100.0%	Total ILCR:	3.7E-02	100.0%	HI:	2.9E+02	100.0%

NOTES:
NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-23
ON-SITE RESIDENTS - AREA A - FUTURE SCENARIO
RESIDENTIAL DERMAL CONTACT WITH SHALLOW GROUNDWATER FROM LOCATION B-20W
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics; DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics; DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	20000	8023	
EF	Exposure frequency (d/yr)	350	350	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/event	CS	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	0.2	0.2	

							Adult Residents						Child Residents (0 - 6 Yrs Old)					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethene	6.1	1.0E-02	7.2E-03	4.4E-05	NA	0.009	5.2E-03	--	--	1.2E-02	1.3E+00	7.3%	1.9E-03	--	--	2.3E-02	2.5E+00	7.3%
2-Butanone	4.3	1.1E-03	6.7E-04	2.9E-06	NA	0.6	3.4E-04	--	--	7.9E-04	1.3E-03	0.0%	1.3E-04	--	--	1.5E-03	2.5E-03	0.0%
4-methyl-2-pentanone	16	3.3E-03	2.5E-03	3.9E-05	NA	0.08	4.6E-03	--	--	1.1E-02	1.3E-01	0.7%	1.7E-03	--	--	2.0E-02	2.5E-01	0.7%
Benzene	0.31	2.1E-01	1.3E-01	4.1E-05	0.029	NA	4.8E-03	1.4E-04	4.4%	1.1E-02	--	--	1.8E-03	5.2E-05	4.4%	2.1E-02	--	--
Methylene chloride	ND	4.5E-03	3.0E-03	NA	0.0075	0.06	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene	ND	4.8E-02	5.6E-02	NA	0.052	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Toluene	5.4	4.5E-02	3.1E-02	1.7E-04	NA	0.2	2.0E-02	--	--	4.7E-02	2.3E-01	1.3%	7.5E-03	--	--	0.7E-02	4.4E-01	1.3%
Trichloroethene	ND	1.6E-02	1.5E-02	NA	0.011	0.006	--	--	--	--	--	--	--	--	--	--	--	--
Vinyl chloride	3.3	7.3E-03	4.1E-03	1.4E-05	1.9	NA	1.6E-03	3.0E-03	95.3%	3.7E-03	--	--	6.0E-04	1.1E-03	95.3%	7.0E-03	--	--
2-Methylphenol	1.8	1.0E-02	7.8E-03	1.4E-05	NA	0.05	1.7E-03	--	--	3.9E-03	7.7E-02	0.4%	6.2E-04	--	--	7.2E-03	1.4E-01	0.4%
2,4-Dimethylphenol	1.4	1.5E-02	1.3E-02	1.8E-05	NA	0.02	2.1E-03	--	--	5.0E-03	2.5E-01	1.4%	8.0E-04	--	--	9.3E-03	4.7E-01	1.4%
4-Methylphenol	21	1.8E-02	1.4E-02	3.0E-04	NA	0.005	3.5E-02	--	--	8.1E-02	1.6E+01	88.4%	1.3E-02	--	--	1.5E-01	3.0E+01	88.4%
Bis(2-ethylhexyl)phthalate	ND	3.3E-02	1.9E-01	NA	0.014	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Aldrin	ND	1.6E-03	7.7E-03	NA	17	0.00003	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	0.00014	6.7E-04	3.8E-03	5.3E-10	9.1	0.000013	6.2E-08	5.6E-07	0.0%	1.4E-07	1.1E-02	0.1%	2.3E-08	2.1E-07	0.0%	2.7E-07	2.1E-02	0.1%
Aluminum	80.6	1.0E-03	NA	1.6E-05	NA	1	1.9E-03	--	--	4.4E-03	4.4E-03	0.0%	7.1E-04	--	--	8.3E-03	8.3E-03	0.0%
Antimony	ND	1.0E-03	NA	NA	NA	0.0004	--	--	--	--	--	--	--	--	--	--	--	--
Arsenic	0.2	1.0E-03	NA	4.0E-08	1.75	0.0003	4.7E-06	8.2E-06	0.3%	1.1E-05	3.7E-02	0.2%	1.8E-06	3.1E-06	0.3%	2.1E-05	6.9E-02	0.2%
Barium	7.27	1.0E-03	NA	1.5E-06	NA	0.07	1.7E-04	--	--	4.0E-04	5.7E-03	0.0%	6.4E-05	--	--	7.5E-04	1.1E-02	0.0%
Beryllium	0.0106	1.0E-03	NA	2.1E-09	4.3	0.005	2.5E-07	1.1E-06	0.0%	5.8E-07	1.2E-04	0.0%	9.3E-08	4.0E-07	0.0%	1.1E-06	2.2E-04	0.0%
Cadmium	0.0459	1.0E-03	NA	9.2E-09	NA	0.0005	1.1E-06	--	--	2.5E-06	5.0E-03	0.0%	4.0E-07	--	--	4.7E-06	9.4E-03	0.0%
Chromium	0.187	1.0E-03	NA	3.7E-08	NA	0.005	4.4E-06	--	--	1.0E-05	2.0E-03	0.0%	1.6E-06	--	--	1.9E-05	3.8E-03	0.0%
Copper	0.279	1.0E-03	NA	5.6E-08	NA	0.0371	6.6E-06	--	--	1.5E-05	4.1E-04	0.0%	2.5E-06	--	--	2.9E-05	7.7E-04	0.0%
Lead	0.298	1.0E-03	NA	6.0E-08	NA	NA	7.0E-06	--	--	1.6E-05	--	--	2.6E-06	--	--	3.1E-05	--	--
Manganese	2.76	1.0E-03	NA	5.5E-07	NA	0.005	6.5E-05	--	--	1.5E-04	3.0E-02	0.2%	2.4E-05	--	--	2.8E-04	5.7E-02	0.2%
Nickel	0.114	1.0E-03	NA	2.3E-08	NA	0.02	2.7E-06	--	--	6.2E-06	3.1E-04	0.0%	1.0E-06	--	--	1.2E-05	5.8E-04	0.0%
Vanadium	0.396	1.0E-03	NA	7.9E-08	NA	0.007	9.3E-06	--	--	2.2E-05	3.1E-03	0.0%	3.5E-06	--	--	4.1E-05	5.8E-03	0.0%
							Total ILCR:	3.2E-03	100.0%	HI:	1.8E+01	100.0%	Total ILCR:	1.2E-03	100.0%	HI:	3.4E+01	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
ND - Not detected.

TABLE D-24
ON-SITE RESIDENTS - AREA A- FUTURE SCENARIO
INHALATION OF VOLATILIZED ORGANICS IN SHOWER AIR
SHALLOW GROUNDWATER FROM LOCATION B-20W
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (CA*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFI
HQ = CDI/RfDI

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFI	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDI	Oral reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of volatilized chemical in shower air, Andelman Model (mg/m3)	CS	CS	
RR	Respiration rate (m3/hr)	0.83	0.6	
ET	Exposure time (hrs/d)	0.2	0.2	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFI 1/(mg/kg/	RfDI (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Benzene	4.36	0.029	0.00171	4.2E-03	1.2E-04	0.1%	9.9E-03	5.8E+00	6.5%	2.9E-03	8.3E-05	0.1%	3.3E-02	2.0E+01	6.5%
1,2-Dichloroethane	102	0.091	0.00286	9.9E-02	9.0E-03	4.8%	2.3E-01	8.1E+01	91.5%	6.7E-02	6.1E-03	4.5%	7.8E-01	2.7E+02	91.5%
4-Methyl-2-pentanone	0.301	NA	0.0229	2.9E-04	--	--	6.8E-04	3.0E-02	0.0%	2.0E-04	--	--	2.3E-03	1.0E-01	0.0%
Methylene chloride	ND	0.00164	0.857	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene	ND	0.00203	NA	--	--	--	--	--	--	--	--	--	--	--	--
Trichloroethene	ND	0.006	NA	--	--	--	--	--	--	--	--	--	--	--	--
Toluene	87.9	NA	0.114	8.6E-02	--	--	2.0E-01	1.8E+00	2.0%	5.8E-02	--	--	6.7E-01	5.9E+00	2.0%
Vinyl chloride [1]	691	0.3	NA	6.7E-01	1.8E-01	95.2%	1.6E+00	--	--	4.5E-01	1.3E-01	95.5%	5.3E+00	--	--
				Total ILCR:	1.9E-01	100.0%		HI:	8.9E+01	100.0%	Total ILCR:	1.4E-01	100.0%		HI:	3.0E+02	100.0%

NOTES:
[1] The ILCRs for vinyl chloride were estimated using the one-hit equation, where ILCR = 1 - exp(-CDI x CSF).
NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-25
ON-SITE RESIDENTS - AREA A - FUTURE SCENARIO
RESIDENTIAL INGESTION OF SHALLOW GROUNDWATER FROM LOCATION B-20W
ORGANIC AND FILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/d)	2	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethene	6.1	NA	0.009	7.2E-02	--	--	1.7E-01	1.9E+01	10.4%	3.3E-02	--	--	2.6E-01	2.9E+01	10.4%
4-methyl-2-pentanone	16	NA	0.08	1.9E-01	--	--	4.4E-01	5.5E+00	3.1%	8.8E-02	--	--	6.8E-01	8.5E+00	3.1%
Benzene	0.31	0.029	NA	3.6E-03	1.1E-04	0.1%	8.5E-03	--	--	1.7E-03	4.9E-05	0.1%	1.3E-02	--	--
Methylene chloride	ND	0.0075	0.06	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene	ND	0.052	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Toluene	5.4	NA	0.2	6.3E-02	--	--	1.5E-01	7.4E-01	0.4%	3.0E-02	--	--	2.3E-01	1.2E+00	0.4%
Trichloroethene	ND	0.011	0.006	--	--	--	--	--	--	--	--	--	--	--	--
Vinyl chloride	3.3	1.9	NA	3.9E-02	7.4E-02	94.6%	9.0E-02	--	--	1.8E-02	3.4E-02	94.6%	1.4E-01	--	--
2-Methylphenol	1.8	NA	0.05	2.1E-02	--	--	4.9E-02	9.9E-01	0.6%	9.9E-03	--	--	7.7E-02	1.5E+00	0.6%
2,4-Dimethylphenol	1.4	NA	0.02	1.6E-02	--	--	3.8E-02	1.9E+00	1.1%	7.7E-03	--	--	6.0E-02	3.0E+00	1.1%
4-Methylphenol	21	NA	0.005	2.5E-01	--	--	5.8E-01	1.2E+02	64.6%	1.2E-01	--	--	8.9E-01	1.8E+02	64.6%
Bis(2-ethylhexyl)phthalate	ND	0.014	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Aldrin	ND	17	0.00003	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	0.00014	9.1	0.000013	1.6E-06	1.5E-05	0.0%	3.8E-06	3.0E-01	0.2%	7.7E-07	7.0E-06	0.0%	6.0E-06	4.6E-01	0.2%
Dissolved Arsenic	0.2	1.75	0.0003	2.3E-03	4.1E-03	5.3%	5.5E-03	1.8E+01	10.3%	1.1E-03	1.9E-03	5.3%	8.5E-03	2.8E+01	10.3%
Dissolved Barium	6.06	NA	0.07	7.1E-02	--	--	1.7E-01	2.4E+00	1.3%	3.3E-02	--	--	2.6E-01	3.7E+00	1.3%
Dissolved Lead	ND	NA	NA	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Manganese	2.63	NA	0.005	3.1E-02	--	--	7.2E-02	1.4E+01	8.1%	1.4E-02	--	--	1.1E-01	2.2E+01	8.1%
				Total ILCR:	7.8E-02	100.0%		HI:	1.8E+02	100.0%	Total ILCR:	3.6E-02	100.0%	HI:	2.8E+02	100.0%

NOTES:
NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-26
ON-SITE RESIDENTS - AREA A - FUTURE SCENARIO
RESIDENTIAL DERMAL CONTACT WITH SHALLOW GROUNDWATER FROM LOCATION B-20W
ORGANIC AND FILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	20000	8023	
EF	Exposure frequency (d/yr)	350	350	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/even	CS	CS	
Kp	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	0.2	0.2	

								Adult Residents						Child Residents (0 - 6 Yrs Old)					
								Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)		DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethene	6.1	1.0E-02	7.2E-03	4.4E-05	NA	0.009		5.2E-03	--	--	1.2E-02	1.3E+00	7.3%	1.9E-03	--	--	2.3E-02	2.5E+00	7.3%
4-methyl-2-pentanone	16	3.3E-03	2.5E-03	3.9E-05	NA	0.08		4.6E-03	--	--	1.1E-02	1.3E-01	0.7%	1.7E-03	--	--	2.0E-02	2.5E-01	0.7%
Benzene	0.31	2.1E-01	1.3E-01	4.1E-05	0.029	NA		4.8E-03	1.4E-04	4.4%	1.1E-02	--	--	1.8E-03	5.2E-05	4.4%	2.1E-02	--	--
Methylene chloride	ND	4.5E-03	3.0E-03	NA	0.0075	0.06		--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene	ND	4.8E-02	5.6E-02	NA	0.052	0.01		--	--	--	--	--	--	--	--	--	--	--	--
Toluene	5.4	4.5E-02	3.1E-02	1.7E-04	NA	0.2		2.0E-02	--	--	4.7E-02	2.3E-01	1.3%	7.5E-03	--	--	8.7E-02	4.4E-01	1.3%
Trichloroethene	ND	1.6E-02	1.5E-02	NA	0.011	0.006		--	--	--	--	--	--	--	--	--	--	--	--
Vinyl chloride	3.3	7.3E-03	4.1E-03	1.4E-05	1.9	NA		1.6E-03	3.0E-03	95.4%	3.7E-03	--	--	6.0E-04	1.1E-03	95.4%	7.0E-03	--	--
2-Methylphenol	1.8	1.0E-02	7.8E-03	1.4E-05	NA	0.05		1.7E-03	--	--	3.9E-03	7.7E-02	0.4%	6.2E-04	--	--	7.2E-03	1.4E-01	0.4%
2,4-Dimethylphenol	1.4	1.5E-02	1.3E-02	1.8E-05	NA	0.02		2.1E-03	--	--	5.0E-03	2.5E-01	1.4%	8.0E-04	--	--	9.3E-03	4.7E-01	1.4%
4-Methylphenol	21	1.8E-02	1.4E-02	3.0E-04	NA	0.005		3.5E-02	--	--	8.1E-02	1.6E+01	88.5%	1.3E-02	--	--	1.5E-01	3.0E+01	88.5%
Bis(2-ethylhexyl)phthalate	ND	3.3E-02	1.9E-01	NA	0.014	0.02		--	--	--	--	--	--	--	--	--	--	--	--
Aldrin	ND	1.6E-03	7.7E-03	NA	17	0.00003		--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	0.00014	6.7E-04	3.8E-03	5.3E-10	9.1	0.000013		6.2E-08	5.6E-07	0.0%	1.4E-07	1.1E-02	0.1%	2.3E-08	2.1E-07	0.0%	2.7E-07	2.1E-02	0.1%
Dissolved Arsenic	0.2	1.0E-03	NA	4.0E-08	1.75	0.0003		4.7E-06	8.2E-06	0.3%	1.1E-05	3.7E-02	0.2%	1.8E-06	3.1E-06	0.3%	2.1E-05	6.8E-02	0.2%
Dissolved Barium	6.06	1.0E-03	NA	1.2E-06	NA	0.07		1.4E-04	--	--	3.3E-04	4.7E-03	0.0%	5.3E-05	--	--	6.2E-04	8.9E-03	0.0%
Dissolved Lead	NA	1.0E-03	NA	NA	NA	NA		--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Manganese	2.63	1.0E-03	NA	5.3E-07	NA	0.005		6.2E-05	--	--	1.4E-04	2.9E-02	0.2%	2.3E-05	--	--	2.7E-04	5.4E-02	0.2%
							Total ILCR:	3.2E-03		100.0%	HI:	1.8E+01	100.0%	Total ILCR:	1.2E-03	100.0%	HI:	3.4E+01	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-27
ON-SITE RESIDENTS - AREA A - FUTURE SCENARIO
RESIDENTIAL INGESTION OF SHALLOW GROUNDWATER FROM LOCATION B-20WSS
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/d)	2	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethene	1.6	NA	0.009	1.9E-02	--	--	4.4E-02	4.9E+00	11.9%	8.8E-03	--	--	6.8E-02	7.6E+00	11.9%
4-methyl-2-pentanone	0.22	NA	0.08	2.6E-03	--	--	6.0E-03	7.5E-02	0.2%	1.2E-03	--	--	9.4E-03	1.2E-01	0.2%
Benzene	ND	0.029	NA	--	--	--	--	--	--	--	--	--	--	--	--
Methylene chloride	0.057	0.0075	0.06	6.7E-04	5.0E-06	0.2%	1.6E-03	2.6E-02	0.1%	3.1E-04	2.3E-06	0.2%	2.4E-03	4.0E-02	0.1%
Tetrachloroethene	0.62	0.052	0.01	7.3E-03	3.8E-04	16.1%	1.7E-02	1.7E+00	4.1%	3.4E-03	1.8E-04	16.1%	2.6E-02	2.6E+00	4.1%
Toluene	1.2	NA	0.2	1.4E-02	--	--	3.3E-02	1.6E-01	0.4%	6.6E-03	--	--	5.1E-02	2.6E-01	0.4%
Trichloroethene	1.8	0.011	0.006	2.1E-02	2.3E-04	9.9%	4.9E-02	8.2E+00	20.0%	9.9E-03	1.1E-04	9.9%	7.7E-02	1.3E+01	20.0%
Vinyl chloride	ND	1.9	NA	--	--	--	--	--	--	--	--	--	--	--	--
2-Methylphenol	0.28	NA	0.05	3.3E-03	--	--	7.7E-03	1.5E-01	0.4%	1.5E-03	--	--	1.2E-02	2.4E-01	0.4%
2,4-Dimethylphenol	0.025	NA	0.02	2.9E-04	--	--	6.8E-04	3.4E-02	0.1%	1.4E-04	--	--	1.1E-03	5.3E-02	0.1%
4-Methylphenol	0.1	NA	0.005	1.2E-03	--	--	2.7E-03	5.5E-01	1.3%	5.5E-04	--	--	4.3E-03	8.5E-01	1.3%
Bis(2-ethylhexyl)phthalate	ND	0.014	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Aldrin	ND	17	0.00003	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Aluminum	132	NA	1	1.5E+00	--	--	3.6E+00	3.6E+00	8.8%	7.2E-01	--	--	5.6E+00	5.6E+00	8.8%
Antimony	ND	NA	0.0004	--	--	--	--	--	--	--	--	--	--	--	--
Arsenic	0.0681	1.75	0.0003	8.0E-04	1.4E-03	59.5%	1.9E-03	6.2E+00	15.1%	3.7E-04	6.5E-04	59.5%	2.9E-03	9.7E+00	15.1%
Barium	0.57	NA	0.07	6.7E-03	--	--	1.6E-02	2.2E-01	0.5%	3.1E-03	--	--	2.4E-02	3.5E-01	0.5%
Beryllium	0.0067	4.3	0.005	7.9E-05	3.4E-04	14.4%	1.8E-04	3.7E-02	0.1%	3.7E-05	1.6E-04	14.4%	2.9E-04	5.7E-02	0.1%
Cadmium	ND	NA	0.0005	--	--	--	--	--	--	--	--	--	--	--	--
Chromium	0.353	NA	0.005	4.1E-03	--	--	9.7E-03	1.9E+00	4.7%	1.9E-03	--	--	1.5E-02	3.0E+00	4.7%
Copper	0.183	NA	0.0371	2.1E-03	--	--	5.0E-03	1.4E-01	0.3%	1.0E-03	--	--	7.8E-03	2.1E-01	0.3%
Lead	0.381	NA	NA	4.5E-03	--	--	1.0E-02	--	--	2.1E-03	--	--	1.6E-02	--	--
Manganese	2.06	NA	0.005	2.4E-02	--	--	5.6E-02	1.1E+01	27.5%	1.1E-02	--	--	8.8E-02	1.8E+01	27.5%
Nickel	0.352	NA	0.02	4.1E-03	--	--	9.6E-03	4.8E-01	1.2%	1.9E-03	--	--	1.5E-02	7.5E-01	1.2%
Vanadium	0.344	NA	0.007	4.0E-03	--	--	9.4E-03	1.3E+00	3.3%	1.9E-03	--	--	1.5E-02	2.1E+00	3.3%
				Total ILCR:	2.4E-03	100.0%	HI:	4.1E+01	100.0%	Total ILCR:	1.1E-03	100.0%	HI:	6.4E+01	100.0%

NOTES:
NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-28
ON-SITE RESIDENTS - AREA A - FUTURE SCENARIO
RESIDENTIAL DERMAL CONTACT WITH SHALLOW GROUNDWATER FROM LOCATION B-20WSS
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	20000	8023	
EF	Exposure frequency (d/yr)	350	350	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/eve	CS	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	0.2	0.2	

