EPA Contract Number
68-W6-0053

RTI Project Number
92U-7298-027

Risk Assessment Technical Background Document for the Chlorinated Aliphatics Listing Determination

July 30, 1999

Prepared by
Center for Environmental Analysis
Research Triangle Institute
Research Triangle Park, NC 27709

and

The Office of Solid Waste
U.S. Environmental Protection Agency
401 M Street, SW (5307W)
Washington, DC 20460

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List of Acronyms

CalEPA - California Environmental Protection Agency
CDDs - Chlorinated dibenzodioxins
CDFs - Chlorinated dibenzofurans
CFR - Code of Federal Regulations
CHEMDAT8 - EPA Model for Estimating Organic Emissions
COCs - Constituents of Concern
COPCs - Constituents of Potential Concern
CSF - Cancer Slope Factor
DAF - Dilution/Attenuation Factor
EDF - Environmental Defense Fund
EPA - Environmental Protection Agency
EPACMTP - EPA's Composite Model for Leachate Migration with Transformation Products
EDC - Ethylene Dichloride
EPC - Exposure Point Concentration
f_{oc} - Fraction Organic Carbon
HEAST - Health Effects Assessment Summary Tables
HpCDD - Heptachlorodibenzodioxin
HpCDF - Heptachlorodibenzofuran
HWIR - Hazardous Waste Identification Rule
HxCDD - Hexachlorodibenzodioxin
HxCDF - Hexachlorodibenzofuran
HQ - Hazard Quotient
IEM - Indirect Exposure Model
ISCST3 - Industrial Source Complex Dispersion Model
K_d - Soil/water partition coefficient
K_{oc} - Organic carbon partition coefficient
K_{ow} - Octanol/water partition coefficient
LOAEL - Lowest observed adverse effect level
$\text{LOAEL}_{\text{HEC}}$ - Lowest observed adverse effect level - human equivalent concentration
MCL - Maximum Contaminant Level
NCEA - National Center for Environmental Assessment
NOAEL - No observed adverse effect level
$\text{NOAEL}_{\text{HEC}}$ - No observed adverse effect level - human equivalent concentration
NPDES - National Pollutant Discharge Elimination System
NWS - National Weather Service
OCDD - Octachlorodibenzodioxin
OCDF - Octachlorodibenzofuran
PCBs - Polychlorinated biphenyls
PDF - Probability density function
PeCDD - Pentachlorodibenzodioxin
PeCDF - Pentachlorodibenzofuran
PM10 - Particulate matter with a diameter of 10 micrometers or less
PM30 - Particulate matter with a diameter of 30 micrometers or less

List of Acronyms (continued)

POTW	-	Publicly owned treatment works
ppm	-	Parts per million
PrOTW	-	Privately owned treatment works
QA/QC	-	Quality Assurance/Quality Control
RCRA	-	Resource Conservation and Recovery Act
RfC	-	Reference Concentration
RfD	-	Reference Dose
SAB	-	Science Advisory Board
SVOC	-	Semivolatile Organic Compound
TCDD	-	2,3,7,8-Tetrachlorodibenzo-p-dioxin
TCDF	-	Tetrachlorodibenzofuran
TCLP	-	Toxicity Characteristic Leaching Procedure
TEQ	-	Toxicity Equivalent
TOC	-	Total Organic Carbon
TWA	-	Time weighted average
UACs	-	Unitized air concentrations
URF	-	Unit Risk Factor
USLE	-	Universal Soil Loss Equation
VCM	-	Vinyl Chloride Monomer
VCM-A	-	Vinyl Chloride Monomer production process that uses mercuric chloride catalyst in an acetylene-based process
VOCs	-	Volatile Organic Compounds

1.0 INTRODUCTION

1.1 Background

In 1989, the Environmental Defense Fund (EDF) sued the U.S. Environmental Protection Agency (EPA), in part, for failing to meet the statutory deadlines of Section 3001(e)(2) of the Resource Conservation and Recovery Act (RCRA; EDF vs. Browner; Civ.No. 89-0598 D.D.C.). To resolve most of the issues in the case, EDF and EPA entered into a consent decree that sets out an extensive series of deadlines for promulgating RCRA rules and for completing certain studies and reports. Paragraph 1.m. of the consent decree requires EPA to propose a hazardous waste listing determination for wastewaters and wastewater treatment sludges generated from the production of chlorinated aliphatic[1] chemicals, specifically, those chemicals described in the (existing) F024 listing description (40 Code of Federal Regulations [CFR] Part 261, Subpart D). According to the consent decree, EPA must propose the listing determination by July 30, 1999, and promulgate the final listing determination on or before September 30, 2000.

For listing determinations, we summarize the results of our technical data collection and analysis efforts in three key documents: a Listing Determination Technical Background Document, an Economics Background Document and a Risk Assessment Technical Background Document. The Listing Determination Technical Background Document is the “primary” background document and provides a description of the methodologies EPA employed to characterize the industry and wastes that are the subject of the listing determination, a summary of the data and information collected, and supporting data analyses. The Economics Background Document provides estimates of national industry compliance costs for the proposed listing decisions. The Risk Assessment Technical Background Document provides the results of EPA’s human health and ecological risk assessments for the wastes that are the subject of the listing determination. This document represents the Chlorinated Aliphatics Listing Determination Risk Assessment Technical Background Document.

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[1] Chlorinated aliphatics include any organic compound characterized by straight-chain, branch-chain, or cyclic hydrocarbons containing one to five carbons, with varying amounts of chlorine substitution. Hydrocarbons are organic compounds composed of the atoms hydrogen and carbon. Aliphatics occur where the chemical bonds between the carbon atoms are single, double, or triple covalent bonds (not aromatic bonds). When an aliphatic hydrocarbon is chlorinated, one or more of the hydrogen atoms in the “aliphatic hydrocarbon” have been replaced with chlorine atoms, at different positions and at multiple positions.

1.2 Purpose of this Risk Assessment

This risk assessment was developed to provide estimates of cancer and noncancer human health risks for individuals (receptors) who may be exposed to releases from waste management units (that is, tanks, landfills, and land treatment units) used to manage wastewaters and wastewater treatment sludges from the production of chlorinated aliphatic chemicals. The risk assessment also includes a screening analysis that provides a general indication of the potential risks to ecological receptors. The risk results will be used to help EPA decide whether to propose listing of certain wastewaters and wastewater treatment sludges from the manufacture of chlorinated aliphatic chemicals as hazardous wastes.

The risk assessment evaluates only those wastewaters and wastewater treatment sludges that currently are identified as nonhazardous.[2] As explained in greater detail in Section 2, the specific wastewaters and wastewater treatment sludges that we evaluate in this risk assessment are

- All chlorinated aliphatic wastewaters, with the exception of those generated from the production of vinyl chloride monomer using mercuric chloride catalyst in an acetylene-based process
- Sludges generated from the treatment of wastewaters from the production of ethylene dichloride/vinyl chloride monomer (EDC/VCM) via the “balanced process”
- Sludges generated from the treatment of wastewaters from the production of methyl chloride.

1.3 Overview of the Risk Assessment Methodology

EPA’s human health risk assessment provides estimates of the incremental human health risks resulting from exposure to contaminants in chlorinated aliphatics wastewaters, EDC/VCM sludges, and methyl chloride sludges. The incremental human health risks are expressed as estimates of excess lifetime cancer risk for individuals exposed to carcinogenic

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[2] Some sludges generated by this industry already are designated as hazardous because they include material derived from wastes that EPA previously listed as hazardous waste.

(cancer-causing) contaminants and hazard quotients (HQs) for contaminants that produce noncancer health effects.

The human health risk assessment that EPA conducted to support the chlorinated aliphatics listing determination included five primary tasks: (1) establishing that there are constituents in the wastes that are of concern to EPA and that warrant analysis to determine their risk to human health; (2) identifying scenarios under which contaminants are released from a waste management unit and transported in the environment to a human receptor; (3) using fate and transport models, estimating the concentrations of contaminants to which the receptor might be exposed; (4) quantifying the receptor's exposure to contaminants and the contaminant's toxicity to the receptor, and (5) describing the receptor's predicted risk.