							Adult Residents						Child Residents (0 - 6 Yrs Old)					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	1.6	1.0E-02	7.2E-03	1.2E-05	NA	0.009	1.4E-03	--	--	3.2E-03	3.5E-01	13.0%	5.1E-04	--	--	5.9E-03	6.6E-01	13.0%
4-methyl-2-pentanone	0.22	3.3E-03	2.5E-03	5.4E-07	NA	0.08	6.3E-05	--	--	1.5E-04	1.8E-03	0.1%	2.4E-05	--	--	2.8E-04	3.5E-03	0.1%
Benzene	ND	2.1E-01	1.3E-01	NA	0.029	NA	--	--	--	--	--	--	--	--	--	--	--	--
Methylene chloride	0.057	4.5E-03	3.0E-03	1.7E-07	0.0075	0.06	2.0E-05	1.5E-07	0.1%	4.7E-05	7.8E-04	0.0%	7.5E-06	5.6E-08	0.1%	8.8E-05	1.5E-03	0.0%
Tetrachloroethene	0.62	4.8E-02	5.6E-02	3.5E-05	0.052	0.01	4.1E-03	2.1E-04	85.0%	9.6E-03	9.6E-01	35.3%	1.5E-03	8.0E-05	85.0%	1.8E-02	1.8E+00	35.3%
Toluene	1.2	4.5E-02	3.1E-02	3.8E-05	NA	0.2	4.4E-03	--	--	1.0E-02	5.2E-02	1.9%	1.7E-03	--	--	1.9E-02	9.7E-02	1.9%
Trichloroethene	1.8	1.6E-02	1.5E-02	2.6E-05	0.011	0.006	3.1E-03	3.4E-05	13.6%	7.2E-03	1.2E+00	44.5%	1.2E-03	1.3E-05	13.6%	1.4E-02	2.3E+00	44.5%
Vinyl chloride	ND	7.3E-03	4.1E-03	NA	1.9	NA	--	--	--	--	--	--	--	--	--	--	--	--
2-Methylphenol	0.28	1.0E-02	7.8E-03	2.2E-06	NA	0.05	2.6E-04	--	--	6.0E-04	1.2E-02	0.4%	9.6E-05	--	--	1.1E-03	2.2E-02	0.4%
2,4-Dimethylphenol	0.025	1.5E-02	1.3E-02	3.2E-07	NA	0.02	3.8E-05	--	--	8.9E-05	4.4E-03	0.2%	1.4E-05	--	--	1.7E-04	8.3E-03	0.2%
4-Methylphenol	0.1	1.8E-02	1.4E-02	1.4E-06	NA	0.005	1.7E-04	--	--	3.9E-04	7.7E-02	2.8%	6.2E-05	--	--	7.2E-04	1.4E-01	2.8%
Bis(2-ethylhexyl)phthalate	ND	3.3E-02	1.9E-01	NA	0.014	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Aldrin	ND	1.6E-03	7.7E-03	NA	17	0.00003	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	6.7E-04	3.8E-03	NA	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Aluminum	132	1.0E-03	NA	2.9E-05	NA	1	3.1E-03	--	--	7.2E-03	7.2E-03	0.3%	1.2E-03	--	--	1.4E-02	1.4E-02	0.3%
Antimony	ND	1.0E-03	NA	NA	NA	0.0004	--	--	--	--	--	--	--	--	--	--	--	--
Arsenic	0.0681	1.0E-03	NA	1.4E-08	1.75	0.0003	1.6E-06	2.8E-06	1.1%	3.7E-06	1.2E-02	0.5%	6.0E-07	1.0E-06	1.1%	7.0E-06	2.3E-02	0.5%
Barium	0.57	1.0E-03	NA	1.1E-07	NA	0.07	1.3E-05	--	--	3.1E-05	4.5E-04	0.0%	5.0E-06	--	--	5.8E-05	8.4E-04	0.0%
Beryllium	0.0067	1.0E-03	NA	1.3E-09	4.3	0.005	1.6E-07	6.8E-07	0.3%	3.7E-07	7.3E-05	0.0%	5.9E-08	2.5E-07	0.3%	6.9E-07	1.4E-04	0.0%
Cadmium	ND	1.0E-03	NA	NA	NA	0.0005	--	--	--	--	--	--	--	--	--	--	--	--
Chromium	0.353	1.0E-03	NA	7.1E-08	NA	0.005	8.3E-06	--	--	1.9E-05	3.9E-03	0.1%	3.1E-06	--	--	3.6E-05	7.2E-03	0.1%
Copper	0.183	1.0E-03	NA	3.7E-08	NA	0.0371	4.3E-06	--	--	1.0E-05	2.7E-04	0.0%	1.6E-06	--	--	1.9E-05	5.1E-04	0.0%
Lead	0.381	1.0E-03	NA	7.5E-08	NA	NA	8.9E-06	--	--	2.1E-05	--	--	-3.3E-06	--	--	3.9E-05	--	--
Manganese	2.06	1.0E-03	NA	4.1E-07	NA	0.005	4.8E-05	--	--	1.1E-04	2.3E-02	0.8%	1.8E-05	--	--	2.1E-04	4.2E-02	0.8%
Nickel	0.352	1.0E-03	NA	7.0E-08	NA	0.02	8.3E-06	--	--	1.9E-05	9.5E-04	0.0%	3.1E-06	--	--	3.6E-05	1.8E-03	0.0%
Vanadium	0.344	1.0E-03	NA	6.9E-08	NA	0.007	8.1E-06	--	--	1.9E-05	2.7E-03	0.1%	3.0E-06	--	--	3.5E-05	5.0E-03	0.1%
							Total ILCR:	2.5E-04	100.0%		HI: 2.7E+00	100.0%	Total ILCR:	9.4E-05	100.0%		HI: 5.1E+00	100.0%

NOTES:

[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lag times, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-29
ON-SITE RESIDENTS - AREA A- FUTURE SCENARIO
INHALATION OF VOLATILIZED ORGANICS IN SHOWER AIR
SHALLOW GROUNDWATER FROM LOCATION B-20WSS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (CA*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFI
HQ = CDI/RfDI

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFI	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDI	Oral reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of volatilized chemical in shower air, Andelman Model (mg/m3)	CS	CS	
RR	Respiration rate (m3/hr)	0.83	0.6	
ET	Exposure time (hrs/d)	0.2	0.2	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFI 1/(mg/kg/d)	RfDI (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Benzene	ND	0.029	0.00171	--	--	--	--	--	--	--	--	--	--	--	--
1,2-Dichloroethane	26.8	0.091	0.00286	2.6E-02	2.4E-03	87.9%	6.1E-02	2.1E+01	98.2%	1.8E-02	1.6E-03	87.9%	2.1E-01	7.2E+01	98.2%
4-Methyl-2-pentanone	0.053	NA	0.0229	5.2E-05	--	--	1.2E-04	5.3E-03	0.0%	3.5E-05	--	--	4.1E-04	1.8E-02	0.0%
Methylene chloride	0.3	0.00164	0.857	2.9E-04	4.8E-07	0.0%	6.8E-04	8.0E-04	0.0%	2.0E-04	3.2E-07	0.0%	2.3E-03	2.7E-03	0.0%
Tetrachloroethene	41	0.00203	NA	4.0E-02	8.1E-05	3.0%	9.3E-02	--	--	2.7E-02	5.5E-05	3.0%	3.1E-01	--	--
Trichloroethene	41.9	0.006	NA	4.1E-02	2.5E-04	9.1%	9.5E-02	--	--	2.8E-02	1.7E-04	9.1%	3.2E-01	--	--
Toluene	19.5	NA	0.114	1.9E-02	--	--	4.4E-02	3.9E-01	1.8%	1.3E-02	--	--	1.5E-01	1.3E+00	1.8%
Vinyl chloride	ND	0.3	NA	--	--	--	--	--	--	--	--	--	--	--	--
				Total ILCR:	2.7E-03	100.0%	HI:	2.2E+01	100.0%	Total ILCR	1.8E-03	100.0%	HI:	7.3E+01	100.0%

NOTES:
NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-30
ON-SITE RESIDENTS - AREA A - FUTURE SCENARIO
RESIDENTIAL INGESTION OF SHALLOW GROUNDWATER FROM LOCATION B-20WSS
ORGANIC AND FILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/d)	2	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	1.6	NA	0.009	1.9E-02	--	--	4.4E-02	4.9E+00	27.1%	8.8E-03	--	--	6.8E-02	7.6E+00	27.1%
4-methyl-2-pentanone	0.22	NA	0.08	2.6E-03	--	--	6.0E-03	7.5E-02	0.4%	1.2E-03	--	--	9.4E-03	1.2E-01	0.4%
Benzene	ND	0.029	NA	--	--	--	--	--	--	--	--	--	--	--	--
Methylene chloride	0.057	0.0075	0.06	6.7E-04	5.0E-06	0.6%	1.6E-03	2.6E-02	0.1%	3.1E-04	2.3E-06	0.6%	2.4E-03	4.0E-02	0.1%
Tetrachloroethene	0.62	0.052	0.01	7.3E-03	3.8E-04	44.1%	1.7E-02	1.7E+00	9.5%	3.4E-03	1.8E-04	44.1%	2.6E-02	2.6E+00	9.5%
Toluene	1.2	NA	0.2	1.4E-02	--	--	3.3E-02	1.6E-01	0.9%	6.6E-03	--	--	5.1E-02	2.6E-01	0.9%
Trichloroethene	1.8	0.011	0.006	2.1E-02	2.3E-04	27.1%	4.9E-02	8.2E+00	45.8%	9.9E-03	1.1E-04	27.1%	7.7E-02	1.3E+01	45.8%
Vinyl chloride	ND	1.9	NA	--	--	--	--	--	--	--	--	--	--	--	--
2-Methylphenol	0.28	NA	0.05	3.3E-03	--	--	7.7E-03	1.5E-01	0.9%	1.5E-03	--	--	1.2E-02	2.4E-01	0.9%
2,4-Dimethylphenol	0.025	NA	0.02	2.9E-04	--	--	6.8E-04	3.4E-02	0.2%	1.4E-04	--	--	1.1E-03	5.3E-02	0.2%
4-Methylphenol	0.1	NA	0.005	1.2E-03	--	--	2.7E-03	5.5E-01	3.1%	5.5E-04	--	--	4.3E-03	8.5E-01	3.1%
Bis(2-ethylhexyl)phthalate	ND	0.014	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Aldrin	ND	17	0.00003	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	*ND*	*9.1*	*0.000013*	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Arsenic	0.0118	1.75	0.0003	1.4E-04	2.4E-04	28.2%	3.2E-04	1.1E+00	6.0%	6.5E-05	1.1E-04	28.2%	5.0E-04	1.7E+00	6.0%
Dissolved Barium	ND	NA	0.07	0.0E+00	--	--	0.0E+00	0.0E+00	0.0%	0.0E+00	--	--	0.0E+00	0.0E+00	0.0%
Dissolved Lead	ND	NA	NA	0.0E+00	--	--	0.0E+00	--	--	0.0E+00	--	--	0.0E+00	--	--
Dissolved Manganese	0.156	NA	0.005	2.3E-03	--	--	5.4E-03	1.1E+00	6.0%	1.1E-03	--	--	8.4E-03	1.7E+00	6.0%
					Total ILCR:	8.6E-04		HI:	1.8E+01	100.0%	Total ILCR:	4.0E-04		HI:	2.8E+01	100.0%

NOTES:
NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-31
ON-SITE RESIDENTS - AREA A - FUTURE SCENARIO
RESIDENTIAL DERMAL CONTACT WITH SHALLOW GROUNDWATER FROM LOCATION B-20WSS
ORGANIC AND FILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	20000	8023	
EF	Exposure frequency (d/yr)	350	350	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/even	CS	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	0.2	0.2	

							Adult Residents						Child Residents (0 - 6 Yrs Old)					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethene	1.6	1.0E-02	7.2E-03	1.2E-05	NA	0.009	1.4E-03	--	--	3.2E-03	3.5E-01	13.2%	5.1E-04	--	--	5.9E-03	6.6E-01	13.2%
4-methyl-2-pentanone	0.22	3.3E-03	2.5E-03	5.4E-07	NA	0.08	6.3E-05	--	--	1.5E-04	1.8E-03	0.1%	2.4E-05	--	--	2.8E-04	3.5E-03	0.1%
Benzene	ND	2.1E-01	1.3E-01	NA	0.029	NA	--	--	--	--	--	--	--	--	--	--	--	--
Methylene chloride	0.057	4.5E-03	3.0E-03	1.7E-07	0.0075	0.06	2.0E-05	1.5E-07	0.1%	4.7E-05	7.8E-04	0.0%	7.5E-06	5.6E-08	0.1%	8.8E-05	1.5E-03	0.0%
Tetrachloroethene	0.62	4.8E-02	5.6E-02	3.5E-05	0.052	0.01	4.1E-03	2.1E-04	86.0%	9.6E-03	9.6E-01	35.9%	1.5E-03	8.0E-05	86.0%	1.8E-02	1.8E+00	35.9%
Toluene	1.2	4.5E-02	3.1E-02	3.8E-05	NA	0.2	4.4E-03	--	--	1.0E-02	5.2E-02	1.9%	1.7E-03	--	--	1.9E-02	9.7E-02	1.9%
Trichloroethene	1.8	1.6E-02	1.5E-02	2.6E-05	0.011	0.006	3.1E-03	3.4E-05	13.8%	7.2E-03	1.2E+00	45.2%	1.2E-03	1.3E-05	13.8%	1.4E-02	2.3E+00	45.2%
Vinyl chloride	ND	7.3E-03	4.1E-03	NA	1.9	NA	--	--	--	--	--	--	--	--	--	--	--	--
2-Methylphenol	0.28	1.0E-02	7.8E-03	2.2E-06	NA	0.05	2.6E-04	--	--	6.0E-04	1.2E-02	0.5%	9.6E-05	--	--	1.1E-03	2.2E-02	0.5%
2,4-Dimethylphenol	0.025	1.5E-02	1.3E-02	3.2E-07	NA	0.02	3.8E-05	--	--	8.9E-05	4.4E-03	0.2%	1.4E-05	--	--	1.7E-04	8.3E-03	0.2%
4-Methylphenol	0.1	1.8E-02	1.4E-02	1.4E-06	NA	0.005	1.7E-04	--	--	3.9E-04	7.7E-02	2.9%	6.2E-05	--	--	7.2E-04	1.4E-01	2.9%
Bis(2-ethylhexyl)phthalate	ND	3.3E-02	1.9E-01	NA	0.014	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Aldrin	ND	1.6E-03	7.7E-03	NA	17	0.00003	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	6.7E-04	3.8E-03	NA	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Arsenic	0.0118	1.0E-03	NA	2.4E-09	1.75	0.0003	2.8E-07	4.8E-07	0.2%	6.5E-07	2.2E-03	0.1%	1.0E-07	1.8E-07	0.2%	1.2E-06	4.0E-03	0.1%
Dissolved Barium	ND	1.0E-03	NA	NA	NA	0.07	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Lead	ND	1.0E-03	NA	NA	NA	NA	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Manganese	0.198	1.0E-03	NA	4.0E-08	NA	0.005	4.6E-06	--	--	1.1E-05	2.2E-03	0.1%	1.7E-06	--	--	2.0E-05	4.1E-03	0.1%
							Total ILCR:	2.5E-04	100.0%	HI:	2.7E+00	100.0%	Total ILCR:	9.3E-05	100.0%	HI:	5.0E+00	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, tegimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-32
ON-SITE RESIDENTS - AREA A - FUTURE SCENARIO
RESIDENTIAL INGESTION OF DEEP (YORKTOWN) GROUNDWATER FROM LOCATION A-MW1B
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DALY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/d)	2	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.038	0.091	NA	4.5E-04	4.1E-05	1.5%	1.0E-03	--	--	2.1E-04	1.9E-05	1.5%	1.9E-03	--	--
1,2-Dichloroethene	0.54	NA	0.009	6.3E-03	--	--	1.5E-02	1.6E+00	22.9%	3.0E-03	--	--	2.3E-02	2.6E+00	22.9%
Benzene	ND	0.029	NA	--	--	--	--	--	--	--	--	--	--	--	--
Chloroform	ND	0.0061	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene	ND	0.052	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Trichloroethene	0.1	0.011	0.006	1.2E-03	1.3E-05	0.5%	2.7E-03	4.6E-01	6.4%	5.5E-04	6.0E-06	0.5%	4.3E-03	7.1E-01	6.4%
Vinyl chloride	0.1	1.9	NA	1.2E-03	2.2E-03	83.2%	2.7E-03	--	--	5.5E-04	1.0E-03	83.2%	4.3E-03	--	--
Bis(2-chloroethyl)ether	ND	1.1	NA	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Aluminum	8.56	NA	1	1.0E-01	--	--	2.3E-01	2.3E-01	3.3%	4.7E-02	--	--	3.6E-01	3.6E-01	3.3%
Arsenic	0.0193	1.75	0.0003	2.3E-04	4.0E-04	14.8%	5.3E-04	1.8E+00	24.6%	1.1E-04	1.9E-04	14.8%	8.2E-04	2.7E+00	24.6%
Chromium	0.0259	NA	0.005	3.0E-04	--	--	7.1E-04	1.4E-01	2.0%	1.4E-04	--	--	1.1E-03	2.2E-01	2.0%
Lead	0.0098	NA	NA	1.2E-04	--	--	2.7E-04	--	--	5.4E-05	--	--	4.2E-04	--	--
Manganese	0.49	NA	0.005	5.8E-03	--	--	1.3E-02	2.7E+00	37.4%	2.7E-03	--	--	2.1E-02	4.2E+00	37.4%
Thallium	ND	NA	0.00008	--	--	--	--	--	--	--	--	--	--	--	--
Vanadium	0.064	NA	0.007	7.5E-04	--	--	1.8E-03	2.5E-01	3.5%	3.5E-04	--	--	2.7E-03	3.9E-01	3.5%
				Total ILCR:	2.7E-03	100.0%	HI:	7.2E+00	100.0%	Total ILCR:	1.3E-03	100.0%	HI:	1.1E+01	100.0%

NOTES:
NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-33
ON-SITE RESIDENTS - AREA A - FUTURE SCENARIO
RESIDENTIAL DERMAL CONTACT WITH DEEP (YORKTOWN) GROUNDWATER FROM LOCATION A-MW1B
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	20000	8023	
EF	Exposure frequency (d/yr)	350	350	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/even	CS	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	0.2	0.2	

							Adult Residents						Child Residents (0 - 6 Yrs Old)					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.038	5.3E-03	3.9E-03	1.5E-07	0.091	NA	1.7E-05	1.6E-06	1.6%	4.0E-05	--	--	6.5E-06	5.9E-07	1.6%	7.6E-05	--	--
1,2-Dichloroethene	0.54	1.0E-02	7.2E-03	3.9E-06	NA	0.009	4.6E-04	--	--	1.1E-03	1.2E-01	60.7%	1.7E-04	--	--	2.0E-03	2.2E-01	60.7%
Benzene	ND	2.1E-01	1.3E-01	NA	0.029	NA	--	--	--	--	--	--	--	--	--	--	--	--
Chloroform	ND	8.9E-03	7.5E-03	NA	0.0061	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene	ND	4.8E-02	5.6E-02	NA	0.052	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Trichloroethene	0.1	1.6E-02	1.5E-02	1.5E-06	0.011	0.006	1.7E-04	1.9E-06	2.0%	4.0E-04	6.7E-02	34.3%	6.4E-05	7.1E-07	2.0%	7.5E-04	1.3E-01	34.3%
Vinyl chloride	0.1	7.3E-03	4.1E-03	4.1E-07	1.9	NA	4.9E-05	9.2E-05	95.6%	1.1E-04	--	--	1.8E-05	3.5E-05	95.6%	2.1E-04	--	--
Bis(2-chloroethyl)ether	ND	2.1E-03	2.1E-03	NA	1.1	NA	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	6.7E-04	3.8E-03	NA	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Aluminum	8.56	1.0E-03	NA	1.7E-06	NA	1	2.0E-04	--	--	4.7E-04			7.5E-05	--	--	8.8E-04		
Arsenic	0.0193	1.0E-03	NA	3.9E-09	1.75	0.0003	4.5E-07	7.9E-07	0.8%	1.1E-06	3.5E-03	1.8%	1.7E-07	3.0E-07	0.8%	2.0E-06	6.6E-03	1.8%
Chromium	0.0259	1.0E-03	NA	5.2E-09	NA	0.005	6.1E-07	--	--	1.4E-06	2.8E-04	0.1%	2.3E-07	--	--	2.7E-06	5.3E-04	0.1%
Lead	0.0098	1.0E-03	NA	2.0E-09	NA	NA	2.3E-07	--	--	5.4E-07	--	--	8.6E-08	--	--	1.0E-06	--	--
Manganese	0.49	1.0E-03	NA	9.8E-08	NA	0.005	1.2E-05	--	--	2.7E-05	5.4E-03	2.8%	4.3E-06	--	--	5.0E-05	1.0E-02	2.8%
Thallium	ND	1.0E-03	NA	NA	NA	0.00008	--	--	--	--	--	--	--	--	--	--	--	--
Vanadium	0.064	1.0E-03	NA	1.3E-08	NA	0.007	1.5E-06	--	--	3.5E-06	5.0E-04	0.3%	5.6E-07	--	--	6.6E-06	9.4E-04	0.3%
							Total ILCR:	9.7E-05	100.0%	HI:	2.0E-01	100.0%	Total ILCR:	3.6E-05	100.0%	HI:	3.7E-01	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-34
ON-SITE RESIDENTS - AREA A- FUTURE SCENARIO
INHALATION OF VOLATILIZED ORGANICS IN SHOWER AIR
DEEP (YORKTOWN) GROUNDWATER FROM LOCATION A-MW1B
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (CA*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFI
HQ = CDI/RfDI

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFI	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDI	Oral reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of volatilized chemical in shower air, Andelman Model (mg/m3)	CS	CS	
RR	Respiration rate (m3/hr)	0.83	0.6	
ET	Exposure time (hrs/d)	0.2	0.2	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFI 1/(mg/kg/d)	RfDI (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.095	0.091	0.00286	9.3E-05	8.4E-06	0.1%	2.2E-04	7.6E-02	100.0%	6.2E-05	5.7E-06	0.1%	7.3E-04	2.5E-01	100.0%
1,2-Dichloroethene	9.05	NA	NA	8.8E-03	--	--	2.1E-02	--	--	6.0E-03			6.9E-02	--	--
Trichloroethene	2.33	0.006	NA	2.3E-03	1.4E-05	0.2%	5.3E-03	--	--	1.5E-03	9.2E-06	0.2%	1.8E-02	--	--
Vinyl chloride	20.9	0.3	NA	2.0E-02	6.1E-03	99.6%	4.8E-02	--	--	1.4E-02	4.1E-03	99.6%	1.6E-01	--	--
				Total ILCR:	6.1E-03	100.0%		HI:	7.6E-02	100.0%	Total ILCR:	4.1E-03	100.0%	HI:	2.5E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-35
ON-SITE RESIDENTS - AREA A - FUTURE SCENARIO
RESIDENTIAL INGESTION OF DEEP (YORKTOWN) GROUNDWATER FROM LOCATION A-MW1B
ORGANIC AND FILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/d)	2	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.038	0.091	NA	4.5E-04	4.1E-05	1.8%	1.0E-03	--	--	2.1E-04	1.9E-05	1.8%	1.6E-03	--	--
1,2-Dichloroethene	0.54	NA	0.009	6.3E-03	--	--	1.5E-02	1.6E+00	47.6%	3.0E-03	--	--	2.3E-02	2.6E+00	47.6%
Benzene	ND	0.029	NA	--	--	--	--	--	--	--	--	--	--	--	--
Chloroform	ND	0.0061	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene	ND	0.052	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Trichloroethene	0.1	0.011	0.006	1.2E-03	1.3E-05	0.6%	2.7E-03	4.6E-01	13.2%	5.5E-04	6.0E-06	0.6%	4.3E-03	7.1E-01	13.2%
Vinyl chloride	0.1	1.9	NA	1.2E-03	2.2E-03	97.7%	2.7E-03	--	--	5.5E-04	1.0E-03	97.7%	4.3E-03	--	--
Bis(2-chloroethyl)ether	ND	1.1	NA	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Aluminum	0.693	NA	1	8.1E-03	--	--	1.9E-02	1.9E-02	0.5%	3.8E-03	--	--	3.0E-02	3.0E-02	0.5%
Dissolved Arsenic	ND	1.75	0.0003	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Manganese	0.244	NA	0.005	2.9E-03	--	--	6.7E-03	1.3E+00	38.7%	1.3E-03	--	--	1.0E-02	2.1E+00	38.7%
				Total ILCR:	2.3E-03	100.0%	HI:	3.5E+00	100.0%	Total ILCR:	1.1E-03	100.0%	HI:	5.4E+00	100.0%

NOTES:
NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-36
ON-SITE RESIDENTS - AREA A - FUTURE SCENARIO
RESIDENTIAL DERMAL CONTACT WITH DEEP (YORKTOWN) GROUNDWATER FROM LOCATION A-MW1B
ORGANIC AND FILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	20000	8023	
EF	Exposure frequency (d/yr)	350	350	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/even	CS	CS	
Kp	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	0.2	0.2	

							Adult Residents						*Child Residents (0 - 6 Yrs Old)*					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.038	5.3E-03	3.9E-03	1.5E-07	0.091	NA	1.7E-05	1.6E-06	1.6%	4.0E-05	--	--	6.5E-06	5.9E-07	1.6%	7.6E-05	--	--
1,2-Dichloroethene	0.54	1.0E-02	7.2E-03	3.9E-06	NA	0.009	4.6E-04	--	--	1.1E-03	1.2E-01	63.0%	1.7E-04	--	--	2.0E-03	2.2E-01	63.0%
Benzene	ND	2.1E-01	1.3E-01	NA	0.029	NA	--	--	--	--	--	--	--	--	--	--	--	--
Chloroform	ND	8.9E-03	7.5E-03	NA	0.0061	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene	ND	4.8E-02	5.6E-02	NA	0.052	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Trichloroethene	0.1	1.6E-02	1.5E-02	1.5E-06	0.011	0.006	1.7E-04	1.9E-06	2.0%	4.0E-04	6.7E-02	35.6%	6.4E-05	7.1E-07	2.0%	7.5E-04	1.3E-01	35.6%
Vinyl chloride	0.1	7.3E-03	4.1E-03	4.1E-07	1.9	NA	4.9E-05	9.2E-05	96.4%	1.1E-04	--	--	1.8E-05	3.5E-05	96.4%	2.1E-04	--	--
Bis(2-chloroethyl)ether	ND	2.1E-03	2.1E-03	NA	1.1	NA	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	6.7E-04	3.8E-03	NA	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Aluminum	0.693	1.0E-03	NA	1.4E-07	NA	1	1.6E-05	--	--	3.8E-05	3.8E-05	0.0%	6.1E-06	--	--	7.1E-05	7.1E-05	0.0%
Dissolved Arsenic	ND	1.0E-03	NA	NA	1.75	0.0003	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Manganese	0.244	1.0E-03	NA	4.9E-08	NA	0.005	5.7E-06	--	--	1.3E-05	2.7E-03	1.4%	2.1E-06	--	--	2.5E-05	5.0E-03	1.4%
							Total ILCR:	9.6E-05	100.0%	HI:	1.9E-01	100.0%	Total ILCR:	3.6E-05	100.0%	HI:	3.5E-01	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-37
ON-SITE RESIDENTS - AREA A- FUTURE SCENARIO
ACCIDENTAL INGESTION OF SURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	100	200	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
FI	Fraction of soil ingested from site	1	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.353	7.7	NA	2.1E-07	1.6E-06	2.4%	4.8E-07	--	--	3.9E-07	3.0E-06	2.4%	4.5E-06	--	--
Aluminum	9766	NA	1	5.7E-03	--	--	1.3E-02	1.3E-02	2.1%	1.1E-02	--	--	1.2E-01	1.2E-01	2.1%
Arsenic	63.6	1.75	0.0003	3.7E-05	6.5E-05	97.6%	8.7E-05	2.9E-01	46.0%	7.0E-05	1.2E-04	97.6%	8.1E-04	2.7E+00	46.0%
Barium	880	NA	0.07	5.2E-04	--	--	1.2E-03	1.7E-02	2.7%	9.6E-04	--	--	1.1E-02	1.6E-01	2.7%
Cadmium	74.5	NA	0.0005	4.4E-05	--	--	1.0E-04	2.0E-01	32.3%	8.2E-05	--	--	9.5E-04	1.9E+00	32.3%
Chromium	101	NA	0.005	5.9E-05	--	--	1.4E-04	2.8E-02	4.4%	1.1E-04	--	--	1.3E-03	2.6E-01	4.4%
Copper	394	NA	0.0371	2.3E-04	--	--	5.4E-04	1.5E-02	2.3%	4.3E-04	--	--	5.0E-03	1.4E-01	2.3%
Lead	577	NA	NA	3.4E-04	--	--	7.9E-04	--	--	6.3E-04	--	--	7.4E-03	--	--
Manganese	124	NA	0.005	7.3E-05	--	--	1.7E-04	3.4E-02	5.4%	1.4E-04	--	--	1.6E-03	3.2E-01	5.4%
Thallium	0.920	NA	0.00008	5.4E-07	--	--	1.3E-06	1.6E-02	2.5%	1.0E-06	--	--	1.2E-05	1.5E-01	2.5%
Vanadium	74.0	NA	0.007	4.3E-05	--	--	1.0E-04	1.4E-02	2.3%	8.1E-05	--	--	9.5E-04	1.4E-01	2.3%
				Total ILCR:	6.7E-05	100.0%		HI:	6.3E-01	Total ILCR:	1.2E-04	100.0%		HI:	5.9E+00	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-38
ON-SITE RESIDENTS - AREA A- FUTURE SCENARIO
DERMAL CONTACT WITH SURFACES SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	1	
ABS	Absorption fraction --			
	Cadmium and default organics:	0.01	0.01	
	Default inorganics:	0.001	0.001	
	PCBs	0.06	0.06	
A	Skin surface area available for contact (cm	5300	2000	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	37	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

					Adult Residents						Child Residents (0 - 6 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.353	0.06	7.7	NA	6.6E-07	5.1E-06	4.7%	1.5E-06	--	--	9.4E-08	7.3E-07	4.7%	1.1E-06	--	--
Aluminum	9766	0.01	NA	1	3.0E-03	--	--	7.1E-03	7.1E-03	1.1%	4.4E-04	--	--	5.1E-03	5.1E-03	1.1%
Arsenic	63.6	0.03	1.75	0.0003	5.9E-05	1.0E-04	95.3%	1.4E-04	4.6E-01	71.9%	8.5E-06	1.5E-05	95.3%	9.9E-05	3.3E-01	71.9%
Barium	880	0.01	NA	0.07	2.7E-04	--	--	6.4E-04	9.1E-03	1.4%	3.9E-05	--	--	4.6E-04	6.5E-03	1.4%
Cadmium	74.5	0.01	NA	0.0005	2.3E-05	--	--	5.4E-05	1.1E-01	16.8%	3.3E-06	--	--	3.9E-05	7.7E-02	16.8%
Chromium	101	0.01	NA	0.005	3.1E-05	--	--	7.3E-05	1.5E-02	2.3%	4.5E-06	--	--	5.2E-05	1.0E-02	2.3%
Copper	394	0.01	NA	0.0371	1.2E-04	--	--	2.9E-04	7.7E-03	1.2%	1.8E-05	--	--	2.0E-04	5.5E-03	1.2%
Lead	577	0.01	NA	NA	1.8E-04	--	--	4.2E-04	--	--	2.6E-05	--	--	3.0E-04	--	--
Manganese	124	0.01	NA	0.005	3.9E-05	--	--	9.0E-05	1.8E-02	2.8%	5.5E-06	--	--	6.5E-05	1.3E-02	2.8%
Thallium	0.920	0.01	NA	0.00008	2.9E-07	--	--	6.7E-07	8.3E-03	1.3%	4.1E-08	--	--	4.8E-07	6.0E-03	1.3%
Vanadium	74.0	0.01	NA	0.007	2.3E-05	--	--	5.4E-05	7.7E-03	1.2%	3.3E-06	--	--	3.8E-05	5.5E-03	1.2%
					Total ILCR:	1.1E-04	100.0%	HI:	6.4E-01	100.0%	Total ILCR:	1.6E-05	100.0%	HI:	4.6E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-39
ON-SITE RESIDENTS - AREA A- FUTURE SCENARIO
INHALATION OF FUGITIVE DUSTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d) (Ca*RR*ET*EF*ED)/(BW*AT)
Where: Ca = Cs * (1/PEF)

ILCR = CDI*CSFI
HQ = CDI/RfDI

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFI	Inhalation cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDI	Inhalation reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of chemical in air as fugitive dusts (mg/m3)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
PEF	Particulate emission factor (m3/kg)	4.63E+09	4.63E+09	
RR	Respiration rate (m3/hr)	0.83	0.6	
ET	Exposure time (hrs/d)	1	2	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

					Adult Residents						Child Residents (0 - 6 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	Ca (mg/m3)	CSFI 1/(mg/kg/	RfDI (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.353	7.61E-11	NA	NA	3.7E-13	--	--	8.7E-13	--	--	5.0E-13	--	--	5.8E-12	--	--
Aluminum	9766	2.11E-06	NA	NA	1.0E-08	--	--	2.4E-08	--	--	1.4E-08	--	--	1.6E-07	--	--
Arsenic	63.6	1.37E-08	15.1	0.0003	6.7E-11	1.0E-09	16.9%	1.6E-10	5.2E-07	1.4%	9.0E-11	1.4E-09	16.9%	1.1E-09	3.5E-06	1.4%
Barium	880	1.90E-07	NA	0.000143	9.3E-10	--	--	2.2E-09	1.5E-05	40.8%	1.3E-09	--	--	1.5E-08	1.0E-04	40.8%
Cadmium	74.5	1.61E-08	6.3	NA	7.8E-11	4.9E-10	8.3%	1.8E-10	--	--	1.1E-10	6.7E-10	8.3%	1.2E-09	--	--
Chromium	101	2.18E-08	42	NA	1.1E-10	4.5E-09	74.8%	2.5E-10	--	--	1.4E-10	6.0E-09	74.8%	1.7E-09	--	--
Copper	394	8.51E-08	NA	NA	4.1E-10	--	--	9.7E-10	--	--	5.6E-10	--	--	6.5E-09	--	--
Lead	577	1.25E-07	NA	NA	6.1E-10	--	--	1.4E-09	--	--	8.2E-10	--	--	9.6E-09	--	--
Manganese	124	2.69E-08	NA	0.0000143	1.3E-10	--	--	3.1E-10	2.1E-05	57.7%	1.8E-10	--	--	2.1E-09	1.4E-04	57.7%
Thallium	0.920	1.99E-10	NA	NA	9.7E-13	--	--	2.3E-12	--	--	1.3E-12	--	--	1.5E-11	--	--
Vanadium	74.0	1.60E-08	NA	NA	7.8E-11	--	--	1.8E-10	--	--	1.1E-10	--	--	1.2E-09	--	--
					Total ILCR:	6.0E-09	100.0%	HI:	3.7E-05	100.0%	Total ILCR:	8.0E-09	100.0%	HI:	2.5E-04	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-40
ON-SITE RESIDENTS - AREA A- FUTURE SCENARIO
ACCIDENTAL INGESTION OF SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*ET*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Local Adult Resident	Local Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/hour)	0.05	0.05	
ET	Exposure time (hours/event)	2.6	2.6	
EF	Exposure Frequency (events/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Tetrachloroethene	0.0053	0.052	0.01	3.9E-07	2.1E-08	0.5%	9.2E-07	9.2E-05	0.3%	1.5E-06	7.7E-08	0.5%	1.7E-05	1.7E-03	0.3%
Trichloroethene	0.0096	0.011	0.006	7.1E-07	7.8E-09	0.2%	1.7E-06	2.8E-04	0.9%	2.7E-06	2.9E-08	0.2%	3.1E-05	5.2E-03	0.9%
Vinyl chloride	0.0053	1.9	NA	3.9E-07	7.5E-07	17.8%	9.2E-07	--	--	1.5E-06	2.8E-06	17.8%	1.7E-05	--	--
Xylenes(total)	0.003	NA	2	2.2E-07	--	--	5.2E-07	2.6E-07	0.0%	8.4E-07	--	--	9.8E-06	4.9E-06	0.0%
Bis(2-ethylhexyl)phthalate	0.003	0.014	0.02	2.2E-07	3.1E-09	0.1%	5.2E-07	2.6E-05	0.1%	8.4E-07	1.2E-08	0.1%	9.8E-06	4.9E-04	0.1%
4,4'-DDD	0.00013	0.24	NA	9.6E-09	2.3E-09	0.1%	2.2E-08	--	--	3.6E-08	8.6E-09	0.1%	4.2E-07	--	--
4,4'-DDE	0.000056	0.34	NA	4.2E-09	1.4E-09	0.0%	9.7E-09	--	--	1.6E-08	5.3E-09	0.0%	1.8E-07	--	--
alpha-Chlordane	0.000015	1.3	0.00006	1.1E-09	1.4E-09	0.0%	2.6E-09	4.3E-05	0.1%	4.2E-09	5.4E-09	0.0%	4.9E-08	8.1E-04	0.1%
Aroclor-1254	0.00044	7.7	NA	3.3E-08	2.5E-07	6.0%	7.6E-08	--	--	1.2E-07	9.4E-07	6.0%	1.4E-06	--	--
delta-BHC	0.000025	1.8	NA	1.9E-09	3.3E-09	0.1%	4.3E-09	--	--	7.0E-09	1.3E-08	0.1%	8.1E-08	--	--
Dieldrin	0.000027	16	0.00005	2.0E-09	3.2E-08	0.8%	4.7E-09	9.3E-05	0.3%	7.5E-09	1.2E-07	0.8%	8.8E-08	1.8E-03	0.3%
gamma-Chlordane	0.000024	1.3	0.00006	1.8E-09	2.3E-09	0.1%	4.2E-09	6.9E-05	0.2%	6.7E-09	8.7E-09	0.1%	7.8E-08	1.3E-03	0.2%
Heptachlor epoxide	0.000006	9.1	0.000013	4.4E-10	4.0E-09	0.1%	1.0E-09	8.0E-05	0.3%	1.7E-09	1.5E-08	0.1%	2.0E-08	1.5E-03	0.3%
Aluminum	6.85	NA	1	5.1E-04	--	--	1.2E-03	1.2E-03	4.0%	1.9E-03	--	--	2.2E-02	2.2E-02	4.0%
Arsenic	0.024	1.75	0.0003	1.8E-06	3.1E-06	74.4%	4.2E-06	1.4E-02	47.1%	6.7E-06	1.2E-05	74.4%	7.9E-05	2.6E-01	47.1%
Barium	0.165	NA	0.07	1.2E-05	--	--	2.9E-05	4.1E-04	1.4%	4.6E-05	--	--	5.4E-04	7.7E-03	1.4%
Lead	0.261747	NA	NA	1.9E-05	--	--	4.5E-05	--	--	7.3E-05	--	--	8.5E-04	--	--
Manganese	0.371	NA	0.005	2.7E-05	--	--	6.4E-05	1.3E-02	43.4%	1.0E-04	--	--	1.2E-03	2.4E-01	43.4%
Mercury	0.0013	0.0003	NA	1.0E-07	3.0E-11	0.0%	2.3E-07	--	--	3.8E-07	1.1E-10	0.0%	4.4E-06	--	--
Silver	0.00459	NA	0.005	3.4E-07	--	--	7.9E-07	1.6E-04	0.5%	1.3E-06	--	--	1.5E-05	3.0E-03	0.5%
Zinc	0.620	NA	0.3	4.6E-05	--	--	1.1E-04	3.6E-04	1.2%	1.7E-04	--	--	2.0E-03	6.7E-03	1.2%
				Total ILCR:	4.2E-06	100.0%		HI:	3.0E-02	100.0%	Total ILCR:	1.6E-05	100.0%	HI:	5.6E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-41
ON-SITE RESIDENTS - AREA A- FUTURE SCENARIO
DERMAL CONTACT WITH SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Local Adult Resident	Local Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	5300	2006	
EF	Exposure frequency (d/yr)	34	137	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	16	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/event)	CS	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	2.6	2.6	

							Adult Residents						Child Residents (0 - 6 Yrs Old)					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Tetrachloroethene	0.0053	0.052	2.0E-01	1.1E-06	0.052	0.01	3.3E-06	1.7E-07	0.1%	7.6E-06	7.6E-04	6.7%	4.7E-06	2.4E-07	0.1%	5.4E-05	5.4E-03	6.7%
Trichloroethene	0.0096	0.011	5.9E-02	5.7E-07	0.011	0.006	1.7E-06	1.9E-08	0.0%	4.0E-06	6.7E-04	5.9%	2.4E-06	2.7E-08	0.0%	2.8E-05	4.7E-03	5.9%
Vinyl chloride	0.0053	1.9	2.2E-02	1.2E-07	1.9	NA	3.5E-07	6.7E-07	0.5%	8.3E-07	--	--	5.0E-07	9.6E-07	0.5%	5.9E-06	--	--
Xylenes(total)	0.003	NA	2.0E-01	6.1E-07	NA	2	1.8E-06	--	--	4.3E-06	2.1E-06	0.0%	2.6E-06	--	--	3.1E-05	1.5E-05	0.0%
Bis(2-ethylhexyl)phthalate	0.003	3.3E-02	6.7E-01	2.0E-06	0.014	0.02	6.1E-06	8.6E-08	0.1%	1.4E-05	7.1E-04	6.3%	8.7E-06	1.2E-07	0.1%	1.0E-04	5.1E-03	6.3%
4,4'-DDD	0.00013	2.8E-01	3.5E+00	4.5E-07	0.24	NA	1.4E-06	3.3E-07	0.3%	3.2E-06	--	--	1.9E-06	4.6E-07	0.3%	2.3E-05	--	--
4,4'-DDE	0.000056	2.4E-01	2.9E+00	1.7E-07	0.34	NA	5.0E-07	1.7E-07	0.1%	1.2E-06	--	--	7.1E-07	2.4E-07	0.1%	8.3E-06	--	--
alpha-Chlordane	0.000015	4.6E-02	1.1E+00	1.6E-08	1.3	0.00006	4.9E-08	6.4E-08	0.1%	1.1E-07	1.9E-03	16.8%	7.0E-08	9.1E-08	0.1%	8.2E-07	1.4E-02	16.8%
Aroclor-1254	0.00044	3.7E-01	1.2E+01	5.3E-06	7.7	NA	1.6E-05	1.2E-04	98.1%	3.7E-05	--	--	2.3E-05	1.7E-04	98.1%	2.6E-04	--	--
delta-BHC	0.000025	2.6E-02	2.6E-01	6.6E-09	1.8	NA	2.0E-08	3.6E-08	0.0%	4.7E-08	--	--	2.8E-08	5.1E-08	0.0%	3.3E-07	--	--
Dieldrin	0.000027	1.6E-02	3.0E-01	8.2E-09	16	0.00005	2.5E-08	4.0E-07	0.3%	5.8E-08	1.2E-03	10.1%	3.5E-08	5.6E-07	0.3%	4.1E-07	8.2E-03	10.1%
gamma-Chlordane	0.000024	4.6E-02	1.1E+00	2.6E-08	1.3	0.00006	7.9E-08	1.0E-07	0.1%	1.8E-07	3.1E-03	26.9%	1.1E-07	1.5E-07	0.1%	1.3E-06	2.2E-02	26.9%
Heptachlor epoxide	0.000006	6.7E-04	1.4E-02	8.1E-11	9.1	0.000013	2.5E-10	2.2E-09	0.0%	5.7E-10	4.4E-05	0.4%	3.5E-10	3.2E-09	0.0%	4.1E-09	3.1E-04	0.4%
Aluminum	6.85	1.0E-03	NA	1.8E-05	NA	1	5.4E-05	--	--	1.3E-04	1.3E-04	1.1%	7.7E-05	--	--	8.9E-04	8.9E-04	1.1%
Arsenic	0.024	1.0E-03	NA	6.3E-08	1.75	0.0003	1.9E-07	3.3E-07	0.3%	4.4E-07	1.5E-03	13.0%	2.7E-07	4.7E-07	0.3%	3.2E-06	1.1E-02	13.0%
Barium	0.165	1.0E-03	NA	4.3E-07	NA	0.07	1.3E-06	--	--	3.0E-06	4.3E-05	0.4%	1.8E-06	--	--	2.2E-05	3.1E-04	0.4%
Lead	0.261747	1.0E-03	NA	6.8E-07	NA	NA	2.1E-06	--	--	4.8E-06	--	--	2.9E-06	--	--	3.4E-05	--	--
Manganese	0.371	1.0E-03	NA	9.6E-07	NA	0.005	2.9E-06	--	--	6.8E-06	1.4E-03	12.0%	4.1E-06	--	--	4.8E-05	9.7E-03	12.0%
Mercury	0.0013	1.0E-03	NA	3.5E-09	0.0003	NA	1.1E-08	3.2E-12	0.0%	2.5E-08	--	--	1.5E-08	4.5E-12	0.0%	1.8E-07	--	--
Silver	0.00459	1.0E-03	NA	1.2E-08	NA	0.005	3.6E-08	--	--	8.4E-08	1.7E-05	0.1%	5.1E-08	--	--	6.0E-07	1.2E-04	0.1%
Zinc	0.620	1.0E-03	NA	1.6E-06	NA	0.3	4.9E-06	--	--	1.1E-05	3.8E-05	0.3%	6.9E-06	--	--	8.1E-05	2.7E-04	0.3%
							Total ILCR:	1.2E-04	100.0%	HI:	1.1E-02	100.0%	Total ILCR:	1.8E-04	100.0%	HI:	8.1E-02	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-42
ON-SITE RESIDENTS - AREA A- FUTURE SCENARIO
ACCIDENTAL INGESTION OF SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d) (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Local Adult Resident	Local Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	100	200	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
FI	Fraction of sediment ingested from site	1	1	
EF	Exposure Frequency (d/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.355	7.7	NA	5.4E-08	4.2E-07	3.4%	1.3E-07	--	--	4.1E-07	3.2E-06	3.4%	4.8E-06	--	--
4,4'-DDE	0.103	0.34	NA	5.9E-09	2.0E-09	0.0%	1.4E-08	--	--	4.4E-08	1.5E-08	0.0%	5.2E-07	--	--
4,4'-DDT	0.049	0.34	NA	2.8E-09	9.4E-10	0.0%	6.5E-09	--	--	2.1E-08	7.1E-09	0.0%	2.4E-07	--	--
Arsenic	118	1.75	0.0003	6.7E-06	1.2E-05	96.5%	1.6E-05	5.2E-02	60.4%	5.1E-05	8.9E-05	96.5%	5.9E-04	2.0E+00	60.4%
Cadmium	48.6	NA	0.0005	2.8E-06	--	--	6.5E-06	1.3E-02	14.9%	2.1E-05	--	--	2.4E-04	4.9E-01	14.9%
Chromium	588	NA	0.005	3.4E-05	--	--	7.8E-05	1.6E-02	18.1%	2.5E-04	--	--	2.9E-03	5.9E-01	18.1%
Copper	553	NA	0.0371	3.2E-05	--	--	7.4E-05	2.0E-03	2.3%	2.4E-04	--	--	2.8E-03	7.5E-02	2.3%
Lead	299	NA	NA	1.7E-05	--	--	4.0E-05	--	--	1.3E-04	--	--	1.5E-03	--	--
Manganese	51.2	NA	0.005	2.9E-06	--	--	6.8E-06	1.4E-03	1.6%	2.2E-05	--	--	2.6E-04	5.1E-02	1.6%
Mercury	0.770	NA	0.0003	4.4E-08	--	--	1.0E-07	3.4E-04	0.4%	3.3E-07	--	--	3.9E-06	1.3E-02	0.4%
Silver	19.5	NA	0.005	1.1E-06	--	--	2.6E-06	5.2E-04	0.6%	8.3E-06	--	--	9.7E-05	1.9E-02	0.6%
Vanadium	77.1	NA	0.007	4.4E-06	--	--	1.0E-05	1.5E-03	1.7%	3.3E-05	--	--	3.9E-04	5.5E-02	1.7%
				Total ILCR:	1.2E-05	100.0%		HI:	8.7E-02	Total ILCR:	9.2E-05	100.0%		HI:	3.3E+00	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-43
ON-SITE RESIDENTS - AREA A- FUTURE SCENARIO
DERMAL CONTACT WITH SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Local Adult Resident	Local Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	1	
ABS	Absorption fraction --			
	Cadmium and default organics:	0.01	0.01	
	Default inorganics:	0.001	0.001	
	PCBs	0.06	0.06	
A	Skin surface area available for contact (cm2)	5300	2006	
EF	Exposure Frequency (d/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