EPA used two different approaches to estimate human health risks: "deterministic risk analysis" and "probabilistic risk analysis." A deterministic risk analysis produces a point estimate of risk or hazard for each receptor based on using a single value for each parameter in the analysis. A probabilistic risk analysis produces a distribution of risks or hazards for each receptor by allowing some of the parameters in the analysis to have more than one value. The term parameter refers to any one of a number of input variables required for the fate and transport, exposure, and risk models that EPA uses to assess risk.

EPA conducted both "central tendency" and "high end" deterministic risk assessments to attempt to quantify the risk or hazard for the "average" receptor in the population (the central-tendency risk) and the risk or hazard for individuals in small but definable "high end" segments of the population (the high end risk). For central tendency deterministic risk analyses, we set all parameters at their central tendency values. For the chlorinated aliphatics risk assessments, the central tendency values generally were either mean or 50th percentile values. For high end deterministic risk analyses, EPA sets two parameters at their high end values, and sets all other parameters at their central tendency values. For the chlorinated aliphatics risk assessments, the high end values generally represent 90th percentile values.

EPA conducted a probabilistic risk assessment to support the results of the deterministic risk analyses and to quantify individual risk at selected percentiles of the risk distribution (for example, 50th percentile, 90th percentile, 95th percentile). EPA conducted probabilistic risk analyses for all of the receptors evaluated in the deterministic risk assessment but restricted the analyses to those contaminants, pathways, and receptors for which risks or hazards exceeded a target cancer risk of $1x10^{-6}$ or, for noncarcinogens, a target hazard quotient of 1. In the probabilistic analysis, EPA developed input data that describe the range of values and the shape of the distribution for the various input parameters. Risk estimates were calculated through repetitive calculations using the fate and transport, exposure, and risk models based on the random selection of input values from the parameter distributions. The result of the probabilistic analysis was a distribution of the risks or hazards for each of the receptors.

The following sections discuss how EPA completed each of the risk assessment tasks. Specifically, Section 2 describes how EPA identified the constituents of potential concern in the wastes, how the wastes are managed, and the scenarios under which contaminants might be released from a waste and transported in the environment to a receptor. Section 3 discusses how EPA modeled contaminant fate and transport in the environment and predicted the concentrations of contaminants at the points where receptors might be exposed. Section 4 presents our methodology for assessing contaminant exposure and toxicity, and Section 5 describes our estimates of predicted risk. EPA's ecological risk screening analysis for the wastes evaluated in this document is presented in Appendix J.

2.0 ESTABLISHING CONTAMINANT EXPOSURE SCENARIOS

This section describes how EPA established the exposure scenarios that we evaluated in the risk assessment. An exposure scenario describes how an individual (a receptor) may come into contact with (be exposed to) contaminants in a waste.

To describe the exposure scenarios, this section

- Identifies the wastes that are the subject of the risk assessment
- Provides the chemical and physical characteristics of the wastes
- Describes how the wastes are managed, including the design, the operation, and the locations of the waste management units
- Identifies the potential receptors of a contaminant release from the waste management units
- Describes the mechanisms by which contaminants may be released from the waste management units and transported to potential receptors
- Identifies how intake or uptake of waste constituents occurs at the point of exposure
- Identifies whether the wastes contain constituents that are of potential concern to EPA because they could impact adversely the health of potential receptors.

The contaminant exposure scenarios form our conceptual framework for conducting the fate and transport and exposure modeling that we use to estimate the contaminant concentrations to which receptors may be exposed (that is, to quantify exposure). As discussed in Section 1, we conducted both deterministic and probabilistic risk analyses to support the chlorinated aliphatics listing determination. Many of the parameters that these analyses require are derived from the exposure scenarios. Some of the parameters are fixed or constant. For other parameters, we develop both central tendency and high end values for use in the deterministic analyses and distributions of parameter values for use in the probabilistic analyses. We generally set central tendency values at the 50th percentile value and high end values at the 90th percentile value (or at the 10th percentile when low values of a parameter generate greater risk) of their distributions. This section, in conjunction with Appendix K, identifies the fixed values, central tendency values, high end values, and parameter distributions that describe the contaminant exposure scenarios.

2.1 Waste Characterization

This section identifies and describes the wastes that are the subject of the risk analyses contained in this background document. Included in this section are the wastes' chemical and physical characteristics and the ways that the chlorinated aliphatics industry manages the wastes.

2.1.1 Waste Identification

The consent decree between EPA and EDF requires that we evaluate wastewaters and wastewater treatment sludges from the production of chlorinated aliphatic chemicals. The chlorinated aliphatics industry reports that approximately 11.5 million metric tons of chlorinated aliphatic wastewaters are generated annually (U.S.EPA 1999). The wastewaters that are the subject of the risk analysis described in this background document are nonhazardous wastewaters derived from the production of chlorinated aliphatic chemicals, excluding those wastewaters that are derived from the production of VCM using mercuric chloride catalyst in an acetylene-based process ("VCM-A," a process employed at only one chlorinated aliphatics facility). For simplicity, we call the wastewaters that are the subject of this risk analysis "chlorinated aliphatics wastewaters," although they do not include VCM-A wastewaters.

Many producers of chlorinated aliphatic chemicals manufacture several different chlorinated aliphatic products at a single facility and commingle the wastewaters generated by all processes prior to treatment in a single wastewater treatment system. Consequently, chlorinated aliphatics wastewaters may be generated from the production of one or more of the following chlorinated aliphatics chemicals:

- EDC/VCM via the balanced process
- Chlorinated methanes
- Chloroprene
- Allyl chloride
- Vinylidene chloride
- Hexachlorocyclopentadiene
- Trichloroethylene
- Tetrachloroethylene
- Carbon tetrachloride
- 1,1,1-Trichloroethane
- Methallyl chloride.

Although this list contains a number of different chlorinated aliphatic chemical products, the majority of the wastewaters within the scope of our evaluation are generated from the production of EDC/VCM. Specifically, of the nearly 10 million metric tons of chlorinated aliphatics products produced in 1996, greater than 85 percent were EDC and VCM manufactured via the "balanced process." The balanced process involves the production of EDC by direct chlorination and oxyhydrochlorination of ethylene, followed by thermal cracking to produce VCM.

We identified four categories of nonhazardous wastewater treatment sludges generated by the chlorinated aliphatics industry. These categories are based primarily upon management practices, but also are based to some extent on particular production processes. The wastewater treatment sludge categories are as follows:

- Wastewater treatment sludges generated from the production of EDC/VCM
- Wastewater treatment sludges generated from the production of methyl chloride (chloromethane)
- Wastewater treatment sludges generated from the production of VCM-A
- Wastewater treatment sludges generated from the production of allyl chloride.

The subjects of the risk analyses contained in this background document are the EDC/VCM and methyl chloride wastewater treatment sludges. The Listing Background Document for the Chlorinated Aliphatics Listing Determination (Proposed Rule) (U.S.EPA 1999) describes EPA's evaluation of VCM-A and allyl chloride sludges. EDC/VCM and methyl chloride sludges are derived from the biological, chemical, and/or physical treatment of wastewaters generated in the manufacture of chlorinated aliphatic chemicals. The methyl chloride sludges, as well as a number of the EDC/VCM sludges, are derived from wastewaters generated not only from chlorinated aliphatic chemical production processes, but from other facility processes and sources as well. EPA is calling "dedicated" those sludges that are generated solely from the treatment of wastewaters generated from the production of chlorinated aliphatic chemicals. Conversely, EPA is calling "nondedicated" those sludges that are generated from wastewaters derived both from chlorinated aliphatic production processes and from other sources.