					Adult Residents						Child Residents (0 - 6 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.955	0.06	7.7	NA	1.7E-07	1.3E-06	6.6%	4.0E-07	--	--	2.5E-07	1.9E-06	6.6%	2.9E-06	--	--
4,4'-DDE	0.103	0.1	0.34	NA	3.1E-08	1.1E-08	0.1%	7.3E-08	--	--	4.5E-08	1.5E-08	0.1%	5.2E-07	--	--
4,4'-DDT	0.049	0.1	0.34	NA	1.5E-08	5.0E-09	0.0%	3.4E-08	--	--	2.1E-08	7.1E-09	0.0%	2.4E-07	--	--
Arsenic	118	0.03	1.75	0.0003	1.1E-05	1.9E-05	93.3%	2.5E-05	8.3E-02	82.1%	1.5E-05	2.7E-05	93.3%	1.8E-04	5.9E-01	82.1%
Cadmium	48.6	0.01	NA	0.0005	1.5E-06	--	--	3.4E-06	6.9E-03	6.8%	2.1E-06	--	--	2.4E-05	4.9E-02	6.8%
Chromium	588	0.01	NA	0.005	1.8E-05	--	--	4.1E-05	8.3E-03	8.2%	2.5E-05	--	--	3.0E-04	5.9E-02	8.2%
Copper	553	0.01	NA	0.0371	1.7E-05	--	--	3.9E-05	1.1E-03	1.0%	2.4E-05	--	--	2.8E-04	7.5E-03	1.0%
Lead	299	0.01	NA	NA	9.0E-06	--	--	2.1E-05	--	--	1.3E-05	--	--	1.5E-04	--	--
Manganese	51.2	0.01	NA	0.005	1.5E-06	--	--	3.6E-06	7.2E-04	0.7%	2.2E-06	--	--	2.6E-05	5.1E-03	0.7%
Mercury	0.770	0.01	NA	0.0003	2.3E-08	--	--	5.4E-08	1.8E-04	0.2%	3.3E-08	--	--	3.9E-07	1.3E-03	0.2%
Silver	19.5	0.01	NA	0.005	5.9E-07	--	--	1.4E-06	2.7E-04	0.3%	8.4E-07	--	--	9.8E-06	2.0E-03	0.3%
Vanadium	77.1	0.01	NA	0.007	2.3E-06	--	--	5.4E-06	7.8E-04	0.8%	3.3E-06	--	--	3.9E-05	5.5E-03	0.8%
					Total ILCR:	2.0E-05	100.0%	HI:	1.0E-01	100.0%	Total ILCR:	2.9E-05	100.0%	HI:	7.2E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-44
ON-SITE RESIDENTS - AREA A- FUTURE SCENARIO
ACCIDENTAL INGESTION OF DEEP SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Local Adult Resident	Local Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	100	200	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
FI	Fraction of sediment ingested from site	1	1	
EF	Exposure Frequency (d/yr)	34	137	
ED	Exposure Duration (yrs)	25	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Acenaphthene	4.1	NA	0.06	2.3E-07	--	--	5.5E-07	9.1E-06	0.0%	1.8E-06	--	--	2.1E-05	3.4E-04	0.0%
Aroclor-1254	0.980	7.7	NA	5.6E-08	4.3E-07	5.7%	1.3E-07	--	--	4.2E-07	3.2E-06	5.7%	4.9E-06	--	--
4,4'-DDE	0.085	0.34	NA	4.8E-09	1.6E-09	0.0%	1.1E-08	--	--	3.6E-08	1.2E-08	0.0%	4.3E-07	--	--
Dieldrin	0.062	16	0.00005	3.5E-09	5.7E-08	0.8%	8.3E-09	1.7E-04	0.2%	2.7E-08	4.3E-07	0.8%	3.1E-07	6.2E-03	0.2%
Arsenic	70.4	1.75	0.0003	4.0E-06	7.0E-06	93.5%	9.4E-06	3.1E-02	29.0%	3.0E-05	5.3E-05	93.5%	3.5E-04	1.2E+00	29.0%
Cadmium	138	NA	0.0005	7.9E-06	--	--	1.8E-05	3.7E-02	34.1%	5.9E-05	--	--	6.9E-04	1.4E+00	34.1%
Chromium	1319	NA	0.005	7.5E-05	--	--	1.8E-04	3.5E-02	32.5%	5.7E-04	--	--	6.6E-03	1.3E+00	32.5%
Lead	434	NA	NA	2.5E-05	--	--	5.8E-05	--	--	1.9E-04	--	--	2.2E-03	--	--
Manganese	50.7	NA	0.005	2.9E-06	--	--	6.7E-06	1.3E-03	1.3%	2.2E-05	--	--	2.5E-04	5.1E-02	1.3%
Mercury	1.1	NA	0.0003	6.3E-08	--	--	1.5E-07	4.9E-04	0.5%	4.7E-07	--	--	5.5E-06	1.8E-02	0.5%
Silver	41.1	NA	0.005	2.3E-06	--	--	5.5E-06	1.1E-03	1.0%	1.8E-05	--	--	2.1E-04	4.1E-02	1.0%
Vanadium	74.0	NA	0.007	4.2E-06	--	--	9.8E-06	1.4E-03	1.3%	3.2E-05	--	--	3.7E-04	5.3E-02	1.3%
Zinc	542	NA	0.3	3.1E-05	--	--	7.2E-05	2.4E-04	0.2%	2.3E-04	--	--	2.7E-03	9.0E-03	0.2%
				Total ILCR:	7.5E-06	100.0%	HI:	1.1E-01	100.0%	Total ILCR:	5.7E-05	100.0%	HI:	4.1E+00	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-45
ON-SITE RESIDENTS - AREA A- FUTURE SCENARIO
DERMAL CONTACT WITH DEEP SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d) (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Local Adult Resident	Local Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	1	
ABS	Absorption fraction --			
	Cadmium and default organics:	0.01	0.01	
	Default inorganics:	0.001	0.001	
	PCBs	0.06	0.06	
A	Skin surface area available for contact (cm2)	5300	2006	
EF	Exposure Frequency (d/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

					Adult Residents						Child Residents (0 - 6 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Acenaphthene	4.1	0.1	NA	0.06	1.2E-06	--	--	2.9E-06	4.8E-05	0.1%	1.8E-06	--	--	2.1E-05	3.4E-04	0.1%
Aroclor-1254	0.980	0.06	7.7	NA	1.8E-07	1.4E-06	10.6%	4.1E-07	--	--	2.5E-07	1.9E-06	10.6%	3.0E-06	--	--
4,4'-DDE	0.095	0.1	0.34	NA	2.6E-08	8.7E-09	0.1%	6.0E-08	--	--	3.7E-08	1.2E-08	0.1%	4.3E-07	--	--
Dieldrin	0.062	0.1	16	0.00005	1.9E-08	3.0E-07	2.3%	4.4E-08	8.7E-04	1.0%	2.7E-08	4.3E-07	2.3%	3.1E-07	6.2E-03	1.0%
Arsenic	70.4	0.03	1.75	0.0003	6.4E-06	1.1E-05	87.0%	1.5E-05	5.0E-02	54.5%	9.1E-06	1.6E-05	87.0%	1.1E-04	3.5E-01	54.5%
Cadmium	138	0.01	NA	0.0005	4.2E-06	--	--	9.7E-06	1.9E-02	21.4%	5.9E-06	--	--	6.9E-05	1.4E-01	21.4%
Chromium	1319	0.01	NA	0.005	4.0E-05	--	--	9.3E-05	1.9E-02	20.4%	5.7E-05	--	--	6.6E-04	1.3E-01	20.4%
Lead	434	0.01	NA	NA	1.3E-05	--	--	3.1E-05	--	--	1.9E-05	--	--	2.2E-04	--	--
Manganese	50.7	0.01	NA	0.005	1.5E-06	--	--	3.6E-06	7.2E-04	0.8%	2.2E-06	--	--	2.5E-05	5.1E-03	0.8%
Mercury	1.1	0.01	NA	0.0003	3.3E-08	--	--	7.8E-08	2.6E-04	0.3%	4.7E-08	--	--	5.5E-07	1.8E-03	0.3%
Silver	41.1	0.01	NA	0.005	1.2E-06	--	--	2.9E-06	5.8E-04	0.6%	1.8E-06	--	--	2.1E-05	4.1E-03	0.6%
Vanadium	74.0	0.01	NA	0.007	2.2E-06	--	--	5.2E-06	7.5E-04	0.8%	3.2E-06	--	--	3.7E-05	5.3E-03	0.8%
Zinc	542	0.01	NA	0.3	1.6E-05	--	--	3.8E-05	1.3E-04	0.1%	2.3E-05	--	--	2.7E-04	9.1E-04	0.1%
					Total ILCR:	1.3E-05	100.0%		HI: 9.1E-02	100.0%	Total ILCR:	1.8E-05	100.0%		HI: 6.5E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-46
ON-SITE RESIDENTS - AREA A- FUTURE SCENARIO
INHALATION OF OUTDOOR AMBIENT AIR
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFI
HQ = CDI/RfDI

Parameter	Description	Local Adult Resident	Local Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFI	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDI	Oral reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of chemical in air (mg/m3)	CS	CS	
RR	Respiration rate (m3/hr)	0.83	0.6	
ET	Exposure time (hrs/d)	1	2	
EF	Exposure Frequency (d/yr)	150	150	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFI 1/(mg/kg/d)	RfDI (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2,4-Trimethylbenzene	0.0003	NA	NA	6.3E-07	--	--	1.5E-06	--	--	8.5E-07	--	--	9.9E-06	--	--
Benzene	0.0007	0.029	0.00171	1.5E-06	4.2E-08	46.8%	3.4E-06	2.0E-03	26.1%	2.0E-06	5.7E-08	46.8%	2.3E-05	1.3E-02	26.1%
Bromomethane	0.0008	NA	0.00143	1.7E-06	--	--	3.9E-06	2.7E-03	35.7%	2.3E-06	--	--	2.6E-05	1.8E-02	35.7%
Hexachlorobutadiene	0.0003	0.077	0.0005	6.3E-07	4.8E-08	53.2%	1.5E-06	2.9E-03	38.2%	8.5E-07	6.5E-08	53.2%	9.9E-06	2.0E-02	38.2%
				Total ILCR:	9.1E-08	100.0%		HI:	7.6E-03	100.0%	Total ILCR:	1.2E-07	100.0%	HI:	5.2E-02	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-47
CONSTRUCTION WORKERS - AREA A- FUTURE SCENARIO
ACCIDENTAL INGESTION OF SUBSURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Future Construction Worker	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
IR	Ingestion Rate (mg/d)	480	
CF	Conversion factor (kg/mg)	1E-06	
FI	Fraction of soil ingested from site	1	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	1	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	365	

				Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Toluene	984	NA	0.2	6.6E-05	--	--	4.6E-03	2.3E-02	84.2%
Aroclor-1254	0.603	7.7	NA	4.0E-08	3.1E-07	36.6%	2.8E-06	--	--
Aroclor-1260	0.948	7.7	NA	6.4E-08	4.9E-07	57.6%	4.5E-06	--	--
Dieldrin	0.046	16	0.00005	3.1E-09	4.9E-08	5.8%	2.2E-07	4.3E-03	15.8%
				Total ILCR:	8.5E-07	100.0%	HI:	2.7E-02	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-48
CONSTRUCTION WORKERS - AREA A- FUTURE SCENARIO
DERMAL CONTACT WITH SUBSURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Future Construction Worker	
DAD	Dermally absorbed dose (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
CF	Conversion factor (kg/mg)	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	
ABS	Absorption fraction --		
	Cadmium and default organics:	0.01	
	Default inorganics:	0.001	
	PCBs	0.06	
A	Skin surface area available for contact (cm2)	4100	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	1	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	365	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Toluene	984	0.25	NA	0.2	1.4E-04	--	--	9.9E-03	4.9E-02	93.0%
Aroclor-1254	0.603	0.06	7.7	NA	2.1E-08	1.6E-07	35.3%	1.5E-06	--	--
Aroclor-1260	0.948	0.06	7.7	NA	3.3E-08	2.5E-07	55.4%	2.3E-06	--	--
Dieldrin	0.046	0.1	16	0.00005	2.6E-09	4.2E-08	9.3%	1.8E-07	3.7E-03	7.0%
					Total ILCR:	4.5E-07	100.0%		HI: 5.3E-02	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-49
CONSTRUCTION WORKERS - AREA A- FUTURE SCENARIO
INHALATION OF FUGITIVE DUSTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)
Where: Ca = Cs * (1/PEF)

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	Future Construction Worker	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFi	Inhalation cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDi	Inhalation reference dose (mg/kg/d)	CS	
Ca	Concentration of chemical in air as fugitive dusts (mg/m3)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
PEF	Particulate emission factor (m3/kg)	4.63E+09	
RR	Respiration rate (m3/hr)	0.83	
ET	Exposure time (hrs/d)	8	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	1	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	365	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	Ca (mg/m3)	CSFi 1/(mg/kg/	RfDi (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Toluene	984	2.12E-07	NA	0.114	2.0E-10	--	--	1.4E-08	1.2E-07	100.0%
Aroclor-1254	0.603	1.30E-10	NA	NA	1.2E-13	--	--	8.5E-12	--	--
Aroclor-1260	0.948	2.05E-10	NA	NA	1.9E-13	--	--	1.3E-11	--	--
Dieldrin	0.046	9.94E-12	16.1	NA	9.2E-15	1.5E-13	100.0%	6.5E-13	--	--
					Total ILCR:	1.5E-13	100.0%	HI:	1.2E-07	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-50
ADULT WORKERS - AREA B LANDFILL AND POND- CURRENT SCENARIO [1]
ACCIDENTAL INGESTION OF SURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Adult Worker	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
IR	Ingestion Rate (mg/d)	50	
CF	Conversion factor (kg/mg)	1E-06	
FI	Fraction of soil ingested from site	1	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

				Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.780	7.7	NA	1.4E-07	1.0E-06	22.8%	3.8E-07	--	--
Arsenic	11.6	1.75	0.0003	2.0E-06	3.5E-06	77.2%	5.7E-06	1.9E-02	35.5%
Cadmium	20.5	NA	0.0005	3.6E-06	--	--	1.0E-05	2.0E-02	37.6%
Chromium	44	NA	0.005	7.7E-06	--	--	2.2E-05	4.3E-03	8.1%
Manganese	102	NA	0.005	1.8E-05	--	--	5.0E-05	1.0E-02	18.7%
				Total ILCR:	4.6E-06	100.0%	HI:	5.3E-02	100.0%

NOTES:
[1] Adult workers include base employees and brig awardees who currently enter the Area B landfill and pond area to perform routine lawn and maintenence work. Conservatively assumes exposures occur 250 days/year for 25 years.
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-51
ADULT WORKERS - AREA B LANDFILL AND POND- CURRENT SCENARIO [1]
DERMAL CONTACT WITH SURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Adult Worker	
DAD	Dermally absorbed dose (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
CF	Conversion factor (kg/mg)	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	
ABS	Absorption fraction --		
	Cadmium and default organics:	0.01	
	Default inorganics:	0.001	
	PCBs	0.06	
A	Skin surface area available for contact (cm2)	4100	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.78	0.06	7.7	NA	6.7E-07	5.2E-06	37.2%	1.9E-06	--	--
Arsenic	11.6	0.03	1.75	0.0003	5.0E-06	8.7E-06	62.8%	1.4E-05	4.7E-02	62.3%
Cadmium	20.5	0.01	NA	0.0005	--	--	--	8.2E-06	1.6E-02	22.0%
Chromium	44.3	0.01	NA	0.005	--	--	--	1.8E-05	3.6E-03	4.8%
Manganese	102	0.01	NA	0.005	--	--	--	4.1E-05	8.2E-03	11.0%
					Total ILCR:	1.4E-05	100.0%	HI:	7.5E-02	100.0%

NOTES:
[1] Adult workers include base employees and brig awardees who currently enter the Area B landfill and pond area to perform routine lawn and maintenence work. Conservatively assumes exposures occur 250 days/year for 25 years.
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-52
ADULT WORKERS - AREA B LANDFILL AND POND- CURRENT SCENARIO [1]
INHALATION OF FUGITIVE DUSTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)
Where: Ca = Cs * (1/PEF)

ILCR = CDI*CSFI
HQ = CDI/RfDI

Parameter	Description	Current Adult Worker	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFi	Inhalation cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDI	Inhalation reference dose (mg/kg/d)	CS	
Ca	Concentration of chemical in air as fugitive dusts (mg/m3)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
PEF	Particulate emission factor (m3/kg)	4.63E+09	
RR	Respiration rate (m3/hr)	0.83	
ET	Exposure time (hrs/d)	10	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	*Averaging time, carcinogens (d)*	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	Ca (mg/m3)	CSFI 1/(mg/kg/d)	RfDI (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.78	1.68E-10	NA	NA	4.9E-12	--	--	1.4E-11	--	--
Arsenic	11.6	2.51E-09	15.1	0.0003	7.3E-11	1.1E-09	8.1%	2.0E-10	6.8E-07	0.5%
Cadmium	20.5	4.43E-09	6.3	NA	1.3E-10	8.1E-10	6.0%	3.6E-10	--	--
Chromium	44.3	9.57E-09	42	NA	2.8E-10	1.2E-08	85.9%	7.8E-10	--	--
Manganese	102	2.20E-08	NA	0.0000143	6.4E-10	--	--	1.8E-09	1.3E-04	99.5%
					Total ILCR:	1.4E-08	100.0%	HI:	1.3E-04	100.0%

NOTES:
[1] Adult workers include base employees and brig awardees who currently enter the Area B landfill and pond area to perform routine lawn and maintenance work. Conservatively assumes exposures occur 250 days/year for 25 years.
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-53
ADULT WORKERS - AREA B LANDFILL AND POND- CURRENT SCENARIO [1]
ACCIDENTAL INGESTION OF SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Adult Worker	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cw	Concentration of chemical in water (mg/L)	CS	
IR	Ingestion rate (L/hour)	0.05	
ET	Exposure time (hours/event)	2.6	
EF	Exposure frequency (events/yr)	12	
ED	Exposure duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

				Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.008	0.091	NA	1.7E-07	1.6E-08	1.3%	4.9E-07	--	--
Benzene	0.012	0.029	NA	2.6E-07	7.6E-09	0.6%	7.3E-07	--	--
Chloroform	0.0240	0.0061	0.01	5.2E-07	3.2E-09	0.3%	1.5E-06	1.5E-04	2.7%
Trichloroethene	0.045	0.011	0.006	9.8E-07	1.1E-08	0.9%	2.7E-06	4.6E-04	8.5%
Vinyl chloride	0.022	1.9	NA	4.8E-07	9.1E-07	75.5%	1.3E-06	--	--
Bis(2-ethylhexyl)phthalate	0.009	0.014	0.02	2.0E-07	2.7E-09	0.2%	5.5E-07	2.7E-05	0.5%
Aluminum	0.690	NA	1	1.5E-05	--	--	4.2E-05	4.2E-05	0.8%
Arsenic	0.0067	1.75	0.0003	1.5E-07	2.6E-07	21.2%	4.1E-07	1.4E-03	25.3%
Manganese	0.272	NA	0.005	5.9E-06	--	--	1.7E-05	3.3E-03	61.5%
Zinc	0.202	NA	0.3	4.4E-06	--	--	1.2E-05	4.1E-05	0.8%
				Total ILCR:	1.2E-06	100.0%	HI:	5.4E-03	100.0%

NOTES:
[1] Adult workers include base employees and brig awardees who currently enter the Area B landfill and pond area to perform routine lawn and maintenence work. Conservatively assumes exposures occur 250 days/year for 25 years.
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-54
ADULT WORKERS - AREA B LANDFILL AND POND- CURRENT SCENARIO [1]
DERMAL CONTACT WITH SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [2])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Adult Worker	
DAD	Dermally absorbed dose (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	
A	Skin surface area available for contact (cm2)	4100	
EF	Exposure frequency (d/yr)	12	
ED	Exposure duration (yrs)	25	
EV	Event frequency (events/day)	1	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	
Cw	Concentration of chemical in water (mg/L)	CS	
CF	Conversion factor (L/cm3)	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/event)	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	
tevent	Duration of event (hours/event)	2.6	

							Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.008	5.3E-03	1.7E-02	1.4E-07	0.091	NA	9.6E-08	8.7E-09	1.0%	2.7E-07	--	--
Benzene	0.012	2.1E-01	6.5E-01	7.8E-06	0.029	NA	5.4E-06	1.6E-07	17.3%	1.5E-05	--	--
Chloroform	0.0240	8.9E-03	3.1E-02	7.5E-07	0.0061	0.01	5.2E-07	3.2E-09	0.4%	1.5E-06	1.5E-04	7.4%
Trichloroethene	0.045	1.6E-02	5.9E-02	2.7E-06	0.011	0.006	1.8E-06	2.0E-08	2.2%	5.1E-06	8.5E-04	43.2%
Vinyl chloride	0.022	7.3E-03	2.2E-02	4.8E-07	1.9	NA	3.3E-07	6.3E-07	70.3%	9.3E-07	--	--
Bis(2-ethylhexyl)phthalate	0.009	3.3E-02	6.7E-01	6.1E-06	0.014	0.02	4.2E-06	5.8E-08	6.5%	1.2E-05	5.8E-04	29.6%
Aluminum	0.690	1.0E-03	NA	1.8E-06	NA	1	1.2E-06	--	--	3.5E-06	3.5E-06	0.2%
Arsenic	0.0067	1.0E-03	NA	1.7E-08	1.75	0.0003	1.2E-08	2.1E-08	2.3%	3.4E-08	1.1E-04	5.7%
Manganese	0.272	1.0E-03	NA	7.1E-07	NA	0.005	4.9E-07	--	--	1.4E-06	2.7E-04	13.8%
Zinc	0.202	1.0E-03	NA	5.3E-07	NA	0.3	3.6E-07	--	--	1.0E-06	3.4E-06	0.2%
							Total ILCR:	9.0E-07	100.0%		HI: 2.0E-03	100.0%

NOTES:
[1] Adult workers include base employees and brig awardees who currently enter the Area B landfill and pond area to perform routine lawn and maintenance work. Conservatively assumes exposures occur 250 days/year for 25 years.
[2] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-55
ADULT WORKERS - AREA B LANDFILL AND POND- CURRENT SCENARIO [1]
ACCIDENTAL INGESTION OF SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Adult Worker	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	
IR	Ingestion Rate (mg/d)	50	
CF	Conversion factor (kg/mg)	1E-06	
FI	Fraction of sediment ingested from site	1	
EF	Exposure Frequency (d/yr)	12	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

				Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Vinyl chloride	0.060	1.9	NA	5.0E-10	9.6E-10	0.1%	1.4E-09	--	--
4,4'-DDD	4.20	0.24	NA	3.5E-08	8.5E-09	0.9%	9.9E-08	--	--
4,4'-DDE	0.850	0.34	NA	7.1E-09	2.4E-09	0.2%	2.0E-08	--	--
Aroclor-1254	5.78	7.7	NA	4.9E-08	3.7E-07	38.1%	1.4E-07	--	--
Dieldrin	0.068	16	5E-05	5.7E-10	9.1E-09	0.9%	1.6E-09	3.2E-05	0.4%
Aluminum	22768	NA	1	1.9E-04	--	--	5.3E-04	5.3E-04	6.7%
Antimony	16	NA	0.0004	1.3E-07	--	--	3.8E-07	9.4E-04	11.8%
Arsenic	38.0	1.75	0.0003	3.2E-07	5.6E-07	56.9%	8.9E-07	3.0E-03	37.3%
Beryllium	0.76	4.3	0.005	6.4E-09	2.7E-08	2.8%	1.8E-08	3.6E-06	0.0%
Cadmium	34.5	NA	0.0005	2.9E-07	--	--	8.1E-07	1.6E-03	20.3%
Copper	298	NA	0.0371	2.5E-06	--	--	7.0E-06	1.9E-04	2.4%
Lead	455	NA	NA	3.8E-06	--	--	1.1E-05	--	--
Manganese	240	NA	0.005	2.0E-06	--	--	5.6E-06	1.1E-03	14.1%
Mercury	0.35	NA	0.0003	2.9E-09	--	--	8.2E-09	2.7E-05	0.3%
Silver	13.5	NA	0.005	1.1E-07	--	--	3.2E-07	6.4E-05	0.8%
Vanadium	114	NA	0.007	9.6E-07	--	--	2.7E-06	3.8E-04	4.8%
Zinc	995	NA	0.3	8.3E-06	--	--	2.3E-05	7.8E-05	1.0%
				Total ILCR:	9.8E-07	100.0%	HI:	8.0E-03	100.0%

NOTES:
[1] Adult workers include base employees and brig awardees who currently enter the Area B landfill and pond area to perform routine lawn and maintenence work. Conservatively assumes exposures occur 250 days/year for 25 years.
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-56
ADULT WORKERS - AREA B LANDFILL AND POND- CURRENT SCENARIO [1]
DERMAL CONTACT WITH SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Current Adult Worker	
DAD	Dermally absorbed dose (mg/kg/d)	CS	(Chemical Specific)
ILCR	*Incremental lifetime cancer risk*	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	
CF	Conversion factor (kg/mg)	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	
ABS	Absorption fraction —		
	Cadmium and default organics:	0.01	
	Default inorganics:	0.001	
	PCBs	0.06	
A	Skin surface area available for contact (cm2)	4100	
EF	Exposure Frequency (d/yr)	12	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Vinyl chloride	0.060	0.25	1.9	NA	1.0E-08	2.0E-08	0.6%	2.9E-08	–	–
4,4'-DDD	4.20	0.1	0.24	NA	2.9E-07	6.9E-08	2.0%	8.1E-07	–	–
4,4'-DDE	0.850	0.1	0.34	NA	5.8E-08	2.0E-08	0.6%	1.6E-07	–	–
Aroclor-1254	5.78	0.06	7.7	NA	2.4E-07	1.8E-06	53.8%	6.7E-07	–	–
Dieldrin	0.068	0.1	16	5E-05	4.7E-09	7.4E-08	2.2%	1.3E-08	2.6E-04	2.2%
Aluminum	22768	0.01	NA	1	1.6E-04	–	–	4.4E-04	4.4E-04	3.8%
Antimony	16	0.01	NA	0.0004	1.1E-07	–	–	3.1E-07	7.7E-04	6.6%
Arsenic	38.0	0.03	1.75	0.0003	7.8E-07	1.4E-06	40.2%	2.2E-06	7.3E-03	62.8%
Beryllium	0.76	0.01	4.3	0.005	5.2E-09	2.2E-08	0.7%	1.5E-08	2.9E-06	0.0%
Cadmium	34.5	0.01	NA	0.0005	2.4E-07	–	–	6.6E-07	1.3E-03	11.4%
Copper	298	0.01	NA	0.0371	2.0E-06	–	–	5.7E-06	1.5E-04	1.3%
Lead	455	0.01	NA	NA	3.1E-06	–	–	8.8E-06	–	–
Manganese	240	0.01	NA	0.005	1.6E-06	–	–	4.6E-06	9.2E-04	7.9%
Mercury	0.35	0.01	NA	0.0003	2.4E-09	–	–	6.7E-09	2.2E-05	0.2%
Silver	13.5	0.01	NA	0.005	9.3E-08	–	–	2.6E-07	5.2E-05	0.4%
Vanadium	114	0.01	NA	0.007	7.9E-07	–	–	2.2E-06	3.1E-04	2.7%
Zinc	995	0.01	NA	0.3	6.8E-06	–	–	1.9E-05	6.4E-05	0.5%
					Total ILCR:	3.4E-06	100.0%	HI:	1.2E-02	100.0%

NOTES:
[1] Adult workers include base employees and brig awardees who currently enter the Area B landfill and pond area to perform routine lawn and maintenance work. Conservatively assumes exposures occur 250 days/year for 25 years.
NA - Toxicity criterion not available.
– Not applicable.

TABLE D-57
ADULT WORKERS - AREA B LANDFILL AND POND- CURRENT SCENARIO [1]
INHALATION OF OUTDOOR AMBIENT AIR
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	Current Adult Worker	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFi	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDi	Oral reference dose (mg/kg/d)	CS	
Ca	Concentration of chemical in air (mg/m3)	CS	
RR	Respiration rate (m3/hr)	0.83	
ET	Exposure time (hrs/d)	10	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	9125	

				Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFi 1/(mg/kg/	RfDi (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2,4-Trimethylbenzene	0.0003	NA	NA	8.7E-06	--	--	2.4E-05	--	--
Benzene	0.0007	0.029	0.00171	2.0E-05	5.9E-07	46.8%	5.7E-05	3.3E-02	26.1%
Bromomethane	0.0008	NA	0.00143	2.3E-05	--	--	6.5E-05	4.5E-02	35.7%
Hexachlorobutadiene	0.0003	0.077	0.0005	8.7E-06	6.7E-07	53.2%	2.4E-05	4.9E-02	38.2%
				Total ILCR:	1.3E-06	100.0%	HI:	1.3E-01	100.0%

NOTES:
[1] Adult workers include base employees and brig awardees who currently enter the Area B landfill and pond area to perform routine lawn and maintenence work. Conservatively assumes exposures occur 250 days/year for 25 years.
NA - Toxicity criterion not available.
-- *Not applicable.*

TABLE D-58
SCHOOL EMPLOYEES AND CHILDREN - AREA B ELEMENTARY SCHOOL - CURRENT SCENARIO
ACCIDENTAL INGESTION OF SURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Adult School Employee	Child (6 - 12 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	50	100	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
FI	Fraction of soil ingested from site	1	1	
EF	Exposure Frequency (d/yr)	250	180	
ED	Exposure Duration (yrs)	25	6	
BW	Body weight (kg)	70	31	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	9125	2190	

				Adult School Employee						Child (6 - 12 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Antimony	7.8	NA	0.0004	1.4E-06	--	--	3.8E-06	9.5E-03	6.3%	1.1E-06	--	--	1.2E-05	3.1E-02	6.3%
Arsenic	25.1	1.75	0.0003	4.4E-06	7.7E-06	100.0%	1.2E-05	4.1E-02	27.2%	3.4E-06	6.0E-06	100.0%	4.0E-05	1.3E-01	27.2%
Chromium	869	NA	0.005	1.5E-04	--	--	4.3E-04	8.5E-02	56.5%	1.2E-04	--	--	1.4E-03	2.8E-01	56.5%
Manganese	61.2	NA	0.005	1.1E-05	--	--	3.0E-05	6.0E-03	4.0%	8.3E-06	--	--	9.7E-05	1.9E-02	4.0%
Vanadium	128	NA	0.007	2.2E-05	--	--	6.3E-05	8.9E-03	5.9%	1.7E-05	--	--	2.0E-04	2.9E-02	5.9%
				Total ILCR:	7.7E-06	100.0%	HI:	1.5E-01	100.0%	Total ILCR:	6.0E-06	100.0%	HI:	4.9E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-59
SCHOOL EMPLOYEES AND CHILDREN - AREA B ELEMENTARY SCHOOL - CURRENT SCENARIO
DERMAL CONTACT WITH SURFACES SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Adult School Employee	Child (6 - 12 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	1	
ABS	Absorption fraction --			
	Cadmium and default organics:	0.01	0.01	
	Default inorganics:	0.001	0.001	
	PCBs	0.06	0.06	
A	Skin surface area available for contact (cm2)	4100	3242	
EF	Exposure Frequency (d/yr)	250	180	
ED	Exposure Duration (yrs)	25	6	
BW	Body weight (kg)	70	31	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	9125	2190	

					Adult School Employee						Child (6 - 12 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Antimony	7.8	0.01	NA	0.0004	1.1E-06	--	--	3.1E-06	7.8E-03	4.1%	3.4E-07	--	--	4.0E-06	1.0E-02	4.1%
Arsenic	25.1	0.03	1.75	0.0003	1.1E-05	1.9E-05	100.0%	3.0E-05	1.0E-01	52.9%	3.3E-06	5.8E-06	100.0%	3.9E-05	1.3E-01	52.9%
Chromium	869	0.01	NA	0.005	1.2E-04	--	--	3.5E-04	7.0E-02	36.6%	3.8E-05	--	--	4.5E-04	9.0E-02	36.6%
Manganese	61.2	0.01	NA	0.005	8.8E-06	--	--	2.5E-05	4.9E-03	2.6%	2.7E-06	--	--	3.2E-05	6.3E-03	2.6%
Vanadium	128	0.01	NA	0.007	1.8E-05	--	--	5.1E-05	7.3E-03	3.9%	5.7E-06	--	--	6.6E-05	9.4E-03	3.9%
					Total ILCR:	1.9E-05	100.0%	HI:	1.9E-01	100.0%	Total ILCR:	5.8E-06	100.0%	HI:	2.4E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-60
SCHOOL EMPLOYEES AND CHILDREN - AREA B ELEMENTARY SCHOOL - CURRENT SCENARIO
INHALATION OF FUGITIVE DUSTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)
Where: Ca = Cs * (1/PEF)

ILCR = CDI*CSFI
HQ = CDI/RfDI

Parameter	Description	Adult School Employee	Child (6 - 12 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	*(Chemical Specific)*
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFI	Inhalation cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDI	Inhalation reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of chemical in air as fugitive dusts (mg/m3)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
PEF	Particulate emission factor (m3/kg)	4.63E+09	4.63E+09	
RR	Respiration rate (m3/hr)	0.83	0.83	
ET	Exposure time (hrs/d)	1	2	
EF	Exposure Frequency (d/yr)	250	180	
ED	Exposure Duration (yrs)	25	6	
BW	Body weight (kg)	70	31	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	9125	2190	

					Adult School Employee						Child (6 - 12 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	Ca (mg/m3)	CSFI 1/(mg/kg/d)	RfDI (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Antimony	7.8	1.68E-09	NA	NA	4.9E-12	--	--	1.4E-11	--	--	3.8E-12	--	--	4.4E-11	--	--
Arsenic	25.1	5.42E-09	15.1	0.0003	1.6E-11	2.4E-10	1.0%	4.4E-11	1.5E-07	1.9%	1.2E-11	1.9E-10	1.0%	1.4E-10	4.8E-07	1.9%
Chromium	869	1.88E-07	42	NA	5.4E-10	2.3E-08	99.0%	1.5E-09	--	--	4.2E-10	1.8E-08	99.0%	5.0E-09	--	--
Manganese	61.2	1.32E-08	NA	0.0000143	3.8E-11	--	--	1.1E-10	7.5E-06	98.1%	3.0E-11	--	--	3.5E-10	2.4E-05	98.1%
Vanadium	128	2.76E-08	NA	NA	8.0E-11	--	--	2.2E-10	--	--	6.3E-11	--	--	7.3E-10	--	--
					Total ILCR:	2.3E-08	100.0%		HI: 7.7E-06	100.0%	Total ILCR:	1.8E-08	100.0%		HI: 2.5E-05	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-61
SCHOOL EMPLOYEES AND CHILDREN - AREA B ELEMENTARY SCHOOL - CURRENT SCENARIO
ACCIDENTAL INGESTION OF SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*ET*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Adult School Employee	Child (6 - 12 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/hour)	0.05	0.05	
ET	Exposure time (hours/event)	2.6	2.6	
EF	Exposure Frequency (events/yr)	12	137	
ED	Exposure Duration (yrs)	25	6	
BW	Body weight (kg)	70	31	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	9125	2190	

				Adult School Employee						Child (6 - 12 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
4,4'-DDD	0.00004	0.24	NA	8.3E-10	2.0E-10	0.0%	2.3E-09	--	--	5.1E-09	1.2E-09	0.0%	6.0E-08	--	--
Arsenic	0.012	1.75	0.0003	2.6E-07	4.6E-07	100.0%	7.3E-07	2.4E-03	25.7%	1.6E-06	2.8E-06	100.0%	1.9E-05	2.8E-06	0.1%
Lead	0.054000	NA	NA	1.2E-06	--	--	3.3E-06	--	--	7.3E-06	--	--	8.5E-05	--	--
Manganese	0.574	NA	0.005	1.3E-05	--	--	3.5E-05	7.0E-03	73.8%	7.7E-05	--	--	9.0E-04	4.7E-05	1.6%
Zinc	0.199	NA	0.3	4.3E-06	--	--	1.2E-05	4.1E-05	0.4%	2.7E-05	--	--	3.1E-04	2.8E-03	98.3%
				Total ILCR	4.6E-07	100.0%	HI:	9.5E-03	100.0%	Total ILCR:	2.8E-06	100.0%	HI:	2.9E-03	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-62
SCHOOL EMPLOYEES AND CHILDREN - AREA B ELEMENTARY SCHOOL - CURRENT SCENARIO
DERMAL CONTACT WITH SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Adult School Employee	Child (6 - 12 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	4100	3242	
EF	Exposure frequency (d/yr)	12	137	
ED	Exposure duration (yrs)	25	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	31	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	9125	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/event)	CS	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	2.6	2.6	

							Adult School Employee						Child (6 - 12 Yrs Old)					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
4,4'-DDD	0.000038	2.8E-01	3.5E+00	1.3E-07	0.24	NA	9.1E-08	2.2E-08	36.8%	2.6E-07	–	–	4.5E-07	1.1E-07	36.8%	5.2E-06	–	–
Arsenic	0.012	1.0E-03	NA	3.1E-08	1.75	0.0003	2.1E-08	3.8E-08	63.2%	6.0E-08	2.0E-04	25.7%	1.0E-07	1.8E-07	63.2%	1.2E-06	4.1E-03	25.7%
Lead	0.054	1.0E-03	NA	1.4E-07	NA	NA	9.7E-08	–	–	2.7E-07	–	–	4.7E-07	–	–	5.5E-06	–	–
Manganese	0.574	1.0E-03	NA	1.5E-06	NA	0.005	1.0E-06	–	–	2.9E-06	5.7E-04	73.8%	5.0E-06	–	–	5.9E-05	1.2E-02	73.8%
Zinc	0.199	1.0E-03	NA	5.2E-07	NA	0.3	3.6E-07	–	–	1.0E-06	3.3E-06	0.4%	1.7E-06	–	–	2.0E-05	6.8E-05	0.4%
							Total ILCR:	5.9E-08	100.0%	HI:	7.8E-04	100.0%	Total ILCR:	2.9E-07	100.0%	HI:	1.6E-02	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
– Not applicable.

TABLE D-63
SCHOOL EMPLOYEES AND CHILDREN - AREA B ELEMENTARY SCHOOL - CURRENT SCENARIO
ACCIDENTAL INGESTION OF SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Adult School Employee	Child (6 - 12 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	50	100	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
FI	Fraction of sediment ingested from site	1	1	
EF	Exposure Frequency (d/yr)	12	137	
ED	Exposure Duration (yrs)	25	6	
BW	Body weight (kg)	70	31	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	9125	2190	

				Adult School Employee						Child (6 - 12 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
NO COPCs	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--

NOTES:
-- Not applicable.

TABLE D-64
SCHOOL EMPLOYEES AND CHILDREN - AREA B ELEMENTARY SCHOOL - CURRENT SCENARIO
DERMAL CONTACT WITH SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Adult School Employee	Child (6 - 12 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	1	
ABS	Absorption fraction –			
	Cadmium and default organics:	0.01	0.01	
	Default inorganics:	0.001	0.001	
	PCBs	0.06	0.06	
A	Skin surface area available for contact (cm2)	5300	2006	
EF	Exposure Frequency (d/yr)	12	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

					Adult School Employee						Child (6 - 12 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
NO COPCs	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–

NOTES:
– Not applicable.

TABLE D-65
SCHOOL EMPLOYEES AND CHILDREN - AREA B ELEMENTARY SCHOOL - CURRENT SCENARIO
INHALATION OF INDOOR AIR
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	Adult School Employee	Child (6 - 12 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFi	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDi	Oral reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of chemical in air (mg/m3)	CS	CS	
RR	Respiration rate (m3/hr)	0.83	0.83	
ET	Exposure time (hrs/d)	9	7	
EF	Exposure Frequency (d/yr)	250	180	
ED	Exposure Duration (yrs)	25	6	
BW	Body weight (kg)	70	31	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	9125	2190	

				Adult School Employee						Child (6 - 12 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFi 1/(mg/kg/d)	RfDi (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2,4-Trimethylbenzene	0.0003	NA	NA	7.8E-06	--	--	2.2E-05	--	--	2.4E-06	--	--	2.8E-05	--	--
1,4-Dichlorobenzene	0.0006	NA	0.229	1.6E-05	--	--	4.4E-05	1.9E-04	0.3%	4.8E-06	--	--	5.5E-05	2.4E-04	0.3%
Benzene	0.0007	0.029	0.00171	1.8E-05	5.3E-07	46.8%	5.1E-05	3.0E-02	40.5%	5.5E-06	1.6E-07	46.8%	6.5E-05	3.8E-02	40.5%
Hexachlorobutadiene	0.0003	0.077	0.0005	7.8E-06	6.0E-07	53.2%	2.2E-05	4.4E-02	59.3%	2.4E-06	1.8E-07	53.2%	2.8E-05	5.5E-02	59.3%
				Total ILCR:	1.1E-06	100.0%		HI: 7.4E-02	100.0%	Total ILCR:	3.4E-07	100.0%		HI: 9.4E-02	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

1,2,4-Trimethylbenzene	0.0003
Benzene	0.0007
Hexachlorobutadiene	0.0003

TABLE D-66
SCHOOL EMPLOYEES AND CHILDREN - AREA B ELEMENTARY SCHOOL - CURRENT SCENARIO
INHALATION OF OUTDOOR AMBIENT AIR
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	Adult School Employee	Child (6 - 12 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFi	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDi	Oral reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of chemical in air (mg/m3)	CS	CS	
RR	Respiration rate (m3/hr)	0.83	0.83	
ET	Exposure time (hrs/d)	1	1	
EF	Exposure Frequency (d/yr)	250	180	
ED	Exposure Duration (yrs)	25	6	
BW	Body weight (kg)	70	31	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	9125	2190	

				Adult School Employee						Child (6 - 12 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFi 1/(mg/kg/d)	RfDi (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2,4-Trimethylbenzene	0.0003	NA	NA	8.7E-07	--	--	2.4E-06	--	--	3.4E-07	--	--	4.0E-06	--	--
Benzene	0.0007	0.029	0.00171	2.0E-06	5.9E-08	46.8%	5.7E-06	3.3E-03	26.1%	7.9E-07	2.3E-08	46.8%	9.2E-06	5.4E-03	26.1%
Bromomethane	0.0008	NA	0.00143	2.3E-06	--	--	6.5E-06	4.5E-03	35.7%	9.1E-07	--	--	1.1E-05	7.4E-03	35.7%
Hexachlorobutadiene	0.0003	0.077	0.0005	8.7E-07	6.7E-08	53.2%	2.4E-06	4.9E-03	38.2%	3.4E-07	2.6E-08	53.2%	4.0E-06	7.9E-03	38.2%
				Total ILCR:	1.3E-07	100.0%	HI:	1.3E-02	100.0%	Total ILCR:	4.9E-08	100.0%	HI:	2.1E-02	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

BSC

TABLE D-67
ON-SITE RESIDENTS - AREA B - FUTURE SCENARIO
RESIDENTIAL INGESTION OF SHALLOW GROUNDWATER FROM LOCATION B-MW11A
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/d)	2	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,1-Dichloroethene	ND	0.6	0.009	--	--	--	--	--	--	--	--	--	--	--	--
1,2-Dichloroethane	0.058	0.091	NA	6.8E-04	6.2E-05	0.3%	1.6E-03	--	--	3.2E-04	2.9E-05	0.3%	2.5E-03	--	--
1,2-Dichloroethene	1.6	NA	0.009	1.9E-02	--	--	4.4E-02	4.9E+00	27.8%	8.8E-03	--	--	6.8E-02	7.6E+00	27.8%
Benzene	0.029	0.029	NA	3.4E-04	9.9E-06	0.0%	7.9E-04	--	--	1.6E-04	4.6E-06	0.0%	1.2E-03	--	--
Chlorobenzene	ND	NA	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene	ND	0.052	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Trichloroethene	0.044	0.011	0.008	5.2E-04	5.7E-06	0.0%	1.2E-03	2.0E-01	1.1%	2.4E-04	2.7E-06	0.0%	1.9E-03	3.1E-01	1.1%
Vinyl chloride	0.84	1.9	NA	1.1E-02	2.1E-02	95.7%	2.6E-02	--	--	5.2E-03	9.8E-03	95.7%	4.0E-02	--	--
1,4-Dichlorobenzene	0.003	0.024	NA	3.5E-05	8.5E-07	0.0%	8.2E-05	--	--	1.6E-05	3.9E-07	0.0%	1.3E-04	--	--
Bis(2-chloroethyl)ether	ND	1.1	NA	--	--	--	--	--	--	--	--	--	--	--	--
Bis(2-ethylhexyl)phthalate	ND	0.014	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Dieldrin	ND	16	0.00005	--	--	--	--	--	--	--	--	--	--	--	--
gamma-BHC	ND	1.3	0.0003	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Aluminum	88.8	NA	1	1.0E+00	--	--	2.4E+00	2.4E+00	13.9%	4.9E-01	--	--	3.8E+00	3.8E+00	13.9%
Antimony	ND	NA	0.0004	--	--	--	--	--	--	--	--	--	--	--	--
Arsenic	0.032	1.75	0.0003	3.8E-04	6.8E-04	3.0%	8.8E-04	2.9E+00	16.7%	1.8E-04	3.1E-04	3.0%	1.4E-03	4.5E+00	16.7%
Barium	0.210	NA	0.07	2.5E-03	--	--	5.8E-03	8.2E-02	0.5%	1.2E-03	--	--	8.9E-03	1.3E-01	0.5%
Beryllium	0.0041	4.3	0.005	4.8E-05	2.1E-04	0.9%	1.1E-04	2.2E-02	0.1%	2.2E-05	9.7E-05	0.9%	1.7E-04	3.5E-02	0.1%
Cadmium	ND	NA	0.0005	--	--	--	--	--	--	--	--	--	--	--	--
Chromium	0.141	NA	0.005	1.7E-03	--	--	3.9E-03	7.7E-01	4.4%	7.7E-04	--	--	6.0E-03	1.2E+00	4.4%
Copper	0.0592	NA	0.0371	7.0E-04	--	--	1.6E-03	4.4E-02	0.2%	3.2E-04	--	--	2.5E-03	6.8E-02	0.2%
Lead	0.0448	NA	NA	5.3E-04	--	--	1.2E-03	--	--	2.5E-04	--	--	1.9E-03	--	--
Manganese	0.906	NA	0.005	1.1E-02	--	--	2.5E-02	5.0E+00	28.3%	5.0E-03	--	--	3.9E-02	7.7E+00	28.3%
Mercury	ND	NA	0.0003	--	--	--	--	--	--	--	--	--	--	--	--
Nickel	0.0276	NA	0.02	3.2E-04	--	--	7.6E-04	3.8E-02	0.2%	1.5E-04	--	--	1.2E-03	5.9E-02	0.2%
Vanadium	0.297	NA	0.007	3.5E-03	--	--	8.1E-03	1.2E+00	6.6%	1.6E-03	--	--	1.3E-02	1.8E+00	6.6%
Zinc	0.231	NA	0.3	2.7E-03	--	--	6.3E-03	2.1E-02	0.1%	1.3E-03	--	--	9.8E-03	3.3E-02	0.1%
				Total ILCR:	2.2E-02	100.0%	HI:	1.8E+01	100.0%	Total ILCR:	1.0E-02	100.0%	HI:	2.7E+01	100.0%

NOTES
NA - Toxicity criterion not available.
ND - Not detected
-- Not applicable.