2.1.2 Waste Characterization Strategy

EPA characterized the wastewaters and wastewater treatment sludges generated by the chlorinated aliphatics industry by

- Surveying the industry regarding their waste generation and waste management practices

- Conducting site visits to perform engineering evaluations of the processes that generate the wastestreams of concern and evaluations of the waste management practices employed by the industry

- Conducting site visits to sample the wastestreams that are the subject of the listing determination.

The characterization methods produced information pertaining to

- The sources of wastewaters and wastewater treatment sludges generated from the production of chlorinated aliphatic chemicals
- The volumes of wastewaters and wastewater treatment sludges generated
- The methods by which wastewaters and wastewater treatment sludges are managed
- The chemical composition of wastewaters and wastewater treatment sludges
- The physical characteristics of wastewaters and wastewater treatment sludges.

The results of these waste characterization efforts, their purpose, scope, and results, are described in detail in The Listing Background Document for the Chlorinated Aliphatics Listing Determination Proposed Rule (U.S. EPA 1999).

To support the chlorinated aliphatics listing determination, EPA collected and analyzed samples of wastewaters from the production of chlorinated aliphatic chemicals, wastewater treatment sludges from the production of EDC/VCM, and wastewater treatment sludges from the production of methyl chloride. We used the results of these waste analyses to establish the “constituents of potential concern” (COPCs) in the wastes (Section 2.3).

EPA collected and analyzed 41 samples of wastewaters generated from the production of chlorinated aliphatic chemicals. EPA collected six of these samples at the influent (or “headworks”) of wastewater treatment systems that manage only wastewaters derived from the production of chlorinated aliphatic chemicals. We call these samples “dedicated” chlorinated aliphatics wastewater samples,[1] and we chose to use data from these samples in assessing the risks and hazards attributable to the management of chlorinated aliphatic wastewaters. The assessment of dedicated sample data allows us to evaluate without question what risks are attributable to the wastes of concern to EPA.

EPA collected and analyzed seven samples of nonhazardous EDC/VCM sludges. (Some sludges generated by this industry already are designated as hazardous because they include material derived from wastes that EPA previously listed as hazardous waste.) Four were samples of sludges that were derived from wastewater treatment systems that manage only EDC/VCM process wastewaters. These samples are dedicated EDC/VCM sludge samples. The other three were samples of sludges that result from the treatment of EDC/VCM process wastewaters combined with wastewaters from non-EDC/VCM processes and sources. As was the case for chlorinated aliphatics wastewaters, EPA chose to use only the dedicated EDC/VCM sample data in our analysis of the risks and hazards posed by EDC/VCM wastewater treatment sludges.

The methyl chloride sludge, generated by only one facility, results from treatment of the combined wastewaters from the facility’s methyl chloride process and other processes and sources. The facility reports that approximately 18 percent of the wastewater that generates the sludge is from the methyl chloride process. The remainder of the wastewater is from other processes. Because the sludge, as generated, is not dedicated and there is no means to obtain a dedicated sample of the methyl chloride sludge, we conducted our risk assessment using the data for the nondedicated methyl chloride sludge sample.

2.1.3 Chemical and Physical Characteristics of the Wastes

Tables 2-1, 2-2, and 2-3 present the results of the analysis of the nonhazardous dedicated chlorinated aliphatics wastewater samples, the nonhazardous dedicated EDC/VCM

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[1] “Dedicated” chlorinated aliphatic wastewaters are those that comprise only chlorinated aliphatics process wastewaters, that is, wastewaters generated from the production of the chlorinated aliphatic chemicals of concern to this listing determination.

Table 2-1. Results of the Analysis of Nonhazardous Dedicated Chlorinated Aliphatics Wastewater Samples

Constituent	CAS No.		OG-01		OG-03		PL-01		PL-02		PL-03		GL-02	Central Tendency Conc.	High End Conc.
Volatile Organics - Method 8260A µg/L															
1,2-Dichloroethane	107062		82	J	2.4		6		11	<	2.5		57	26.78	82
2-Chloro-1,3-butadiene	126998	<	2.5	<	2.5		10		8		16	<	2.5	6.83	16
Acetone	67641	<	10	J	16		120	J	13		85	<	10	42.33	120
Allyl chloride	107051		17	J	2.1	<	2.5	<	2.5	<	2.5	<	2.5	4.85	17
Bromodichloromethane	75274	<	2.5	<	2.5	<	2.5	<	2.5	<	2.5	<	2.5	2.50	2.5
Bromoform	75252	J	1.6	<	2.5	<	2.5	<	2.5	<	2.5	<	2.5	2.35	2.5
Carbon disulfide	75150	J	2.2	<	2.5		12	J	3.2	<	2.5	<	2.5	4.15	12
Chlorobenzene	108907	<	2.5	<	2.5		10		8.0		7	<	2.5	5.47	10
Chloroethane	75003	<	5	<	5	<	5		16	<	5	<	5	6.83	16
Chloroform	67663		91		63		9		320		24		700	201.1	700
cis-1,2-Dichloroethylene	156592	<	2.5	<	2.5	<	2.5		7	<	2.5	<	2.5	3.27	7.1
Chlorodibromomethane	124481	J	1.3	<	2.5	<	2.5	<	2.5	<	2.5	<	2.5	2.30	2.5
Ethylbenzene	100414	<	2.5	<	2.5	<	2.5	J	2.9	J	2.8	<	2.5	2.62	2.9
Methyl ethyl ketone	78933	<	2.5	<	2.5		35	<	2.5	J	2.9	<	2.5	7.98	35
Methylene chloride	75092	<	5	<	5	J	5.3	<	5	<	5	<	5	5.05	5.3
Styrene	100425	<	2.5	<	2.5		7	<	2.5		6	<	2.5	3.92	7.3
Tetrachloroethylene	127184	<	2.5	<	2.5	<	2.5		9	<	2.5	<	2.5	3.55	8.8
trans-1,2-Dichloroethylene	156605	<	2.5	<	2.5	<	2.5	J	3.0	<	2.5	<	2.5	2.58	3
Trichloroethylene	79016	<	2.5	<	2.5	<	2.5		11	<	2.5	<	2.5	3.92	11
Semivolatile Organics - Method 8270B µg/L															
4-Aminobiphenyl	92671	<	20	<	20	<	20	<	20	<	20	J	20	20	20
Benzoic acid	65850		20		20		23		140	<	10	<	10	37.17	140
Benzyl alcohol	100516	<	5	<	5	<	5	<	5		13	<	5	6.33	13
Bis(2-chloroethyl)ether	111444	<	5		260	<	5	<	5		59	<	5	56.50	260
Bis(2-chloroisopropyl)ether	39638329	<	5	<	5		24	<	5	<	5	<	5	8.17	24
Diethyl phthalate	84662	<	5	<	5		90	<	5	<	5	<	5	19.17	90
Dimethyl phthalate	131113	<	5	<	5	<	5	J	8.7	<	5	<	5	5.62	8.7
Bis(2-ethylhexyl)phthalate	117817	<	5	<	5	<	5	<	5	J	7.4	<	5	5.40	7.4
Hexachlorobenzene	118741	<	5	<	5	<	5	J	5.0	<	5	<	5	5.00	5
2-Methylphenol	95487	<	5	<	5		14	<	5	<	5	<	5	6.50	14
4-Methylphenol	106445	<	5	<	5		24	<	5	<	5	<	5	8.17	24

(continued)

Table 2-1. (continued)