TABLE D-68
ON-SITE RESIDENTS - AREA B - FUTURE SCENARIO
RESIDENTIAL DERMAL CONTACT WITH SHALLOW GROUNDWATER FROM LOCATION B-MW11A
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	20000	6023	
EF	Exposure frequency (d/yr)	350	350	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/eve	CS	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	0.2	0.2	

							Adult Residents						Child Residents (0 - 6 Yrs Old)					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
1,1-Dichloroethane	ND	1.6E-02	1.2E-02	NA	0.6	0.009	--	--	--	--	--	--	--	--	--	--	--	--
1,2-Dichloroethane	0.058	5.0E-03	3.9E-03	2.3E-07	0.091	NA	2.6E-05	2.4E-06	0.3%	6.2E-05	--	--	9.9E-06	9.0E-07	0.3%	1.2E-04	--	--
1,2-Dichloroethene	1.6	1.0E-02	7.2E-03	1.2E-05	NA	0.009	1.4E-03	--	--	3.2E-03	3.5E-01	86.6%	5.1E-04	--	--	5.9E-03	6.6E-01	86.6%
Benzene	0.029	2.1E-01	1.3E-01	3.8E-06	0.029	NA	4.5E-04	1.3E-05	1.5%	1.1E-03	--	--	1.7E-04	4.9E-06	1.5%	2.0E-03	--	--
Chlorobenzene	ND	4.1E-02	3.3E-02	NA	NA	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene	ND	4.8E-02	5.6E-02	NA	0.052	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Trichloroethene	0.044	1.6E-02	1.5E-02	6.5E-07	0.011	0.006	7.6E-05	8.3E-07	0.1%	1.8E-04	2.9E-02	7.3%	2.8E-05	3.1E-07	0.1%	3.3E-04	5.5E-02	7.3%
Vinyl chloride	0.94	7.3E-03	4.1E-03	3.9E-06	1.9	NA	4.6E-04	8.7E-04	97.9%	1.1E-03	--	--	1.7E-04	3.3E-04	97.9%	2.0E-03	--	--
1,4-Dichlorobenzene	0.003	6.2E-02	6.4E-02	1.9E-07	0.024	NA	2.2E-05	5.4E-07	0.1%	5.2E-05	--	--	8.4E-06	2.0E-07	0.1%	9.8E-05	--	--
Bis(2-chloroethyl)ether	ND	2.1E-03	2.1E-03	NA	1.1	NA	--	--	--	--	--	--	--	--	--	--	--	--
Bis(2-ethylhexyl)phthalate	ND	3.3E-02	1.9E-01	NA	0.014	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Dieldrin	ND	1.9E-02	8.4E-02	NA	16	0.00005	--	--	--	--	--	--	--	--	--	--	--	--
gamma-BHC	ND	1.4E-02	3.9E-02	NA	1.3	0.0003	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	6.7E-04	3.8E-03	NA	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Aluminum	88.8	1.0E-03	NA	1.8E-05	NA	1	2.1E-03	--	--	4.9E-03	4.9E-03	1.2%	7.8E-04	--	--	9.1E-03	9.1E-03	1.2%
Antimony	ND	1.0E-03	NA	NA	NA	0.0004	--	--	--	--	--	--	--	--	--	--	--	--
Arsenic	0.032	1.0E-03	NA	6.4E-09	1.75	0.0003	7.5E-07	1.3E-06	0.1%	1.8E-06	5.8E-03	1.4%	2.8E-07	4.9E-07	0.1%	3.3E-06	1.1E-02	1.4%
Barium	0.210	1.0E-03	NA	4.2E-08	NA	0.07	4.9E-06	--	--	1.2E-05	1.6E-04	0.0%	1.8E-06	--	--	2.2E-05	3.1E-04	0.0%
Beryllium	0.0041	1.0E-03	NA	8.2E-10	4.3	0.005	9.6E-08	4.1E-07	0.0%	2.2E-07	4.5E-05	0.0%	3.6E-08	1.6E-07	0.0%	4.2E-07	8.4E-05	0.0%
Cadmium	ND	1.0E-03	NA	NA	NA	0.0005	--	--	--	--	--	--	--	--	--	--	--	--
Chromium	0.141	1.0E-03	NA	2.8E-08	NA	0.005	3.3E-06	--	--	7.7E-06	1.5E-03	0.4%	1.2E-06	--	--	1.4E-05	2.9E-03	0.4%
Copper	0.0592	1.0E-03	NA	1.2E-08	NA	0.0371	1.4E-06	--	--	3.2E-06	8.7E-05	0.0%	5.2E-07	--	--	6.1E-06	1.6E-04	0.0%
Lead	0.0448	1.0E-03	NA	9.0E-09	NA	NA	1.1E-06	--	--	2.5E-06	--	--	3.9E-07	--	--	4.6E-06	--	--
Manganese	0.906	1.0E-03	NA	1.8E-07	NA	0.005	2.1E-05	--	--	5.0E-05	9.9E-03	2.4%	8.0E-06	--	--	9.3E-05	1.9E-02	2.4%
Mercury	ND	1.0E-03	NA	NA	NA	0.0003	--	--	--	--	--	--	--	--	--	--	--	--
Nickel	0.0276	1.0E-03	NA	5.5E-09	NA	0.02	6.5E-07	--	--	1.5E-06	7.6E-05	0.0%	2.4E-07	--	--	2.8E-06	1.4E-04	0.0%
Vanadium	0.297	1.0E-03	NA	5.9E-08	NA	0.007	7.0E-06	--	--	1.6E-05	2.3E-03	0.6%	2.6E-06	--	--	3.0E-05	4.4E-03	0.6%
Zinc	0.231	1.0E-03	NA	4.6E-08	NA	0.3	5.4E-06	--	--	1.3E-05	4.2E-05	0.0%	2.0E-06	--	--	2.4E-05	7.9E-05	0.0%
							Total ILCR:	8.5E-04	100.0%	HI:	4.1E-01	100.0%	Total ILCR:	3.3E-04	100.0%	HI:	7.6E-01	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-69
ON-SITE RESIDENTS - AREA B- FUTURE SCENARIO
INHALATION OF VOLATILIZED ORGANICS IN SHOWER AIR
SHALLOW GROUNDWATER FROM LOCATION B-MW11A
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (CA*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFi	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDi	Oral reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of volatilized chemical in shower air, Andelman Model (mg/m3)	CS	CS	
RR	Respiration rate (m3/hr)	0.83	0.6	
ET	Exposure time (hrs/d)	0.2	0.2	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFi 1/(mg/kg/d)	RfDi (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Benzene	0.414	0.029	0.00171	4.0E-04	1.2E-05	0.2%	9.4E-04	5.5E-01	82.7%	2.7E-04	7.9E-06	0.2%	3.2E-03	1.9E+00	82.7%
1,2-Dichloroethane	0.145	0.091	0.00286	1.4E-04	1.3E-05	0.2%	3.3E-04	1.2E-01	17.3%	9.5E-05	8.7E-06	0.2%	1.1E-03	3.9E-01	17.3%
1,2-Dichloroethene	26.8	NA	NA	2.6E-02	--	--	6.1E-02	--	--	1.8E-02	--	--	2.1E-01	--	--
Trichloroethene	1.02	0.006	NA	9.9E-04	6.0E-06	0.1%	2.3E-03	--	--	6.7E-04	4.0E-06	0.1%	7.8E-03	--	--
Vinyl chloride	19.7	0.3	NA	1.9E-02	5.8E-03	99.5%	4.5E-02	--	--	1.3E-02	3.9E-03	99.5%	1.5E-01	--	--
				Total ILCR:	5.8E-03	100.0%	HI:	6.7E-01	100.0%	Total ILCR:	3.9E-03	100.0%	HI:	2.2E+00	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable

TABLE D-70
ON-SITE RESIDENTS - AREA B - FUTURE SCENARIO
RESIDENTIAL INGESTION OF SHALLOW GROUNDWATER FROM LOCATION B-MW11A
ORGANIC AND FILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/d)	2	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,1-Dichloroethene	ND	0.6	0.009	--	--	--	--	--	--	--	--	--	--	--	--
1,2-Dichloroethane	0.058	0.091	NA	8.8E-04	8.2E-05	0.3%	1.6E-03	--	--	3.2E-04	2.9E-05	0.3%	2.5E-03	--	--
t,2-Dichloroethene	1.8	NA	0.009	1.9E-02	--	--	4.4E-02	4.9E+00	54.1%	8.8E-03	--	--	6.8E-02	7.6E+00	54.1%
Benzene	0.029	0.029	NA	3.4E-04	9.9E-06	0.0%	7.9E-04	--	--	1.6E-04	4.6E-06	0.0%	1.2E-03	--	--
Chlorobenzene	ND	NA	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene	ND	0.052	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Trichloroethene	0.044	0.011	0.006	5.2E-04	5.7E-06	0.0%	1.2E-03	2.0E-01	2.2%	2.4E-04	2.7E-06	0.0%	1.9E-03	3.1E-01	2.2%
Vinyl chloride	0.94	1.9	NA	1.1E-02	2.1E-02	99.6%	2.8E-02	--	--	5.2E-03	9.8E-03	99.6%	4.0E-02	--	--
1,4-Dichlorobenzene	0.003	0.024	NA	3.5E-05	8.5E-07	0.0%	8.2E-05	--	--	1.6E-05	3.9E-07	0.0%	1.3E-04	--	--
Bis(2-chloroethyl)ether	ND	1.1	NA	--	--	--	--	--	--	--	--	--	--	--	--
Bis(2-ethylhexyl)phthalate	ND	0.014	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Dieldrin	ND	16	0.00005	--	--	--	--	--	--	--	--	--	--	--	--
gamma-BHC	ND	1.3	0.0003	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Antimony	ND	NA	0.0004	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Arsenic	ND	1.75	0.0003	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Chromium	ND	NA	0.005	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Manganese	0.715	NA	0.005	8.4E-03	--	--	2.0E-02	3.9E+00	43.5%	3.9E-03	--	--	3.0E-02	6.1E+00	43.5%
Dissolved Vanadium	0.004	NA	0.007	4.7E-05	--	--	1.1E-04	1.6E-02	0.2%	2.2E-05	--	--	1.7E-04	2.4E-02	0.2%
					Total ILCR:	2.1E-02	100.0%	HI:	9.0E+00	100.0%	Total ILCR:	9.8E-03	100.0%	HI:	1.4E+01	100.0%

NOTES:
NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-71
ON-SITE RESIDENTS - AREA B - FUTURE SCENARIO
RESIDENTIAL DERMAL CONTACT WITH SHALLOW GROUNDWATER FROM LOCATION B-MW11A
ORGANIC AND FILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For Inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	20000	8023	
EF	Exposure frequency (d/yr)	350	350	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/event)	CS	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	0.2	0.2	

							Adult Residents						Child Residents (0 - 6 Yrs Old)					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
1,1-Dichloroethene	ND	1.6E-02	1.2E-02	NA	0.6	0.009	--	--	--	--	--	--	--	--	--	--	--	--
1,2-Dichloroethene	0.058	5.0E-03	3.9E-03	2.3E-07	0.091	NA	2.6E-05	2.4E-06	0.3%	6.2E-05	--	--	9.9E-06	9.0E-07	0.3%	1.2E-04	--	--
1,2-Dichloroethene	1.6	1.0E-02	7.2E-03	1.2E-05	NA	0.009	1.4E-03	--	--	3.2E-03	3.5E-01	90.4%	5.1E-04	--	--	5.9E-03	6.6E-01	90.4%
Benzene	0.029	2.1E-01	1.3E-01	3.8E-06	0.029	NA	4.5E-04	1.3E-05	1.5%	1.1E-03	--	--	1.7E-04	4.9E-06	1.5%	2.0E-03	--	--
Chlorobenzene	ND	4.1E-02	3.3E-02	NA	NA	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Tetrachloroethene	ND	4.8E-02	5.8E-02	NA	0.052	0.01	--	--	--	--	--	--	--	--	--	--	--	--
Trichloroethene	0.044	1.6E-02	1.5E-02	6.5E-07	0.011	0.006	7.6E-05	8.3E-07	0.1%	1.8E-04	2.9E-02	7.6%	2.8E-05	3.1E-07	0.1%	3.3E-04	5.5E-02	7.6%
Vinyl chloride	0.94	7.3E-03	4.1E-03	3.9E-06	1.9	NA	4.6E-04	8.7E-04	98.1%	1.1E-03	--	--	1.7E-04	3.3E-04	98.1%	2.0E-03	--	--
1,4-Dichlorobenzene	0.003	6.2E-02	6.4E-02	1.9E-07	0.024	NA	2.2E-05	5.4E-07	0.1%	5.2E-05	--	--	8.4E-06	2.0E-07	0.1%	9.8E-05	--	--
Bis(2-chloroethyl)ether	ND	2.1E-03	2.1E-03	NA	1.1	NA	--	--	--	--	--	--	--	--	--	--	--	--
Bis(2-ethylhexyl)phthalate	ND	3.3E-02	1.9E-01	NA	0.014	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Dieldrin	ND	1.8E-02	8.4E-02	NA	16	0.00005	--	--	--	--	--	--	--	--	--	--	--	--
gamma-BHC	ND	1.4E-02	3.9E-02	NA	1.3	0.0003	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	8.7E-04	3.8E-03	NA	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Antimony	ND	1.0E-03	NA	NA	NA	0.0004	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Arsenic	ND	1.0E-03	NA	NA	1.75	0.0003	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Chromium	ND	1.0E-03	NA	NA	NA	0.005	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Manganese	0.715	1.0E-03	NA	1.4E-07	NA	0.005	1.7E-05	--	--	3.9E-05	7.8E-03	2.0%	6.3E-06	--	--	7.3E-05	1.5E-02	2.0%
Dissolved Vanadium	0.004	1.0E-03	NA	8.0E-10	NA	0.007	9.4E-08	--	--	2.2E-07	3.1E-05	0.0%	3.5E-08	--	--	4.1E-07	5.9E-05	0.0%
							Total ILCR:	8.9E-04	100.0%	HI:	3.9E-01	100.0%	Total ILCR:	3.3E-04	100.0%	HI:	7.3E-01	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-72
ON-SITE RESIDENTS - AREA B - FUTURE SCENARIO
RESIDENTIAL INGESTION OF DEEP (YORKTOWN) GROUNDWATER FROM LOCATION B-MW198
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/d)	2	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.003	0.091	NA	3.5E-05	3.2E-06	4.9%	8.2E-05	--	--	1.6E-05	1.5E-06	4.9%	1.3E-04	--	--
1,2-Dichloroethene	0.016	NA	0.009	1.9E-04	--	--	4.4E-04	4.9E-02	2.0%	8.8E-05	--	--	6.8E-04	7.6E-02	2.0%
Benzene	0.012	0.029	NA	1.4E-04	4.1E-06	6.3%	3.3E-04	--	--	6.6E-05	1.9E-06	6.3%	5.1E-04	--	--
Chloroform	0.001	0.0061	0.01	1.2E-05	7.2E-08	0.1%	2.7E-05	2.7E-03	0.1%	5.5E-06	3.3E-08	0.1%	4.3E-05	4.3E-03	0.1%
Trichloroethene	0.018	0.011	0.006	2.1E-04	2.3E-06	3.6%	4.9E-04	8.2E-02	3.3%	9.9E-05	1.1E-06	3.6%	7.7E-04	1.3E-01	3.3%
Vinyl chloride	ND	1.9	NA	--	--	--	--	--	--	--	--	--	--	--	--
Dieldrin	ND	16	0.00005	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Aluminum	ND	NA	1	--	--	--	--	--	--	--	--	--	--	--	--
Arsenic	0.0027	1.75	0.0003	3.2E-05	5.5E-05	85.1%	7.4E-05	2.5E-01	10.0%	1.5E-05	2.6E-05	85.1%	1.2E-04	3.8E-01	10.0%
Antimony	ND	NA	0.0004	--	--	--	--	--	--	--	--	--	--	--	--
Barium	0.0986	NA	0.07	1.2E-03	--	--	2.7E-03	3.9E-02	1.6%	5.4E-04	--	--	4.2E-03	6.0E-02	1.6%
Beryllium	ND	4.3	0.005	--	--	--	--	--	--	--	--	--	--	--	--
Cadmium	ND	NA	0.0005	--	--	--	--	--	--	--	--	--	--	--	--
Chromium	ND	NA	0.005	--	--	--	--	--	--	--	--	--	--	--	--
Copper	ND	NA	0.0371	--	--	--	--	--	--	--	--	--	--	--	--
Lead	ND	NA	NA	--	--	--	--	--	--	--	--	--	--	--	--
Manganese	0.373	NA	0.005	4.4E-03	--	--	1.0E-02	2.0E+00	83.0%	2.0E-03	--	--	1.6E-02	3.2E+00	83.0%
Nickel	ND	NA	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Vanadium	ND	NA	0.007	--	--	--	--	--	--	--	--	--	--	--	--
				Total ILCR:	6.5E-05	100.0%	HI:	2.5E+00	100.0%	Total ILCR:	3.0E-05	100.0%	HI:	3.8E+00	100.0%

NOTES:
NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-73
ON-SITE RESIDENTS - AREA B - FUTURE SCENARIO
RESIDENTIAL DERMAL CONTACT WITH DEEP (YORKTOWN) GROUNDWATER FROM LOCATION B-MW19B
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	20000	8023	
EF	Exposure frequency (d/yr)	350	350	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/event)	CS	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	0.2	0.2	

							Adult Residents						Child Residents (0 - 6 Yrs Old)					
							Cercinogens			Noncarcinogens			Cercinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.003	5.3E-03	3.9E-03	1.2E-08	0.091	NA	1.4E-06	1.2E-07	2.1%	3.2E-06	--	--	5.1E-07	4.7E-08	2.1%	6.0E-06	--	--
1,2-Dichloroethene	0.016	1.0E-02	7.2E-03	1.2E-07	NA	0.009	1.4E-05	--	--	3.2E-05	3.5E-03	17.2%	5.1E-06	--	--	5.9E-05	6.6E-03	17.2%
Benzene	0.012	2.1E-01	1.3E-01	1.6E-06	0.029	NA	1.9E-04	5.4E-06	90.3%	4.4E-04	--	--	7.0E-05	2.0E-06	90.3%	8.1E-04	--	--
Chloroform	0.001	8.9E-03	7.5E-03	7.5E-09	0.0061	0.01	8.9E-07	5.4E-09	0.1%	2.1E-06	2.1E-04	1.0%	3.3E-07	2.0E-09	0.1%	3.9E-06	3.9E-04	1.0%
Trichloroethene	0.018	1.6E-02	1.5E-02	2.6E-07	0.011	0.006	3.1E-05	3.4E-07	5.7%	7.2E-05	1.2E-02	59.0%	1.2E-05	1.3E-07	5.7%	1.4E-04	2.3E-02	59.0%
Vinyl chloride	ND	7.3E-03	4.1E-03	NA	1.9	NA	--	--	--	--	--	--	--	--	--	--	--	--
Dieldrin	ND	1.6E-02	8.4E-02	NA	16	0.00005	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	0.7E-04	3.8E-03	NA	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Aluminum	ND	1.0E-03	NA	NA	NA	1	--	--	--	--	--	--	--	--	--	--	--	--
Arsenic	0.0027	1.0E-03	NA	5.4E-10	1.75	0.0003	6.3E-08	1.1E-07	1.9%	1.5E-07	4.9E-04	2.4%	2.4E-08	4.2E-08	1.9%	2.8E-07	9.2E-04	2.4%
Antimony	ND	1.0E-03	NA	NA	NA	0.0004	--	--	--	--	--	--	--	--	--	--	--	--
Barium	0.0986	1.0E-03	NA	2.0E-08	NA	0.07	2.3E-06	--	--	5.4E-06	7.7E-05	0.4%	8.7E-07	--	--	1.0E-05	1.4E-04	0.4%
Beryllium	ND	1.0E-03	NA	NA	4.3	0.005	--	--	--	--	--	--	--	--	--	--	--	--
Cadmium	ND	1.0E-03	NA	NA	NA	0.0005	--	--	--	--	--	--	--	--	--	--	--	--
Chromium	ND	1.0E-03	NA	NA	NA	0.005	--	--	--	--	--	--	--	--	--	--	--	--
Copper	ND	1.0E-03	NA	NA	NA	0.0371	--	--	--	--	--	--	--	--	--	--	--	--
Lead	ND	1.0E-03	NA	NA	NA	NA	--	--	--	--	--	--	--	--	--	--	--	--
Manganese	0.373	1.0E-03	NA	7.5E-08	NA	0.005	8.8E-06	--	--	2.0E-05	4.1E-03	20.0%	3.3E-06	--	--	3.8E-05	7.7E-03	20.0%
Nickel	ND	1.0E-03	NA	NA	NA	0.02	--	--	--	--	--	--	--	--	--	--	--	--
Vanadium	ND	1.0E-03	NA	NA	NA	0.007	--	--	--	--	--	--	--	--	--	--	--	--
							Total ILCR:	6.0E-06	100.0%		HI:	2.0E-02	100.0%	Total ILCR:	2.2E-06	100.0%	HI:	3.8E-02	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-74
ON-SITE RESIDENTS - AREA B- FUTURE SCENARIO
INHALATION OF VOLATILIZED ORGANICS IN SHOWER AIR
DEEP (YORKTOWN) GROUNDWATER FROM LOCATION B-MW193
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (CA*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFi	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDi	Oral reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of volatilized chemical in shower air, Andelman Model (mg/m3)	CS	CS	
RR	Respiration rate (m3/hr)	0.83	0.6	
ET	Exposure time (hrs/d)	0.2	0.2	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFi 1/(mg/kg/d)	RfDi (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Benzene	0.171	0.029	0.00171	1.7E-04	4.8E-06	57.1%	3.9E-04	2.3E-01	97.6%	1.1E-04	3.3E-06	57.1%	1.3E-03	7.7E-01	97.6%
Chloroform	0.007	0.0805	NA	6.8E-05	5.5E-07	6.5%	1.6E-05	--	--	4.6E-06	3.7E-07	6.5%	5.4E-05	--	--
1,2-Dichloroethane	0.007	0.091	0.00286	6.8E-05	6.2E-07	7.3%	1.6E-05	5.6E-03	2.4%	4.6E-06	4.2E-07	7.3%	5.4E-05	1.9E-02	2.4%
1,2-Dichloroethene	0.3	NA	NA	2.9E-04	--	--	6.8E-04	--	--	2.0E-04	--	--	2.3E-03	--	--
Trichloroethene	0.42	0.006	NA	4.1E-04	2.5E-06	29.0%	9.6E-04	--	--	2.8E-04	1.7E-06	29.0%	3.2E-03	--	--
				Total ILCR:	8.5E-06	100.0%		HI:	2.3E-01	Total ILCR:	5.7E-06	100.0%		HI:	7.9E-01	100.0%

NOTES:
NA - Toxicity criterion not available
ND - Not detected
-- Not applicable

TABLE D-75
ON-SITE RESIDENTS - AREA B - FUTURE SCENARIO
RESIDENTIAL INGESTION OF DEEP (YORKTOWN) GROUNDWATER FROM LOCATION B-MW19B
ORGANIC AND FILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/d)	2	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	3285	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.003	0.091	NA	3.5E-05	3.2E-06	8.8%	8.2E-05	--	--	1.6E-05	1.5E-06	8.8%	1.3E-04	--	--
1,2-Dichloroethene	0.016	NA	0.009	1.9E-04	--	--	4.4E-04	4.9E-02	2.2%	8.8E-05	--	--	6.8E-04	7.6E-02	2.2%
Benzene	0.012	0.029	NA	1.4E-04	4.1E-06	11.2%	3.3E-04	--	--	6.6E-05	1.9E-06	11.2%	5.1E-04	--	--
Chloroform	0.001	0.0061	0.01	1.2E-05	7.2E-08	0.2%	2.7E-05	2.7E-03	0.1%	5.5E-06	3.3E-08	0.2%	4.3E-05	4.3E-03	0.1%
Trichloroethene	0.018	0.011	0.006	2.1E-04	2.3E-06	6.4%	4.9E-04	8.2E-02	3.7%	9.9E-05	1.1E-06	6.4%	7.7E-04	1.3E-01	3.7%
Vinyl chloride	ND	1.9	NA	--	--	--	--	--	--	--	--	--	--	--	--
Dieldrin	ND	16	0.00005	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Arsenic	0.0013	1.75	0.0003	1.5E-05	2.7E-05	73.4%	3.6E-05	1.2E-01	5.4%	7.1E-06	1.2E-05	73.4%	5.5E-05	1.8E-01	5.4%
Dissolved Manganese	0.356	NA	0.005	4.2E-03	--	--	9.8E-03	2.0E+00	88.5%	2.0E-03	--	--	1.5E-02	3.0E+00	88.5%
				Total ILCR:	3.6E-05	100.0%	HI:	2.2E+00	100.0%	Total ILCR:	1.7E-05	100.0%	HI:	3.4E+00	100.0%

NOTES:
NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-76
ON-SITE RESIDENTS - AREA B - FUTURE SCENARIO
RESIDENTIAL DERMAL CONTACT WITH DEEP (YORKTOWN) GROUNDWATER FROM LOCATION B-MW193
ORGANIC AND FILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	20000	8023	
EF	Exposure frequency (d/yr)	350	350	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/event)	CS	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	0.2	0.2	

							Adult Residents						Child Residents (0 - 6 Yrs Old)					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.003	5.3E-03	3.9E-03	1.2E-08	0.091	NA	1.4E-06	1.2E-07	2.1%	3.2E-06	--	--	5.1E-07	4.7E-08	2.1%	8.0E-06	--	--
1,2-Dichloroethane	0.016	1.0E-02	7.2E-03	1.2E-07	NA	0.009	1.4E-05	--	--	3.2E-05	3.5E-03	17.6%	5.1E-06	--	--	5.0E-05	6.6E-03	17.6%
Benzene	0.012	2.1E-01	1.3E-01	1.6E-06	0.029	NA	1.9E-04	5.4E-06	91.2%	4.4E-04	--	--	7.0E-05	2.0E-06	91.2%	8.1E-04	--	--
Chloroform	0.001	8.9E-03	7.5E-03	7.5E-09	0.0061	0.01	8.9E-07	5.4E-09	0.1%	2.1E-06	2.1E-04	1.0%	3.3E-07	2.0E-09	0.1%	3.9E-06	3.9E-04	1.0%
Trichloroethane	0.018	1.6E-02	1.5E-02	2.6E-07	0.011	0.006	3.1E-05	3.4E-07	5.7%	7.2E-05	1.2E-02	60.5%	1.2E-05	1.3E-07	5.7%	1.4E-04	2.3E-02	60.5%
Vinyl chloride	ND	7.3E-03	4.1E-03	NA	1.9	NA	--	--	--	--	--	--	--	--	--	--	--	--
Dieldrin	ND	1.6E-02	8.4E-02	NA	16	0.00005	--	--	--	--	--	--	--	--	--	--	--	--
Heptachlor epoxide	ND	6.7E-04	3.8E-03	NA	9.1	0.000013	--	--	--	--	--	--	--	--	--	--	--	--
Dissolved Arsenic	0.0013	1.0E-03	NA	2.6E-10	1.75	0.0003	3.1E-08	5.3E-08	0.9%	7.1E-08	2.4E-04	1.2%	1.1E-08	2.0E-08	0.9%	1.3E-07	4.4E-04	1.2%
Dissolved Manganese	0.356	1.0E-03	NA	7.1E-08	NA	0.005	8.4E-06	--	--	2.0E-05	3.9E-03	19.6%	3.1E-06	--	--	3.7E-05	7.3E-03	19.6%
							Total ILCR	5.9E-06	100.0%	HI	2.0E-02	100.0%	Total ILCR	2.2E-06	100.0%	HI	3.7E-02	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lag times, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
ND - Not detected.
-- Not applicable.