Constituent	CAS No.	OG-01	OG-03	PL-01	PL-02	PL-03	GL-02	Central Tendency Conc.	High End Conc.
Di-n-octyl phthalate	117840	< 5	< 5	< 5	< 5	J 5.7	< 5	5.12	5.7
Pentachlorophenol	87865	30	< 10	60	< 10	< 10	< 10	21.67	60
Phenol	108952	< 5	< 5	< 5	110	160	< 5	48.33	160
2,4,5-Trichlorophenol	95954	20	< 5	< 5	< 5	< 5	< 5	7.50	20
2,4,6-Trichlorophenol	88062	22	< 5	93	< 5	< 5	< 5	22.50	93
Total Metals - Methods 6010, 7470 mg/L									
Aluminum	7429905	< 0.10	0.33	11.5	5.68	1.18	44.6	10.57	44.6
Arsenic	7440382	< 0.005	0.01	0.018	< 0.005	< 0.005	0.069	0.02	0.07
Barium	7440393	< 0.1	< 0.1	< 0.10	0.31	< 0.10	< 0.1	0.14	0.31
Beryllium	7440417	< 0.0025	< 0.0025	0.006	< 0.0025	< 0.0025	< 0.0025	0.00	0.01
Calcium	7440702	81.3	10.4	10.7	82.7	40.5	14.4	40.00	82.7
Chromium	7440473	0.03	0.08	0.67	2.86	0.05	0.30	0.67	2.86
Cobalt	7440484	< 0.025	< 0.025	< 0.025	0.06	< 0.025	< 0.025	0.03	0.06
Copper	7440508	0.20	0.10	33.5	16.3	0.08	8.39	9.76	33.5
Iron	7439896	9.2	136	24.3	658	7.23	4.50	139.87	658
Lead	7439921	< 0.0015	0.02	0.010	0.12	0.003	0.006	0.03	0.12
Magnesium	7439954	8.6	< 2.5	10.7	22.9	20.1	2.46	11.21	22.9
Manganese	7439965	0.10	0.55	0.24	3.69	0.52	0.08	0.86	3.69
Mercury	7439976	< 0.00025	< 0.00025	< 0.00025	< 0.00025	0.0008	< 0.00025	0.00034	0.0008
Molybdenum	7439987	< 0.01	< 0.01	< 0.01	0.24	< 0.01	< 0.01	0.05	0.24
Nickel	7440020	0.15	0.07	10.3	40.6	0.09	0.14	8.56	40.6
Potassium	7440097	53.0	27.2	20.2	16.8	6.0	7.2	21.73	53
Sodium	7440235	7,210	2,860	26,400	181	11,200	4,750	8766.83	26400
Vanadium	7440622	< 0.025	< 0.025	< 0.025	< 0.025	< 0.025	< 0.025	0.03	0.03
Zinc	7440666	0.10	0.21	0.66	3.90	0.33	0.21	0.90	3.9
Dioxins/Furans - Method 1613 ng/L									
1,2,3,4,6,7,8-HpCDD	35822469	0.069	< 0.028	0.310	< 0.50	< 0.024	0.880	0.310	0.880
1,2,3,4,6,7,8-HpCDF	67562394	1.90	< 0.028	4.60	7.90	< 0.024	43.0	4.60	43.0
1,2,3,4,7,8,9-HpCDF	55673897	0.240	< 0.028	0.830	1.70	< 0.024	12.0	0.830	12.0
1,2,3,4,7,8-HxCDD	39227286	< 0.025	< 0.028	< 0.0225	< 0.038	< 0.024	0.052	ND	0.052
1,2,3,6,7,8-HxCDD	57653857	< 0.025	< 0.028	< 0.0225	< 0.046	< 0.024	0.091	ND	0.091
1,2,3,7,8,9-HxCDD	19408743	< 0.025	< 0.028	< 0.0225	< 0.047	< 0.024	0.110	ND	0.110
1,2,3,4,7,8-HxCDF	70648269	< 0.070	< 0.028	0.610	2.10	< 0.024	5.30	0.610	5.30

(continued)

Table 2-1. (continued)

Constituent	CAS No.	OG-01	OG-03	PL-01	PL-02	PL-03	GL-02	Central Tendency Conc.	High End Conc.
1,2,3,6,7,8-HxCDF	57117449	0.110	< 0.028	0.280	1.10	< 0.024	1.20	0.280	1.20
1,2,3,7,8,9-HxCDF	72918219	0.098	< 0.028	0.076	0.370	< 0.024	< 1.2	0.076	ND
2,3,4,6,7,8-HxCDF	60851345	0.100	< 0.028	0.120	0.630	< 0.024	0.430	0.120	0.430
2,3,4,7,8-PeCDF	57117314	< 0.025	< 0.028	< 0.0225	0.230	< 0.024	0.210	ND	0.210
2,3,7,8-TCDD	41903575	< 0.005	< 0.007	< 0.0045	< 0.005	< 0.005	0.017	ND	0.017
2,3,7,8-TCDF	51207319	< 0.005	< 0.0165	< 0.0045	0.021	< 0.005	0.082	ND	0.082
OCDD	3268879	0.600	0.19	6.50	4.90	< 0.048	6.90	6.50	6.90
OCDF	39001020	4.60	0.75	140	24.0	0.110	6,000	140	6,000
TCDD - TEQ		0.091	0.044	0.313	0.662	0.0352	7.414	0.313	7.414
Total HpCDD	37871004	0.069	< 0.028	0.510	0.590	< 0.024	1.30	NAP	NAP
Total HpCDF	38998753	3.00	0.85	7.00	9.60	< 0.024	60.0	NAP	NAP
Total HxCDD	34465468	< 0.025	< 0.028	< 0.0225	< 0.047	< 0.024	0.510	NAP	NAP
Total HxCDF	55684941	1.20	0.44	1.70	9.30	< 0.024	9.30	NAP	NAP
Total PeCDF	30402154	0.300	0.15	< 0.0225	2.70	< 0.024	0.440	NAP	NAP
Total TCDD	41903575	< 0.005	< 0.007	< 0.0045	< 0.005	< 0.005	0.049	NAP	NAP
Total TCDF	55722275	0.049	< 0.0165	< 0.0045	0.970	< 0.005	0.860	NAP	NAP
General Chemistry mg/L									
TDS	NA	18,400	6,420	NA	NA	NA	NA	12410	18400
TSS	NA	48	280	1,440	< 10	< 10	308	349.33	1440
TOC	NA	790	34	1,570	85	19	491	498.17	1570
Oil & Grease	NA	NA	NA	< 1	< 1	< 1	< 1	1	1

Note: Central tendency concentration is the average concentration and the high-end concentration is the maximum detected value except for dioxins. Samples PL-01 and GL-02 represent central tendency and high-end concentrations, respectively, for dioxins.

< = Reported value is half the laboratory reporting limit.

NA = Not available.

NAP = Not applicable (toxicity equivalency factor is zero).

ND = Not detected.

J = Estimated value.

Table 2-2. Results of the Analysis of Nonhazardous Dedicated EDC/VCM Sludge Samples

Constituent	CAS No.		OG-04		OG-06		OC-02		GL-01	Central Tendency Conc.	High End Conc.
Volatile Organics - Method 8260A µg/kg											
1,2-Dichloroethane	107062		9	J	2.7	<	13		530	138.7	530
2-Hexanone	591786	J	2.5	<	2.5	<	13*	<	12*	2.5	2.5
Acetone	67641		2,000	<	10	<	50		360	605	2,000
Allyl chloride	107051		8	J	3.5	<	13*	<	12*	5.8	8
Carbon disulfide	75150	<	2.5	<	2.5	<	13		34	13.0	34
Chloroform	67663	J	2.5	J	3.5	<	13		560	144.8	560
Methyl ethyl ketone	78933		120	<	2.5	<	13	<	12	36.9	120
Methylene chloride	75092	<	5.0	<	5	<	25		43	19.5	43
Tetrachloroethylene	127184	<	2.5	<	2.5	<	13	J	18	9.0	18
Trichloroethylene	79016	J	2.8	<	2.5	<	13*	<	12*	2.7	2.8
Vinyl acetate	108054	J	5		7	<	13*	<	12*	5.9	7
Vinyl chloride	75014	<	5.0	<	5	<	25*	J	15	8.3	15
TCLP Volatile Organics - Methods 1311 and 8260A µg/L											
1,2-Dichloroethane	107062	<	2.5	J	2.6	J	4.8		36	11.5	36
4-Methyl-2-pentanone	108101	<	2.5	<	2.5	JB	3.6	JB	3.7	3.1	3.7
Acetone	67641	B	670	B	330	B	23	B	91	278.5	670
Carbon disulfide	75150	<	2.5	<	2.5	<	2.5		7.2	3.7	7.2
Chloroform	67663	<	2.5	<	2.5	<	2.5		32	9.9	32
cis-1,3-Dichloropropene	10061015	J	3.8	<	2.5	<	2.5	<	2.5	2.8	3.8
Methyl ethyl ketone	78933		28	<	2.5	<	2.5		6.8	10.0	28
Methylene chloride	75092		44		23	JB	7.8	JB	9.5	21.1	44
Semivolatile Organics - Method 8270B µg/kg											
Benzoic acid	65850	J	190	<	650*	<	650*	<	6500*	190	190
Bis(2-chloroethyl)ether	111444	<	330		800	<	330	<	3300*	487	800
Bis(2-ethylhexyl)phthalate	117817	J	140		1,870	J	1,200	J	5,900	2,278	5,900
Hexachlorobenzene	118741	J	110	<	325*	<	330*	<	3300*	110	110

(continued)

Table 2-2. (continued)

Constituent	CAS No.	OG-04	OG-06	OC-02	GL-01	Central Tendency Conc.	High End Conc.
TCLP Semivolatile Organics - Methods 1311 and 8270B µg/L							
Benzoic acid	65850	108	< 10	40	38	49.0	108
Bis(2-chloroethyl)ether	111444	< 5	12	< 5	< 5	6.8	12
4-Methylphenol	106445	< 5	< 5	< 5	42	14.3	42
Total Metals - Methods 6010, 7471 mg/kg							
Aluminum	7429905	291	209	579	29,500	7,645	29,500
Arsenic	7440382	5.81	7.1	< 0.5	27	10.0	27
Barium	7440393	< 10	43	98	68	54.7	98
Cadmium	7440439	< 0.25	0.6	< 0.2	< 0.2	0.3	0.63
Calcium	7440702	214,000	13200	17300	4,380	62,220	214,000
Chromium	7440473	12.2	70	25	287	98.5	287
Cobalt	7440484	< 2.5	10	< 2	< 2	4.3	10.4
Copper	7440508	54.5	141	129	4,080	1,101	4,080
Iron	7439896	6,940	158000	40200	8,390	53,383	158,000
Lead	7439921	1.6	13	2	3.6	5.0	13.0
Magnesium	7439954	< 250	2730	4040	1,080	2,024.9	4,040
Manganese	7439965	133	663	324	75	298.7	663
Molybdenum	7439987	< 1	< 1	< 1	2.8	1.4	2.8
Nickel	7440020	31.5	80	34	120	66.3	120
Potassium	7440097	< 250	< 250	< 250	< 250	ND	ND
Sodium	7440235	2,740	2830	9460	2,160	4,297.5	9,460
Vanadium	7440622	14.6	9	< 2	< 2.0	7.1	15
Zinc	7440666	55.6	688	89	149	245.4	688
TCLP Metals - Methods 1311, 6010, and 7470 mg/L							
Arsenic	7440382	0.014	0.005	< 0.005	0.053	0.0193	0.053
Calcium	7440702	848	588	413	204	513.3	848
Cobalt	7440484	< 0.03	0.07	< 0.03	< 0.03	0.04	0.07
Copper	7440508	0.43	< 0.13	< 0.13	22.3	5.7	22.3
Magnesium	7439954	3.2	136	154	21.5	78.7	154
Manganese	7439965	1.7	12.9	0.81	2.0	4.4	12.9

(continued)

Table 2-2. (continued)

Constituent	CAS No.	OG-04	OG-06	OC-02	GL-01	Central Tendency Conc.	High End Conc.
Molybdenum	7439987	< 0.10	0.22	< 0.1	< 0.1	0.1	0.22
Nickel	7440020	0.34	0.67	< 0.1	1.3	0.6	1.3
Potassium	7440097	9.3	5.2	4.1	3.6	5.6	9.3
Zinc	7440666	< 1.0	4.0	< 1.0	< 1.0	1.8	4.0
Dioxins/Furans - Method 1613 ng/kg							
2,3,7,8-TCDF	51207319	1.1	< 3.0	7.9	145	1.1	145
2,3,7,8-TCDD	1746016	< 0.30	< 0.6	< 0.3	39	ND	39
1,2,3,7,8-PeCDF	57117416	8.4	21.0	27.5	< 0.6	8.4	ND
2,3,4,7,8-PeCDF	57117314	10.8	22.5	12.4	127	10.8	127
1,2,3,7,8-PeCDD	40321764	< 1.4	< 2.8	< 0.9	< 40	ND	ND
1,2,3,4,7,8-HxCDF	67562394	108	107	65	1,425	108	1,425
1,2,3,6,7,8-HxCDF	57117449	84	< 16.0	13.8	< 300	84	ND
2,3,4,6,7,8-HxCDF	60851345	72	32.8	7.2	648	72	648
1,2,3,7,8,9-HxCDF	72918219	38.9	< 40	15.5	< 140	38.9	ND
1,2,3,4,7,8-HxCDD	39227286	8.4	< 2.8	< 0.9	< 20.0	8.4	ND
1,2,3,6,7,8-HxCDD	57653857	7.8	< 2.8	< 0.9	83	7.8	83
1,2,3,7,8,9-HxCDD	19408743	5.6	< 2.8	< 0.9	62	5.6	62
1,2,3,4,6,7,8-HpCDF	67562394	2,100	46	38	20,700	2,100	20,700
1,2,3,4,7,8,9-HpCDF	55673897	413	50	24.4	13,500	413	13,500
1,2,3,4,6,7,8-HpCDD	35822469	234	14.5	3.2	777	234	777
OCDF	39001020	10,800	648	62	212,000	10,800	212,000
OCDD	3268879	2,220	297	41	6,480	2,220	6,480
TCDD - TEQ		78.9	28.3	19.3	907	78.9	907
TCLP Dioxins/Furans - Methods 1311, 1613 ng/L							
Total TCDF	55722275	0.015	< 0.006	< 0.005	0.049	0.0	0.049
Total HxCDF	55684941	< 0.027	< 0.031	< 0.026	0.070	0.0	0.070
1,2,3,4,6,7,8-HpCDF	67562394	0.083	< 0.031	< 0.026	1.10	0.3	1.10
1,2,3,4,7,8,9-HpCDF	55673897	< 0.027	< 0.031	< 0.026	0.40	0.1	0.400
Total HpCDF	38998753	0.083	< 0.031	< 0.026	2.20	0.6	2.20
OCDF	39001020	0.50	< 0.06	< 0.05	99.0	24.9	99.0

(continued)

Table 2-2. (continued)

Constituent	CAS No.	OG-04	OG-06	OC-02	GL-01	Central Tendency Conc.	High End Conc.
OCDD	3268879	< 0.055	< 0.06	< 0.05	0.20	0.1	0.200
General Chemistry mg/kg							
TOC	NA	NA	NA	3,700	67,900	35,800	67,900
Oil & Grease	NA	NA	NA	680	974	827	974

Note: Central tendency concentration is the average concentration and the high-end concentration is the maximum detected value except for dioxins.

Samples OG-04 and GL-01 represent central tendency and high-end concentrations, respectively for dioxins. All concentrations are based on wet-weights.

* Non-Detect values greater than the highest detected concentration have been excluded from the calculations.

< = Non-Detect values are reported as ½ the method detection limit.

J = Estimated value.

B = Constituent detected in laboratory blank.

ND = Not detected.