TABLE D-77
ON-SITE RESIDENTS - AREA B- FUTURE SCENARIO
INHALATION OF OUTDOOR AMBIENT AIR
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	Local Adult Resident	Local Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFi	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDi	Oral reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of chemical in air (mg/m3)	CS	CS	
RR	Respiration rate (m3/hr)	0.83	0.6	
ET	Exposure time (hrs/d)	1	2	
EF	Exposure Frequency (d/yr)	150	150	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Ca (mg/m3)	CSFi 1/(mg/kg/d)	RfDi (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2,4-Trimethylbenzene	0.0003	NA	NA	6.3E-07	--	--	1.5E-06	--	--	8.5E-07	--	--	9.9E-06	--	--
Benzene	0.0007	0.029	0.00171	1.5E-06	4.2E-08	46.8%	3.4E-06	2.0E-03	26.1%	2.0E-06	5.7E-08	46.8%	2.3E-05	1.3E-02	26.1%
Bromomethane	0.0008	NA	0.00143	1.7E-06	--	--	3.9E-06	2.7E-03	35.7%	2.3E-06	--	--	2.6E-05	1.8E-02	35.7%
Hexachlorobutadiene	0.0003	0.077	0.0005	6.3E-07	4.8E-08	53.2%	1.5E-06	2.8E-03	38.2%	8.5E-07	6.5E-08	53.2%	9.9E-06	2.0E-02	38.2%
				Total ILCR:	9.1E-08	100.0%		HI:	7.6E-03	Total ILCR:	1.2E-07	100.0%		HI:	5.2E-02	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-78
ON-SITE RESIDENTS - AREA B LANDFILL AND POND - FUTURE SCENARIO
ACCIDENTAL INGESTION OF SURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	100	200	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
FI	Fraction of soil ingested from site	1	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.780	7.7	NA	4.6E-07	3.5E-06	22.8%	1.1E-06	--	--	8.5E-07	6.6E-06	22.8%	1.0E-05	--	--
Arsenic	11.6	1.75	0.0003	6.8E-06	1.2E-05	77.2%	1.6E-05	5.3E-02	35.5%	1.3E-05	2.2E-05	77.2%	1.5E-04	4.9E-01	35.5%
Cadmium	20.5	NA	0.0005	1.2E-05	--	--	2.8E-05	5.6E-02	37.6%	2.2E-05	--	--	2.6E-04	5.2E-01	37.6%
Chromium	44	NA	0.005	2.6E-05	--	--	6.1E-05	1.2E-02	8.1%	4.9E-05	--	--	5.7E-04	1.1E-01	8.1%
Manganese	102	NA	0.005	6.0E-05	--	--	1.4E-04	2.8E-02	18.7%	1.1E-04	--	--	1.3E-03	2.6E-01	18.7%
				Total ILCR:	1.5E-05	100.0%	HI:	1.5E-01	100.0%	Total ILCR:	2.9E-05	100.0%	HI:	1.4E+00	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-79
ON-SITE RESIDENTS - AREA B LANDFILL AND POND - FUTURE SCENARIO
DERMAL CONTACT WITH SURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	1	
ABS	Absorption fraction --			
	Cadmium and default organics:	0.01	0.01	
	Default inorganics:	0.001	0.001	
	PCBs	0.06	0.06	
A	Skin surface area available for contact (cm2)	5300	2006	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

					Adult Residents						Child Residents (0 - 6 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.780	0.06	7.7	NA	1.5E-06	1.1E-05	37.2%	3.4E-06	--	--	5.1E-07	4.0E-06	37.2%	6.0E-06	--	--
Arsenic	11.6	0.03	1.75	0.0003	1.1E-05	1.9E-05	62.8%	2.5E-05	8.4E-02	62.3%	3.8E-06	6.7E-06	62.8%	4.5E-05	1.5E-01	62.3%
Cadmium	20.5	0.01	NA	0.0005	6.4E-06	--	--	1.5E-05	3.0E-02	22.0%	2.3E-06	--	--	2.6E-05	5.3E-02	22.0%
Chromium	44	0.01	NA	0.005	1.4E-05	--	--	3.2E-05	6.4E-03	4.8%	4.9E-06	--	--	5.7E-05	1.1E-02	4.8%
Manganese	102	0.01	NA	0.005	3.2E-05	--	--	7.4E-05	1.5E-02	11.0%	1.1E-05	--	--	1.3E-04	2.6E-02	11.0%
					Total ILCR:	3.0E-05	100.0%	HI:	1.4E-01	100.0%	Total ILCR:	1.1E-05	100.0%	HI:	2.4E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-80
ON-SITE RESIDENTS - AREA B LANDFILL AND POND - FUTURE SCENARIO
INHALATION OF FUGITIVE DUSTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)
Where: Ca = Cs * (1/PEF)

ILCR = CDI*CSFI
HQ = CDI/RfDi

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFI	Inhalation cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDi	Inhalation reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of chemical in air as fugitive dusts (mg/m3)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
PEF	Particulate emission factor (m3/kg)	4.63E+09	4.63E+09	
RR	Respiration rate (m3/hr)	0.83	0.6	
ET	Exposure time (hrs/d)	1	2	
EF	Exposure Frequency (d/yr)	150	150	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

					Adult Residents						Child Residents (0 - 6 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	Ca (mg/m3)	CSFI 1/(mg/kg/d)	RfDI (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Aroclor-1260	0.780	1.68E-10	NA	NA	3.5E-13	--	--	8.2E-13	--	--	4.7E-13	--	--	5.5E-12	--	--
Arsenic	11.6	2.51E-09	15.1	0.0003	5.2E-12	7.9E-11	8.1%	1.2E-11	4.1E-08	0.5%	7.1E-12	1.1E-10	8.1%	8.2E-11	2.7E-07	0.5%
Cadmium	20.5	4.43E-09	6.3	NA	9.2E-12	5.8E-11	6.0%	2.2E-11	--	--	1.2E-11	7.9E-11	6.0%	1.5E-10	--	--
Chromium	44	9.50E-09	42	NA	2.0E-11	8.3E-10	85.9%	4.6E-11	--	--	2.7E-11	1.1E-09	85.9%	3.1E-10	--	--
Manganese	102	2.20E-08	NA	0.0000143	4.6E-11	--	--	1.1E-10	7.5E-06	99.5%	6.2E-11	--	--	7.2E-10	5.1E-05	99.5%
					Total ILCR:	9.7E-10	100.0%		HI: 7.5E-06	100.0%	Total ILCR:	1.3E-09	100.0%		HI: 5.1E-05	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-81
ON-SITE RESIDENTS - AREA B LANDFILL AND POND - FUTURE SCENARIO
ACCIDENTAL INGESTION OF SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*ET*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/hour)	0.05	0.05	
ET	Exposure time (hours/event)	2.6	2.6	
EF	Exposure Frequency (events/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.008	0.091	NA	5.9E-07	5.4E-08	1.3%	1.4E-06	--	--	2.2E-06	2.0E-07	1.3%	2.6E-05	--	--
Benzene	0.012	0.029	NA	8.9E-07	2.6E-08	0.6%	2.1E-06	--	--	3.3E-06	9.7E-08	0.6%	3.9E-05	--	--
Chloroform	0.0240	0.0061	0.01	1.8E-06	1.1E-08	0.3%	4.2E-06	4.2E-04	2.7%	6.7E-06	4.1E-08	0.3%	7.8E-05	7.8E-03	2.7%
Trichloroethene	0.045	0.011	0.006	3.3E-06	3.7E-08	0.9%	7.8E-06	1.3E-03	8.5%	1.3E-05	1.4E-07	0.9%	1.5E-04	2.4E-02	8.5%
Vinyl chloride	0.022	1.9	NA	1.6E-06	3.1E-06	75.5%	3.8E-06	--	--	6.1E-06	1.2E-05	75.5%	7.2E-05	--	--
Bis(2-ethylhexyl)phthalate	0.009	0.014	0.02	6.7E-07	9.3E-09	0.2%	1.6E-06	7.8E-05	0.5%	2.5E-06	3.5E-08	0.2%	2.9E-05	1.5E-03	0.5%
Aluminum	0.690	NA	1	5.1E-05	--	--	1.2E-04	1.2E-04	0.8%	1.9E-04	--	--	2.2E-03	2.2E-03	0.8%
Arsenic	0.0067	1.75	0.0003	5.0E-07	8.7E-07	21.2%	1.2E-06	3.9E-03	25.3%	1.9E-06	3.3E-06	21.2%	2.2E-05	7.3E-02	25.3%
Manganese	0.272	NA	0.005	2.0E-05	--	--	4.7E-05	9.4E-03	61.5%	7.6E-05	--	--	8.8E-04	1.8E-01	61.5%
Zinc	0.202	NA	0.3	1.5E-05	--	--	3.5E-05	1.2E-04	0.8%	5.6E-05	--	--	6.6E-04	2.2E-03	0.8%
				Total ILCR:	4.1E-06	100.0%	HI:	1.5E-02	100.0%	Total ILCR:	1.5E-05	100.0%	HI:	2.9E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-82
ON-SITE RESIDENTS - AREA B LANDFILL AND POND - FUTURE SCENARIO
DERMAL CONTACT WITH SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	5300	2006	
EF	Exposure frequency (d/yr)	34	137	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/event)	CS	CS	
Kp	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	2.6	2.6	

							Adult Residents						Child Residents (0 - 6 Yrs Old)					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
1,2-Dichloroethane	0.008	0.0053	1.7E-02	1.4E-07	0.091	NA	4.2E-07	3.8E-08	1.0%	9.9E-07	–	–	6.0E-07	5.5E-08	1.0%	7.0E-06	–	–
Benzene	0.012	2.1E-01	6.5E-01	7.8E-06	0.029	NA	2.4E-05	6.8E-07	17.3%	5.5E-05	–	–	3.4E-05	9.7E-07	17.3%	3.9E-04	–	–
Chloroform	0.0240	8.9E-03	3.1E-02	7.5E-07	0.0061	0.01	2.3E-06	1.4E-08	0.4%	5.3E-06	5.3E-04	7.4%	3.2E-06	2.0E-08	0.4%	3.8E-05	3.8E-03	7.4%
Trichloroethene	0.045	1.6E-02	5.9E-02	2.7E-06	0.011	0.006	8.0E-06	8.8E-08	2.2%	1.9E-05	3.1E-03	43.2%	1.1E-05	1.3E-07	2.2%	1.3E-04	2.2E-02	43.2%
Vinyl chloride	0.022	7.3E-03	2.2E-02	4.8E-07	1.9	NA	1.5E-06	2.8E-06	70.3%	3.4E-06	–	–	2.1E-06	4.0E-06	70.3%	2.4E-05	–	–
Bis(2-ethylhexyl)phthalate	0.009	3.3E-02	6.7E-01	6.1E-06	0.014	0.02	1.8E-05	2.6E-07	6.5%	4.3E-05	2.1E-03	29.6%	2.6E-05	3.7E-07	6.5%	3.0E-04	1.5E-02	29.6%
Aluminum	0.690	1.0E-03	NA	1.8E-06	NA	1	5.4E-06	–	–	1.3E-05	1.3E-05	0.2%	7.7E-06	–	–	9.0E-05	9.0E-05	0.2%
Arsenic	0.0067	1.0E-03	NA	1.7E-08	1.75	0.0003	5.3E-08	9.2E-08	2.3%	1.2E-07	4.1E-04	5.7%	7.5E-08	1.3E-07	2.3%	8.7E-07	2.9E-03	5.7%
Manganese	0.272	1.0E-03	NA	7.1E-07	NA	0.005	2.1E-06	–	–	5.0E-06	1.0E-03	13.8%	3.0E-06	–	–	3.5E-05	7.1E-03	13.8%
Zinc	0.202	1.0E-03	NA	5.3E-07	NA	0.3	1.6E-06	–	–	3.7E-06	1.2E-05	0.2%	2.3E-06	–	–	2.6E-05	8.8E-05	0.2%
							Total ILCR:	4.0E-06	100.0%	HI:	7.2E-03	100.0%	Total ILCR:	5.6E-06	100.0%	HI:	5.1E-02	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
– Not applicable.

TABLE D-83
ON-SITE RESIDENTS - AREA B LANDFILL AND POND - FUTURE SCENARIO
ACCIDENTAL INGESTION OF SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	100	200	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
FI	Fraction of sediment ingested from site	1	1	
EF	Exposure Frequency (d/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Vinyl chloride	0.060	1.9	NA	3.4E-09	6.5E-09	0.1%	8.0E-09	--	--	2.6E-08	4.9E-08	0.1%	3.0E-07	--	--
4,4'-DDD	4.20	0.24	NA	2.4E-07	5.7E-08	0.9%	5.6E-07	--	--	1.8E-06	4.3E-07	0.9%	2.1E-05	--	--
4,4'-DDE	0.850	0.34	NA	4.8E-08	1.6E-08	0.2%	1.1E-07	--	--	3.6E-07	1.2E-07	0.2%	4.3E-06	--	--
Aroclor-1254	5.78	7.7	NA	3.3E-07	2.5E-06	38.1%	7.7E-07	--	--	2.5E-06	1.9E-05	38.1%	2.9E-05	--	--
Dieldrin	0.068	16	5E-05	3.9E-09	6.2E-08	0.9%	9.0E-09	1.8E-04	0.4%	2.9E-08	4.6E-07	0.9%	3.4E-07	6.8E-03	0.4%
Aluminum	22768	NA	1	1.3E-03	--	--	3.0E-03	3.0E-03	6.7%	9.8E-03	--	--	1.1E-01	1.1E-01	6.7%
Antimony	16	NA	0.0004	9.1E-07	--	--	2.1E-06	5.3E-03	11.8%	6.9E-06	--	--	8.0E-05	2.0E-01	11.8%
Arsenic	38.0	1.75	0.0003	2.2E-06	3.8E-06	56.9%	5.1E-06	1.7E-02	37.3%	1.6E-05	2.9E-05	56.9%	1.9E-04	6.3E-01	37.3%
Beryllium	0.76	4.3	0.005	4.3E-08	1.9E-07	2.8%	1.0E-07	2.0E-05	0.0%	3.3E-07	1.4E-06	2.8%	3.8E-06	7.6E-04	0.0%
Cadmium	34.5	NA	0.0005	2.0E-06	--	--	4.6E-06	9.2E-03	20.3%	1.5E-05	--	--	1.7E-04	3.5E-01	20.3%
Copper	298	NA	0.0371	1.7E-05	--	--	4.0E-05	1.1E-03	2.4%	1.3E-04	--	--	1.5E-03	4.0E-02	2.4%
Lead	455	NA	NA	2.6E-05	--	--	6.0E-05	--	--	1.9E-04	--	--	2.3E-03	--	--
Manganese	240	NA	0.005	1.4E-05	--	--	3.2E-05	6.4E-03	14.1%	1.0E-04	--	--	1.2E-03	2.4E-01	14.1%
Mercury	0.35	NA	0.0003	2.0E-08	--	--	4.7E-08	1.6E-04	0.3%	1.5E-07	--	--	1.8E-06	5.8E-03	0.3%
Silver	13.5	NA	0.005	7.7E-07	--	--	1.8E-06	3.6E-04	0.8%	5.8E-06	--	--	6.8E-05	1.4E-02	0.8%
Vanadium	114	NA	0.007	6.5E-06	--	--	1.5E-05	2.2E-03	4.8%	4.9E-05	--	--	5.7E-04	8.2E-02	4.8%
Zinc	995	NA	0.3	5.7E-05	--	--	1.3E-04	4.4E-04	1.0%	4.3E-04	--	--	5.0E-03	1.7E-02	1.0%
				Total ILCR:	6.7E-06	100.0%		HI:	4.5E-02	100.0%	Total ILCR:	5.0E-05	100.0%	HI:	1.7E+00	100.0%

NOTES:
-- Not applicable.

TABLE D-84
ON-SITE RESIDENTS - AREA B LANDFILL AND POND - FUTURE SCENARIO
DERMAL CONTACT WITH SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	1	
ABS	Absorption fraction --			
	Cadmium and default organics:	0.01	0.01	
	Default inorganics:	0.001	0.001	
	PCBs	0.06	0.06	
A	Skin surface area available for contact (cm2)	5300	2006	
EF	Exposure Frequency (d/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

					Adult Residents						Child Residents (0 - 6 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Vinyl chloride	0.060	0.25	1.9	NA	4.5E-08	8.6E-08	0.6%	1.1E-07	--	--	6.5E-08	1.2E-07	0.6%	7.5E-07	--	--
4,4'-DDD	4.20	0.1	0.24	NA	1.3E-06	3.0E-07	2.0%	3.0E-06	--	--	1.8E-06	4.3E-07	2.0%	2.1E-05	--	--
4,4'-DDE	0.850	0.1	0.34	NA	2.6E-07	8.7E-08	0.6%	6.0E-07	--	--	3.7E-07	1.2E-07	0.6%	4.3E-06	--	--
Aroclor-1254	5.78	0.06	7.7	NA	1.0E-06	8.1E-06	53.8%	2.4E-06	--	--	1.5E-06	1.1E-05	53.8%	1.7E-05	--	--
Dieldrin	0.068	0.1	16	5E-05	2.0E-08	3.3E-07	2.2%	4.8E-08	9.5E-04	2.2%	2.9E-08	4.7E-07	2.2%	3.4E-07	6.8E-03	2.2%
Aluminum	22768	0.01	NA	1	6.9E-04	--	--	1.6E-03	1.6E-03	3.8%	9.8E-04	--	--	1.1E-02	1.1E-02	3.8%
Antimony	16	0.01	NA	0.0004	4.8E-07	--	--	1.1E-06	2.8E-03	6.6%	6.9E-07	--	--	8.0E-06	2.0E-02	6.6%
Arsenic	38.0	0.03	1.75	0.0003	3.4E-06	6.0E-06	40.2%	8.0E-06	2.7E-02	62.8%	4.9E-06	8.6E-06	40.2%	5.7E-05	1.9E-01	62.8%
Beryllium	0.76	0.01	4.3	0.005	2.3E-08	9.9E-08	0.7%	5.4E-08	1.1E-05	0.0%	3.3E-08	1.4E-07	0.7%	3.8E-07	7.6E-05	0.0%
Cadmium	34.5	0.01	NA	0.0005	1.0E-06	--	--	2.4E-06	4.9E-03	11.4%	1.5E-06	--	--	1.7E-05	3.5E-02	11.4%
Copper	298	0.01	NA	0.0371	9.0E-06	--	--	2.1E-05	5.7E-04	1.3%	1.3E-05	--	--	1.5E-04	4.0E-03	1.3%
Lead	455	0.01	NA	NA	1.4E-05	--	--	3.2E-05	--	--	2.0E-05	--	--	2.3E-04	--	--
Manganese	240	0.01	NA	0.005	7.3E-06	--	--	1.7E-05	3.4E-03	7.9%	1.0E-05	--	--	1.2E-04	2.4E-02	7.9%
Mercury	0.35	0.01	NA	0.0003	1.1E-08	--	--	2.5E-08	8.2E-05	0.2%	1.5E-08	--	--	1.8E-07	5.9E-04	0.2%
Silver	13.5	0.01	NA	0.005	4.1E-07	--	--	9.5E-07	1.9E-04	0.4%	5.8E-07	--	--	6.8E-06	1.4E-03	0.4%
Vanadium	114	0.01	NA	0.007	3.5E-06	--	--	8.1E-06	1.2E-03	2.7%	4.9E-06	--	--	5.7E-05	8.2E-03	2.7%
Zinc	995	0.01	NA	0.3	3.0E-05	--	--	7.0E-05	2.3E-04	0.5%	4.3E-05	--	--	5.0E-04	1.7E-03	0.5%
					Total ILCR:	1.5E-05	100.0%		HI: 4.3E-02	100.0%	Total ILCR:	2.1E-05	100.0%		HI: 3.0E-01	100.0%

NOTES:
-- Not applicable.