Table 2-3. Results of the Analysis of the Methyl Chloride Sludge

Constituent	CAS No.	DC-01
Volatile Organics - Method 8260A µg/kg		
Acetone	67641	2200
Methylene chloride	75092	12000
TCLP Volatile Organics - Methods 1311 and 8260A µg/L		
Acetone	67641	150
Carbon disulfide	75150	6
Methylene chloride	75092	J 9.1
Semivolatile Organics - Method 8270B µg/kg		
None detected		ND
TCLP Semivolatile Organics - Methods 1311 and 8270B µg/L		
Benzoic acid	65850	J 13
Total Metals - Methods 6010, 7471 mg/kg		
Aluminum	7429905	1930
Arsenic	7440382	1.9
Calcium	7440702	77200
Chromium	7440473	7
Copper	7440508	643
Iron	7439896	5680
Lead	7439921	7
Magnesium	7439954	23300
Manganese	7439965	109
Nickel	7440020	9.1
Zinc	7440666	574
TCLP Metals - Methods 1311, 6010, and 7470 mg/L		
Aluminum	7429905	2.4
Arsenic	7440322	0.002
Calcium	7440702	1470
Copper	7440508	5.3
Magnesium	7439954	81
Manganese	7439965	4.1
Dioxins/Furans - Method 1613 ng/kg		
1,2,3,4,6,7,8-HpCDF	67562394	3.1
1,2,3,4,6,7,8-HpCDD	35822469	7
OCDF	39001020	9.6
OCDD	3268879	44
TCDD - TEQ		0.155
TCLP Dioxins/Furans - Methods 1311, 1613 ng/L		
None detected		ND

(continued)

Table 2-3. (continued)

Constituent	CAS No.	DC-01
General Chemistry mg/kg		
TOC	NA	42100
Oil & Grease	NA	65400
BTU	NA	3199
Percent Solids	NA	53.6

All concentrations are reported on a wet weight basis.
J = Estimated value.
NA = Not available.
ND = Not detected.

sludge samples, and the methyl chloride sludge samples. EPA analyzed the samples for 44 volatile organic compounds (VOCs), 68 semivolatile organic compounds (SVOCs), 24 metals and other inorganics, 17 dioxin and furan congeners[2], and several general chemical and physical properties (for example, total organic carbon, total suspended solids, total oil and grease). For sludge samples, EPA performed both total analyses and toxicity characteristic leaching procedure (TCLP) analyses. Detailed Quality Assurance Project Plans for the waste sampling effort, Quality Assurance/Quality Control (QA/QC) procedures, and analytical data reports for the analysis effort are provided in the Listing Background Document for Chlorinated Aliphatics Listing Determination (Proposed Rule) (U.S. EPA 1999).

The analytical results for wastewater treatment sludges provided in Tables 2-2 and 2-3 are reported on a "wet weight basis." The "wet weight concentrations" reflect the concentrations of constituents in the sludges "as sampled." The laboratory also reports results on a "dry weight basis," which reflects what the constituent concentrations in the samples would be if all the moisture were removed from the samples, assuming the moisture contains none of the sample constituents (that is, assuming the moisture in the samples is pure water). The dry weight concentrations are reported in Appendix I. The wet weight concentrations most accurately reflect the concentrations of the constituents in the sludges because the sludges are not subjected to further drying after the point in the facilities' processes at which samples were

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[2] EPA classifies the furan congeners as "dioxin-like compounds" because of their structural similarity to the dioxins (U.S. EPA 1994a). In this document we will use the term "dioxin" to represent both the dioxin and furan congeners. EPA also refers to dioxin-like polychlorinated biphenyls (PCBs) as dioxin-like compounds. PCBs were not evaluated in chlorinated alphatics wastes because they are not expected to occur in the wastes.

collected, that is, the sludges are generated and disposed in the “wet” form in which we sampled them (the sludges contain from 41 to 74 percent moisture).

Some of the data presented in Tables 2-1, 2-2, and 2-3 are designated with “qualifiers.” The following is an explanation of the data qualifiers:

- ?<” – The symbol ?<” means that the constituent was not detected at the quantitation limit reported. The value reported in the table is one half of the quantitation limit of the constituent in the sample.

- ?J” – The letter ?J” indicates that the constituent was detected below the quantitation limit and that the reported value is estimated.

- ?B” – The letter ?B” indicates that the constituent was detected in a laboratory method blank.

Tables 2-1, 2-2, and 2-3 include the values that we designated as representing the “central tendency” constituent concentrations and the “high end” constituent concentrations. For all constituents except the dioxins, the central tendency concentration is the mean of the reported values. In calculating the mean, we averaged the constituents reported at concentrations less than the quantitation limit (?<” -values) as one half of the reported quantitation limit. If the quantitation limit for a constituent was unusually high, such that a value of one-half of the quantitation limit was higher than the maximum detected concentration of that constituent, then the sample with the high quantitation limit was not included in calculating the mean concentration for that constituent. A notation is included in the tables in cases where one-half of the quantitation limit exceeds the maximum detected concentration. The high end values are the maximum of the detected concentrations.

For dioxins, we did not calculate the average concentration of each of the congeners or pick the maximum congener concentration reported across all of the samples. Instead, we selected specific samples, and their corresponding dioxin congener concentrations, to represent the central tendency and high end congener concentrations. Because we ultimately evaluate the risk attributable to the dioxin congeners as a group or class, rather than individually, we believe that combining congener concentrations across samples, such as when creating average or maximum values, would result in a distribution of congener concentrations that is unrealistically high. For example, selecting the maximum of each of the detected congener concentrations would create a distribution of congener concentrations that is greater than the distribution of congener concentrations reported in any one sample. Consequently, we selected the high end and central tendency samples based on their total 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) toxicity equivalent (TEQ), calculated assuming that non-detect values are zero. (Section 4.2.3 describes how we calculate TEQs for dioxin.) The high end sample was that sample that had the highest total TCDD TEQ. For the wastewaters, the high end sample for TCDD TEQ concentration was GL-02. For the EDC/VCM sludges, the high end sample was GL-01. For the wastewaters, we selected sample PL-01 as the central tendency sample. This sample had the third highest TCDD TEQ concentration out of six samples. For EDC/VCM sludges, we designated sample OG-04 as the central tendency sample. This sample had the second highest TCDD TEQ concentration of the four samples.

2.1.4 Waste Management

Conducting the risk assessments required that we establish how facilities manage their chlorinated aliphatics wastewaters, EDC/VCM sludges, and methyl chloride sludges. The specific types of information required for our analyses included; type, design and operation of the waste management units; the volumes or quantities of waste managed; and the environmental setting where the wastes are managed. We conduct contaminant fate, transport, and exposure modeling scenarios to estimate the concentrations of contaminants to which receptors are exposed. The parameters that define the waste management scenarios are important inputs into the contaminant fate, transport, and exposure models. (See Section 3 for a description of how we conduct fate and transport and exposure modeling.) We obtained much of the necessary model input information from the industry survey responses; however public data sources contained some of the information at various levels of specificity, and we had to develop some of the information using engineering judgement.

The following sections describe how we ultimately defined the management of the wastewaters and wastewater treatment sludges for the purpose of conducting our risk analyses. The specific parameters that define the waste management scenarios and that are required in our analyses are presented (with their data sources) in Appendix K.

Facilities were not required to report to us information that describes the environmental setting (for example, geology, hydrogeology, climate) of those locations where their wastes are managed. For parameters that describe environmental setting, EPA uses databases that specify regional characteristics based on the waste management unit location. For example, EPA considers historical climate data available for all U.S. National Weather Service (NWS) stations to establish the climate at each unit location. Based on proximity, climate, and terrain, we select the most representative meteorological station for each waste management site. We then use data from the representative stations to characterize conditions at each management site. Using this approach, we assume that the meteorological characteristics of a given waste management unit location are the same as those of the representative meteorological station. Similarly, EPA uses regional data to establish the environmental characteristics of the surface and subsurface environment, such as soil type, aquifer type, and depth to groundwater. Consequently, in specifying the location of a waste management unit, EPA by default specifies the values or range of values of a number of other environmental parameters.