TABLE D-85
ON-SITE RESIDENTS - AREA B LANDFILL AND POND - FUTURE SCENARIO
ACCIDENTAL INGESTION OF DEEP SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	100	200	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
FI	Fraction of sediment ingested from site	1	1	
EF	Exposure Frequency (d/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Vinyl chloride	0.010	1.9	NA	5.7E-10	1.1E-09	0.5%	1.3E-09	--	--	4.3E-09	8.2E-09	0.5%	5.0E-08	--	--
4,4'-DDE	0.060	0.34	NA	3.4E-09	1.2E-09	0.5%	8.0E-09	--	--	2.6E-08	8.8E-09	0.5%	3.0E-07	--	--
4,4'-DDT	4.4	0.34	0.0005	2.5E-07	8.5E-08	37.9%	5.9E-07	1.2E-03	18.8%	1.9E-06	6.4E-07	37.9%	2.2E-05	4.4E-02	18.8%
Beryllium	0.56	4.3	0.005	3.2E-08	1.4E-07	61.1%	7.5E-08	1.5E-05	0.2%	2.4E-07	1.0E-06	61.1%	2.8E-06	5.6E-04	0.2%
Cadmium	12.0	NA	0.0005	6.8E-07	--	--	1.6E-06	3.2E-03	51.2%	5.1E-06	--	--	6.0E-05	1.2E-01	51.2%
Manganese	69.6	NA	0.005	4.0E-06	--	--	9.3E-06	1.9E-03	29.7%	3.0E-05	--	--	3.5E-04	7.0E-02	29.7%
					Total ILCR:	2.2E-07	100.0%	HI:	6.2E-03	100.0%	Total ILCR:	1.7E-06	100.0%	HI:	2.3E-01	100.0%

NOTES:
-- Not applicable.

TABLE D-86
ON-SITE RESIDENTS - AREA B LANDFILL AND POND - FUTURE SCENARIO
DERMAL CONTACT WITH DEEP SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d) (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/k	CS	CS	
CF	*Conversion factor (kg/mg)*	*1E-06*	*1E-06*	
AF	Soil to skin adherence factor (mg/cm2-event)	1	1	
ABS	Absorption fraction --			
	Cadmium and default organics:	0.01	0.01	
	Default inorganics:	0.001	0.001	
	PCBs	0.06	0.06	
A	Skin surface area available for contact (cm2)	5300	2006	
EF	Exposure Frequency (d/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

					Adult Residents						Child Residents (0 - 6 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Vinyl chloride	0.010	0.25	1.9	NA	7.6E-09	1.4E-08	2.6%	1.8E-08	--	--	1.1E-08	2.0E-08	2.6%	1.3E-07	--	--
4,4'-DDE	0.060	0.1	0.34	NA	1.8E-08	6.2E-09	1.1%	4.2E-08	--	--	2.6E-08	8.8E-09	1.1%	3.0E-07	--	--
4,4'-DDT	4.4	0.1	0.34	0.0005	1.3E-06	4.5E-07	82.9%	3.1E-06	6.2E-03	69.8%	1.9E-06	6.4E-07	82.9%	2.2E-05	4.4E-02	69.8%
Beryllium	0.56	0.01	4.3	0.005	1.7E-08	7.3E-08	13.3%	3.9E-08	7.9E-06	0.1%	2.4E-08	1.0E-07	13.3%	2.8E-07	5.6E-05	0.1%
Cadmium	12.0	0.01	NA	0.0005	3.6E-07	--	--	8.5E-07	1.7E-03	19.0%	5.2E-07	--	--	6.0E-06	1.2E-02	19.0%
Manganese	69.6	0.01	NA	0.005	2.1E-06	--	--	4.9E-06	9.8E-04	11.0%	3.0E-06	--	--	3.5E-05	7.0E-03	11.0%
					Total ILCR:	5.5E-07	100.0%	HI:	8.9E-03	100.0%	Total ILCR:	7.8E-07	100.0%	HI:	6.3E-02	100.0%

NOTES:
-- Not applicable.

TABLE D-87
ON-SITE RESIDENTS - AREA B, ELEMENTARY SCHOOL AREA - FUTURE SCENARIO
ACCIDENTAL INGESTION OF SURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	100	200	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
FI	Fraction of soil ingested from site	1	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Antimony	7.8	NA	0.0004	4.6E-06	--	--	1.1E-05	2.7E-02	6.3%	8.5E-06	--	--	1.0E-04	2.5E-01	6.3%
Arsenic	25.1	1.75	0.0003	1.5E-05	2.6E-05	100.0%	3.4E-05	1.1E-01	27.2%	2.8E-05	4.8E-05	100.0%	3.2E-04	1.1E+00	27.2%
Chromium	869	NA	0.005	5.1E-04	--	--	1.2E-03	2.4E-01	56.5%	9.5E-04	--	--	1.1E-02	2.2E+00	56.5%
Manganese	61.2	NA	0.005	3.6E-05	--	--	8.4E-05	1.7E-02	4.0%	6.7E-05	--	--	7.8E-04	1.6E-01	4.0%
Vanadium	128	NA	0.007	7.5E-05	--	--	1.8E-04	2.5E-02	5.9%	1.4E-04	--	--	1.6E-03	2.3E-01	5.9%
				Total ILCR:	2.6E-05	100.0%	HI:	4.2E-01	100.0%	Total ILCR:	4.8E-05	100.0%	HI:	3.9E+00	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-88
ON-SITE RESIDENTS - AREA B, ELEMENTARY SCHOOL AREA - FUTURE SCENARIO
DERMAL CONTACT WITH SURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	1	
ABS	Absorption fraction --			
	Cadmium and default organics:	0.01	0.01	
	Default inorganics:	0.001	0.001	
	PCBs	0.06	0.06	
A	Skin surface area available for contact (cm2)	5300	2006	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

					Adult Residents						Child Residents (0 - 6 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Antimony	7.8	0.01	NA	0.0004	2.4E-06	--	--	5.7E-06	1.4E-02	4.1%	8.6E-07	--	--	1.0E-05	2.5E-02	4.1%
Arsenic	25.1	0.03	1.75	0.0003	2.3E-05	4.1E-05	100.0%	5.5E-05	1.8E-01	52.9%	8.3E-06	1.4E-05	100.0%	9.7E-05	3.2E-01	52.9%
Chromium	869	0.01	NA	0.005	2.7E-04	--	--	6.3E-04	1.3E-01	36.6%	9.6E-05	--	--	1.1E-03	2.2E-01	36.6%
Manganese	61.2	0.01	NA	0.005	1.9E-05	--	--	4.4E-05	8.9E-03	2.6%	6.7E-06	--	--	7.8E-05	1.6E-02	2.6%
Vanadium	128	0.01	NA	0.007	4.0E-05	--	--	9.3E-05	1.3E-02	3.9%	1.4E-05	--	--	1.6E-04	2.3E-02	3.9%
					Total ILCR:	4.1E-05	100.0%	HI:	3.4E-01	100.0%	Total ILCR:	1.4E-05	100.0%	HI:	6.1E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-89
ON-SITE RESIDENTS - AREA B, ELEMENTARY SCHOOL AREA - FUTURE SCENARIO
INHALATION OF FUGITIVE DUSTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)
Where: Ca = Cs * (1/PEF)

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFi	Inhalation cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDi	Inhalation reference dose (mg/kg/d)	CS	CS	
Ca	Concentration of chemical in air as fugitive dusts (mg/m3)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
PEF	Particulate emission factor (m3/kg)	4.63E+09	4.63E+09	
RR	Respiration rate (m3/hr)	0.83	0.6	
ET	Exposure time (hrs/d)	1	2	
EF	Exposure Frequency (d/yr)	150	150	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

					Adult Residents						Child Residents (0 - 6 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	Ca (mg/m3)	CSFi 1/(mg/kg/d)	RfDi (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Antimony	7.8	1.68E-09	NA	NA	3.5E-12	--	--	8.2E-12	--	--	4.7E-12	--	--	5.5E-11	--	--
Arsenic	25.1	5.42E-09	15.1	0.0003	1.1E-11	1.7E-10	1.0%	2.6E-11	8.8E-08	1.9%	1.5E-11	2.3E-10	1.0%	1.8E-10	5.9E-07	1.9%
Chromium	869	1.88E-07	42	NA	3.9E-10	1.6E-08	99.0%	9.1E-10	--	--	5.3E-10	2.2E-08	99.0%	6.2E-09	--	--
Manganese	61.2	1.32E-08	NA	0.0000143	2.8E-11	--	--	6.4E-11	4.5E-06	98.1%	3.7E-11	--	--	4.3E-10	3.0E-05	98.1%
Vanadium	128	2.76E-08	NA	NA	5.8E-11	--	--	1.3E-10	--	--	7.8E-11	--	--	9.1E-10	--	--
					Total ILCR:	1.7E-08	100.0%	HI:	4.6E-06	100.0%	Total ILCR:	2.2E-08	100.0%	HI:	3.1E-05	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-90
ON-SITE RESIDENTS - AREA B, ELEMENTARY SCHOOL AREA - FUTURE SCENARIO
ACCIDENTAL INGESTION OF SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cw*IR*ET*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
IR	Ingestion Rate (L/hour)	0.05	0.05	
ET	Exposure time (hours/event)	2.6	2.6	
EF	Exposure Frequency (events/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
4,4'-DDD	0.00004	0.24	NA	2.8E-09	6.8E-10	0.0%	6.6E-09	--	--	1.1E-08	2.5E-09	0.0%	1.2E-07	--	--
Arsenic	0.012	1.75	0.0003	8.9E-07	1.6E-06	100.0%	2.1E-06	6.9E-03	25.7%	3.3E-06	5.9E-06	100.0%	3.9E-05	8.1E-06	0.1%
Lead	0.054000	NA	NA	4.0E-06	--	--	9.3E-06	--	--	1.5E-05	--	--	1.8E-04	--	--
Manganese	0.574	NA	0.005	4.3E-05	--	--	9.9E-05	2.0E-02	73.8%	1.6E-04	--	--	1.9E-03	1.3E-04	1.6%
Zinc	0.199	NA	0.3	1.5E-05	--	--	3.4E-05	1.1E-04	0.4%	5.5E-05	--	--	6.5E-04	8.1E-03	98.3%
				Total ILCR	1.6E-06	100.0%	HI:	2.7E-02	100.0%	Total ILCR:	5.9E-06	100.0%	HI:	8.2E-03	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-91
ON-SITE RESIDENTS - AREA B, ELEMENTARY SCHOOL AREA - FUTURE SCENARIO
DERMAL CONTACT WITH SURFACE WATER
ORGANIC AND UNFILTERED INORGANIC COPCs
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (DA*A*EF*ED*EV)/(BW*AT)
For organics: DA = Cw * CF * Kp' (See Section 3.0 for derivation of Kp' [1])
For inorganics: DA = Cw * CF * Kp * tevent

ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
DA	Absorbed dose per event (mg/cm2-event)	CS	CS	
A	Skin surface area available for contact (cm2)	5300	2006	
EF	Exposure frequency (d/yr)	34	137	
ED	Exposure duration (yrs)	30	6	
EV	Event frequency (events/day)	1	1	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	
Cw	Concentration of chemical in water (mg/L)	CS	CS	
CF	Conversion factor (L/cm3)	0.001	0.001	
Kp'	Adjusted dermal permeability coefficient (cm/event)	CS	CS	
Kp'	Dermal permeability coefficient (cm/hour)	CS	CS	
tevent	Duration of event (hours/event)	2.6	2.6	

							Adult Residents						Child Residents (0 - 6 Yrs Old)					
							Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cw (mg/L)	Kp (cm/hour)	Kp' (cm/event)	DA (mg/cm2-event)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
4,4'-DDD	0.000038	2.8E-01	3.5E+00	1.3E-07	0.24	NA	4.0E-07	9.6E-08	36.8%	9.3E-07	--	--	5.7E-07	1.4E-07	36.8%	6.6E-06	--	--
Arsenic	0.012	1.0E-03	NA	3.1E-08	1.75	0.0003	9.4E-08	1.7E-07	63.2%	2.2E-07	7.3E-04	25.7%	1.3E-07	2.3E-07	63.2%	1.6E-06	5.2E-03	25.7%
Lead	0.054	1.0E-03	NA	1.4E-07	NA	NA	4.2E-07	--	--	9.9E-07	--	--	6.0E-07	--	--	7.0E-06	--	--
Manganese	0.574	1.0E-03	NA	1.5E-06	NA	0.005	4.5E-06	--	--	1.1E-05	2.1E-03	73.8%	6.4E-06	--	--	7.5E-05	1.5E-02	73.8%
Zinc	0.199	1.0E-03	NA	5.2E-07	NA	0.3	1.6E-06	--	--	3.6E-06	1.2E-05	0.4%	2.2E-06	--	--	2.6E-05	8.7E-05	0.4%
							Total ILCR:	2.6E-07	100.0%	HI:	2.9E-03	100.0%	Total ILCR:	3.7E-07	100.0%	HI:	2.0E-02	100.0%

NOTES:
[1] Adjusted dermal permeability coefficients, Kp', were derived for COPCs from chemical-specific dermal permeability coefficients, lagtimes, partitioning constants, and the duration of the exposure event. Equations and values of constants were obtained from Dermal Exposure Assessment: Principles and Applications, Interim Report, USEPA, January, 1992. Both Kp and Kp' are presented in the spreadsheet.

NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-92
ON-SITE RESIDENTS - AREA B, ELEMENTARY SCHOOL AREA - FUTURE SCENARIO
ACCIDENTAL INGESTION OF SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	100	200	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
FI	Fraction of sediment ingested from site	1	1	
EF	Exposure Frequency (d/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
NO COPCs	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--

NOTES:
-- Not applicable.

TABLE D-93
ON-SITE RESIDENTS - AREA B, ELEMENTARY SCHOOL AREA - FUTURE SCENARIO
DERMAL CONTACT WITH SHALLOW SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	1	
ABS	Absorption fraction –			
	Cadmium and default organics:	0.01	0.01	
	Default inorganics:	0.001	0.001	
	PCBs	0.06	0.06	
A	Skin surface area available for contact (cm2)	5300	2006	
EF	Exposure Frequency (d/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

					Adult Residents						Child Residents (0 - 6 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
NO COPCs	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–

NOTES:
– Not applicable.

TABLE D-94
ON-SITE RESIDENTS - AREA B, ELEMENTARY SCHOOL AREA - FUTURE SCENARIO
ACCIDENTAL INGESTION OF DEEP SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
CDI	Chronic daily intake (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	100	200	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
FI	Fraction of sediment ingested from site	1	1	
EF	Exposure Frequency (d/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

				Adult Residents						Child Residents (0 - 6 Yrs Old)					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Lead	310	NA	NA	1.8E-05	--	--	4.1E-05	--	--	1.3E-04	--	--	1.6E-03	--	--
Mercury	0.8	NA	0.0003	4.6E-08	--	--	1.1E-07	3.5E-04	100.0%	3.4E-07	--	--	4.0E-06	1.3E-02	100.0%
				Total ILCR:	--	--	HI:	3.5E-04	100.0%	Total ILCR:	--	--	HI:	1.3E-02	100.0%

NOTES:
-- Not applicable.

TABLE D-95
ON-SITE RESIDENTS - AREA B, ELEMENTARY SCHOOL AREA - FUTURE SCENARIO
DERMAL CONTACT WITH DEEP SEDIMENTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d) (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Adult Resident	On-site Child Resident (0 - 6 Yrs Old)	
DAD	Dermally absorbed dose (mg/kg/d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	CS	
Cs	Concentration of chemical in sediment (mg/k	CS	CS	
CF	Conversion factor (kg/mg)	1E-06	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	1	
ABS	Absorption fraction --			
	Cadmium and default organics:	0.01	0.01	
	Default inorganics:	0.001	0.001	
	PCBs	0.06	0.06	
A	Skin surface area available for contact (cm2	5300	2006	
EF	Exposure Frequency (d/yr)	34	137	
ED	Exposure Duration (yrs)	30	6	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25550	25550	
ATn	Averaging time, noncarcinogens (d)	10950	2190	

					Adult Residents						Child Residents (0 - 6 Yrs Old)					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Lead	310	0.01	NA	NA	9.4E-06	--	--	2.2E-05	--	--	1.3E-05	--	--	1.6E-04	--	--
Mercury	0.8	0.01	NA	0.0003	2.4E-08	--	--	5.6E-08	1.9E-04	100.0%	3.4E-08	--	--	4.0E-07	1.3E-03	100.0%
					Total ILCR:	--	--	HI:	1.9E-04	100.0%	Total ILCR:	--	--	HI:	1.3E-03	100.0%

NOTES:
-- Not applicable.

TABLE D-96
CONSTRUCTION WORKERS - AREA B - FUTURE SCENARIO
ACCIDENTAL INGESTION OF SUBSURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Future Construction Worker	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
IR	Ingestion Rate (mg/d)	480	
CF	Conversion factor (kg/mg)	1E-06	
FI	Fraction of soil ingested from site	1	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	1	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	365	

				Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Trichloroethene	2.83	0.011	0.006	1.9E-07	2.1E-09	0.0%	1.3E-05	2.2E-03	0.3%
Vinyl chloride	0.016	1.9	NA	1.1E-09	2.0E-09	0.0%	7.5E-08	--	--
4,4'-DDD	1.24	0.24	NA	8.3E-08	2.0E-08	0.4%	5.8E-06	--	--
Aroclor-1254	3.11	7.7	NA	2.1E-07	1.6E-06	30.7%	1.5E-05	--	--
Dieldrin	0.491	16	0.00005	3.3E-08	5.3E-07	10.1%	2.3E-06	4.6E-02	7.0%
Aluminum	11351	NA	1	7.6E-04	--	--	5.3E-02	5.3E-02	8.1%
Antimony	5.25	NA	0.0004	3.5E-07	--	--	2.5E-05	6.2E-02	9.4%
Arsenic	21.0	1.75	0.0003	1.4E-06	2.5E-06	47.1%	9.9E-05	3.3E-01	50.0%
Barium	497	NA	0.07	3.3E-05	--	--	2.3E-03	3.3E-02	5.1%
Beryllium	2.11	4.3	0.005	1.4E-07	6.1E-07	11.6%	9.9E-06	2.0E-03	0.3%
Manganese	34.6	NA	0.005	2.3E-06	--	--	1.6E-04	3.2E-02	4.9%
Thallium	1.02	NA	0.00008	6.8E-08	--	--	4.8E-06	6.0E-02	9.1%
Vanadium	55.7	NA	0.007	3.7E-06	--	--	2.6E-04	3.7E-02	5.7%
				Total ILCR:	5.2E-06	100.0%	HI:	6.6E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-97
CONSTRUCTION WORKERS - AREA B - FUTURE SCENARIO
DERMAL CONTACT WITH SUBSURFACE SOILS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

DAD (mg/kg/d)= (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Future Construction Worker	
DAD	Dermally absorbed dose (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg/d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
CF	Conversion factor (kg/mg)	1E-06	
AF	Soil to skin adherence factor (mg/cm2-event)	1	
ABS	Absorption fraction --		
	Cadmium and default organics:	0.01	
	Default Inorganics:	0.001	
	PCBs	0.06	
A	Skin surface area available for contact (cm2)	4100	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	1	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	365	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFo 1/(mg/kg/d)	RfDo (mg/kg/d)	DAD (mg/kg/d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg/d)	HQ	% Contrib. HI
Trichloroethene	2.83	0.25	0.011	0.006	4.1E-07	4.5E-09	0.2%	2.8E-05	4.7E-03	3.1%
Vinyl chloride	0.016	0.25	1.9	NA	2.3E-09	4.4E-09	0.2%	1.6E-07	--	--
4,4'-DDD	1.24	0.1	0.24	NA	7.1E-08	1.7E-08	0.9%	5.0E-06	--	--
Aroclor-1254	3.11	0.06	7.7	NA	1.1E-07	8.2E-07	41.5%	7.5E-06	--	--
Dieldrin	0.491	0.1	16	0.00005	2.8E-08	4.5E-07	22.7%	2.0E-06	3.9E-02	25.9%
Aluminum	11351	0.01	NA	1	6.5E-05	--	--	4.6E-03	4.6E-03	3.0%
Antimony	5.25	0.01	NA	0.0004	3.0E-08	--	--	2.1E-06	5.3E-03	3.5%
Arsenic	21.0	0.03	1.75	0.0003	3.6E-07	6.3E-07	31.8%	2.5E-05	8.4E-02	55.3%
Barium	497	0.01	NA	0.07	2.9E-06	--	--	2.0E-04	2.9E-03	1.9%
Beryllium	2.11	0.01	4.3	0.005	1.2E-08	5.2E-08	2.6%	8.5E-07	1.7E-04	0.1%
Manganese	34.6	0.01	NA	0.005	2.0E-07	--	--	1.4E-05	2.8E-03	1.8%
Thallium	1.02	0.01	NA	0.00008	5.8E-09	--	--	4.1E-07	5.1E-03	3.4%
Vanadium	55.7	0.01	NA	0.007	3.2E-07	--	--	2.2E-05	3.2E-03	2.1%
					Total ILCR:	2.0E-06	100.0%	HI:	1.5E-01	100.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TABLE D-98
CONSTRUCTION WORKERS - AREA B - FUTURE SCENARIO
INHALATION OF FUGITIVE DUSTS
SUMMARY OF CHRONIC DAILY INTAKES (CDIs),
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
CAMP ALLEN LANDFILL

CDI (mg/kg/d)= (Ca*RR*ET*EF*ED)/(BW*AT)
Where: Ca = Cs * (1/PEF)

ILCR = CDI*CSFI
HQ = CDI/RfDI

Parameter	Description	Future Construction Worker	
CDI	Chronic daily intake (mg/kg/d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFI	Inhalation cancer slope factor (1/(mg/kg/d))	CS	
HQ	Hazard quotient	CS	
RfDi	Inhalation reference dose (mg/kg/d)	CS	
Ca	Concentration of chemical in air as fugitive dusts (mg/m3)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
PEF	Particulate emission factor (m3/kg)	4.63E+09	
RR	Respiration rate (m3/hr)	0.83	
ET	Exposure time (hrs/d)	8	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	1	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25550	
ATn	Averaging time, noncarcinogens (d)	365	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	Ca (mg/m3)	CSFI 1/(mg/kg/	RfDi (mg/kg/d)	CDI (mg/kg/d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg/d)	HQ	% Contrib. HI
Trichloroethene	2.83	6.11E-10	0.006	NA	5.7E-13	3.4E-15	0.0%	4.0E-11	–	–
Vinyl chloride	0.016	3.46E-12	0.3	NA	3.2E-15	9.6E-16	0.0%	2.2E-13	–	–
4,4'-DDD	1.24	2.68E-10	NA	NA	2.5E-13	–	–	1.7E-11	–	–
Aroclor-1254	3.11	6.72E-10	NA	NA	6.2E-13	–	–	4.4E-11	–	–
Dieldrin	0.491	1.06E-10	16.1	NA	9.8E-14	1.6E-12	2.3%	6.9E-12	–	–
Aluminum	11351	2.45E-06	NA	1	2.3E-09	–	–	1.6E-07	–	–
Antimony	5.25	1.13E-09	NA	NA	1.1E-12	–	–	7.4E-11	–	–
Arsenic	21.0	4.54E-09	15.1	NA	4.2E-12	6.4E-11	92.5%	2.9E-10	–	–
Barium	497	1.07E-07	NA	0.000143	1.0E-10	–	–	7.0E-09	4.9E-05	59.0%
Beryllium	2.11	4.56E-10	8.4	NA	4.2E-13	3.6E-12	5.2%	3.0E-11	–	–
Manganese	34.6	7.47E-09	NA	0.0000143	6.9E-12	–	–	4.9E-10	3.4E-05	41.0%
Thallium	1.02	2.20E-10	NA	NA	2.0E-13	–	–	1.4E-11	–	–
Vanadium	55.7	1.20E-08	NA	NA	1.1E-11	–	–	7.8E-10	–	–
					Total ILCR:	6.9E-11	100.0%	HI:	8.3E-05	100.0%

NOTES:
NA - Toxicity criterion not available.
– Not applicable.