2.1.4.1 Chlorinated Aliphatics Wastewaters

The responses to the §3007 Survey (U.S.EPA 1999) indicated that nonhazardous chlorinated aliphatic wastewaters currently are managed as follows:

- Most chlorinated aliphatic manufacturers treat wastewaters in onsite, tank-based wastewater treatment systems and directly discharge the waters in accordance with facility-specific National Pollutant Discharge Elimination System (NPDES) permits.

- Some facilities discharge wastewater off site to either publicly- or privately-owned treatment works (POTW or PrOTWs).

- One facility treats its wastewater and then disposes of it in an underground injection well.

- None of the facilities indicate that their wastewaters are managed in surface impoundments.

The majority of the chlorinated aliphatics industry wastewaters are treated in tank-based systems prior to being discharged to an NPDES permitted outfall, POTW, or underground injection well (one facility). Based on survey responses and follow-up discussions with the facilities, many of these treatment systems incorporate biological treatment. Consequently, for the purpose of conducting our risk analysis, we assumed that chlorinated aliphatics wastewaters are managed in aerated, biological treatment tanks because this represents a reasonable management scenario for the wastewaters.

Conducting the risk analysis required that we establish the quantities of wastewaters potentially managed in wastewater treatment tanks. Because we used analytical data for dedicated chlorinated aliphatics wastewater samples in our analysis, we also used dedicated chlorinated aliphatic wastewater quantities in our analysis. We identified eight wastewater quantities (expressed as wastewater generation rates) that represent of the quantities of dedicated chlorinated aliphatics wastewaters discharged to the headworks of chlorinated aliphatics facility wastewater treatment systems. Table 2-4 presents these quantities. In Our probabilistic analysis we evaluated all of the quantities presented in Table 2-4. For our deterministic analysis we set the high end quantity as the maximum quantity and the central tendency quantity as the average quantity.

Table 2-4. Dedicated Chlorinated Aliphatics Wastewater Quantities

Facility	**Dedicated Chlorinated Aliphatics Wastewater Quantity, Mtons/year**
Geon, LaPorte, TX	962,950
PPG, Lake Charles, LA	324,500
PPG, Lake Charles, LA	173,600
PPG, Lake Charles, LA	127,250
Occidental/Oxymar, Gregory, TX	417,000
Occidental/Oxymar, Gregory, TX	157,500
Westlake Monomers, Calvert City, KY	98,000
Dupont/Dow, LaPlace, LA	314,770
Central Tendency Value (average)	**321,946**
High End Value (maximum	**962,950**

Our probabilistic risk analysis included evaluation of all facility locations that manage nonhazardous chlorinated aliphatics wastewaters. To conduct the deterministic risk analysis we had to select specific high end and central tendency facility locations. We selected the central tendency and high end locations based on meteorological parameters (that is, rather than soil parameters) because they would most influence the wastewater treatment tank risk estimates. Table 2-5 lists the facilities and locations where chlorinated aliphatics wastewaters are managed, and their representative meteorological stations. Two of the facilities that generate chlorinated aliphatics wastewaters actually are collocated in a single facility that is jointly operated by two companies, Occidental Chemical Company and Oxymar, located in

Table 2-5. Locations of Facilities that Generate Chlorinated Aliphatics Wastewaters and their Corresponding Meteorological Station Locations

Facility and Location	Meteorological Station
GE, Waterford, NY	Albany, NY
FMC, Baltimore, MD	Baltimore, MD
Formosa, Baton Rouge, LA Occidental, Convent, LA Borden, Geismar, LA Vulcan, Geismar, LA Dow, Plaquemine, LA Georgia Gulf, Plaquemine, LA	Baton Rouge, LA
Westlake, Calvert City, KY	Evansville, IN
Dow Corning, Midland, MI	Flint, MI
Occidental, Deer Park, TX Dow, Freeport, TX Occidental (Oxymar), Gregory, TX Geon, LaPorte, TX Formosa, Point Comfort, TX	Houston, TX
PPG, Lake Charles, LA Condea Vista, Westlake, LA	Lake Charles, LA
Dow Corning, Carrolton, KY DuPont/Dow, Louisville, KY	Louisville, KY
Velsicol, Memphis, TN	Memphis, TN
DuPont/Dow, LaPlace, LA Shell, Norco, LA	New Orleans, LA
Vulcan, Wichita, KS	Wichita, KS

Gregory, TX. Figure 2-1 depicts the facility locations. Although the Corpus Christi, TX, and Victoria, TX, meteorological stations actually are closer to the Gregory, TX, and Point Comfort, TX, locations, respectively, Houston was selected as the representative meteorological station for these sites because the data needed for the air dispersion model were more readily available and the data set was more complete. Selected meteorological data from the Corpus Christi and Victoria stations are used, as described in Section 2.1.4.2, to select modeling locations for EDC/VCM sludges managed in landfills.

We performed an analysis of air dispersion to identify meteorological locations to serve as the central tendency and high end locations for the risk assessment (Section 3.3 describes how we evaluate air dispersion). Appendix D.3 provides detailed results of this analysis. In conducting the dispersion analysis, we considered how both the vapor air concentrations and contaminant wet deposition rates influence risk. Based on the analysis, we identified three meteorological locations for inclusion in the deterministic risk analysis:

- Baltimore to represent the high end meteorological station location for vapor air concentration;
- Baton Rouge to represent the high end meteorological station location for wet deposition of vapor; and
- Memphis to represent the central tendency meteorological station location for both vapor air concentration and wet deposition of vapor.

Ideally, only two locations would have been selected, one central tendency and one high end. The reason why we identified two high end locations is because there was no single location that produced high end results for both vapor concentration and wet deposition of vapors. Given that the wastewaters evaluated contain a range of chemicals, we determined that we should evaluate both high end sites. By including the extra location, we minimized the likelihood of underestimating risks due to inhalation (driven by air concentration) or risks due to ingestion of soil and contaminated food products (driven by air concentration and/or wet deposition, depending on the chemical under consideration).

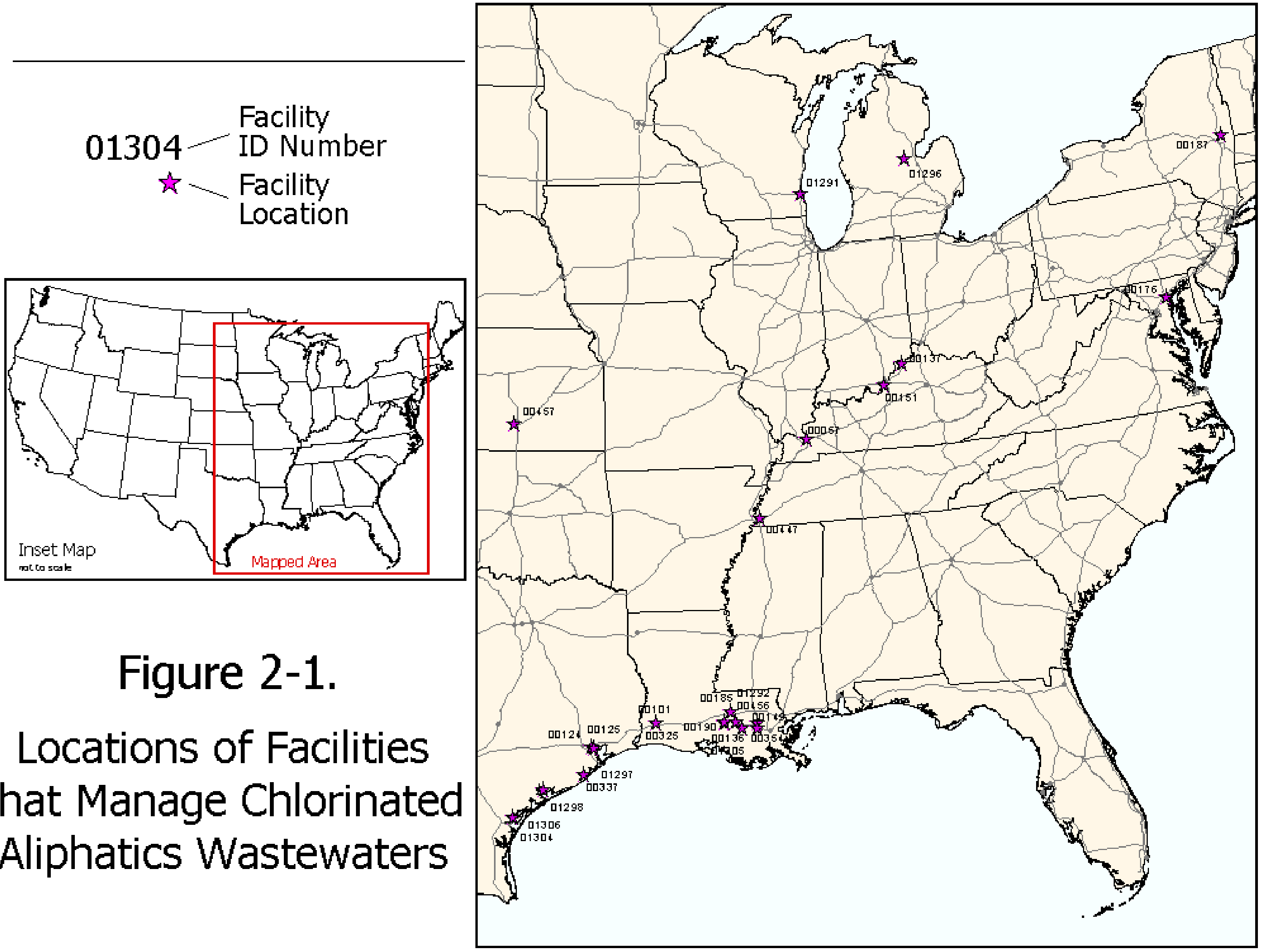

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Figure 2-1.

Locations of Facilities that Manage Chlorinated Aliphatics Wastewaters

The tank characterization data required for our analysis include both tank dimensions and tank operating parameters. In the absence of site-specific data, we assumed that the liquid depth in the tanks in which the wastewaters are treated is 15 feet (ft) and that the tanks have an additional 2.5 ft of freeboard. We assumed that the tanks are located partially in-ground, such that the water column is half above and half below ground (resulting in an above-ground tank height of 10 ft). One facility that reported managing chlorinated aliphatics wastewaters in a biological treatment tank reported that the retention time of wastewater in the tank is two days (U.S.EPA 1999). In the absence of other data, we developed tank dimensions based on facility-reported wastewater generation rates (discussed above), an assumed wastewater depth in the tank of 15 ft, and a retention time of 2 days. We selected operating parameters (such as the number of aerators, the aerator impeller diameter and speed, and the biomass concentration in the tank) that we believe represent typical operating conditions of an aerated biological tank. We assumed that the chlorinated aliphatics industry's tanks retain sufficient structural integrity to prevent wastewater releases to the subsurface (and therefore groundwater) and that overflow and spill controls prevent wastewater released to the ground surface.

2.1.4.2 EDC/VCM Sludges

The responses to the §3007 Survey (U.S.EPA 1999) indicated that nonhazardous EDC/VCM wastewater treatment sludges currently are managed in the following:

- Offsite non-hazardous waste landfills;
- Onsite non-hazardous waste landfills;
- An onsite hazardous waste landfill (this management method is practiced only at one facility); and
- An onsite nonhazardous land treatment unit (this management method is practiced only at one facility).

We assumed that management of nonhazardous chlorinated aliphatics wastewater treatment sludges would occur in offsite municipal waste landfills and, in one location, an onsite land treatment unit. EPA did not evaluate an onsite hazardous waste landfill scenario because evaluating a less protective management scenario that is practiced by the majority of the industry (a nonhazardous landfill) is sufficient for determining if managing wastewater treatment sludges as nonhazardous results in unacceptable human health risks. EPA did not evaluate the risks associated with management of wastewater treatment sludges in onsite nonhazardous waste landfills because we assume our offsite municipal landfill scenario is reasonably similar to an onsite nonhazardous waste landfill scenario and because our municipal landfill database provided information on distances to receptors, etc., needed for our analysis. Based on our site visits, we believe that onsite landfills are not likely to be closer to drinking water wells than municipal landfills.

EDC/VCM Sludges in Municipal Landfill

We assumed that EDC/VCM sludges are disposed in offsite unlined municipal landfills. Based on municipal solid waste management requirements (40 CFR Part 258), we assumed that landfills have runon/runoff controls to prevent releases through runoff and erosion. We assumed that the landfills are covered daily (every 12 hours) with soil, and are capped at the end of their active life, which is 30 years (30 years is the average active lifetime of municipal Subtitle D landfills based on a survey conducted by EPA [DPRA, 1993]). We assumed that the landfills continue to release contaminants into the environment for 40 years after they are closed.

EPA obtained the quantities of EDC/VCM sludges that are managed in landfills from the industry survey responses. Because we used analytical data for dedicated EDC/VCM sludge samples in our analysis, we also used “dedicated” EDC/VCM sludge quantities in our analysis to avoid overestimating the contaminant mass that chlorinated aliphatics facilities dispose in landfills. In cases where a facility’s wastewater treatment sludge is generated from other processes in addition to EDC/VCM, we calculated dedicated EDC/VCM sludge quantities from the volume of EDC/VCM wastewater that the facility reported in their survey response. Specifically, EPA divided the volume of wastewater attributable to EDC/VCM processes by the total volume of wastewater influent, and applied the resultant ratio to the total sludge quantities to obtain the quantity of wastewater treatment sludge attributable to EDC/VCM processes. Two facilities indicated in their survey responses that they send their EDC/VCM sludges to the same landfill. We added the quantities of sludges from these facilities for the purpose of conducting our analyses. In our probabilistic analysis we evaluated all of the dedicated sludge quantities.

For our deterministic analysis we set the high end quantity as the maximum quantity and the central tendency quantity as the average quantity.

The Listing Background Document for the Chlorinated Aliphatics Listing Determination (Proposed Rule) (U.S.EPA 1999) provides the actual locations of the offsite landfills in which EDC/VCM sludges are disposed. For the purposes of conducting this risk assessment, EPA assumed that the offsite landfills are located in the geographic vicinity of the facilities generating the wastewater treatment sludges. That is, that the locations of the municipal landfills are the same as the EDC/VCM facility locations. Table 2-6 provides the locations of the chlorinated aliphatics facilities that manage EDC/VCM sludges in landfills (the assumed locations of the offsite municipal landfills), and the quantities of dedicated EDC/VCM sludge we assumed that the facilities generate. Figure 2-2 depicts the locations of facilities that manage their EDC/VCM sludges in landfills.

Table 2-6. Locations of Facilities that Manage EDC/VCM Sludges in Landfills and the Quantities of EDC/VCM Sludge They Manage

Facility	**Facility Location**	**Sludge Quantity (Metric tons/year)**			**Comments**
		Total	**Chlorinated Aliphatics**	**EDC/ VCM**	
Geon Company	LaPorte, TX	1804	1804	1804	
Occidental (Oxymar)	Gregory, TX	820	820	820	Survey response indicates that these quantities are co-disposed
Formosa	Point Comfort, TX	3688	284*	284*	
Occidental	Gregory, TX	160	160	160	
Occidental	Convent, LA	500	500	500	
Formosa	Baton Rouge, LA	700	107*	107*	
PPG	Lake Charles, LA	2200	2200	581*	Survey response indicates that these quantities are co-disposed
Borden Chemicals	Geismar, LA	2904	311*	311*	
Dow Chemical (2 wastestreams, one biological and one nonbiological))	Freeport, TX	72223	930*	115*	
		5627	756*	101*	
Dow Chemical	Plaquemine, LA	11100	96*	96*	
Central Tendency Value (average) (assuming co-disposal of waste quantities, as indicated)				**542**	
High End Value (maximum)				**1804**	

* estimated based on wastewater quantities

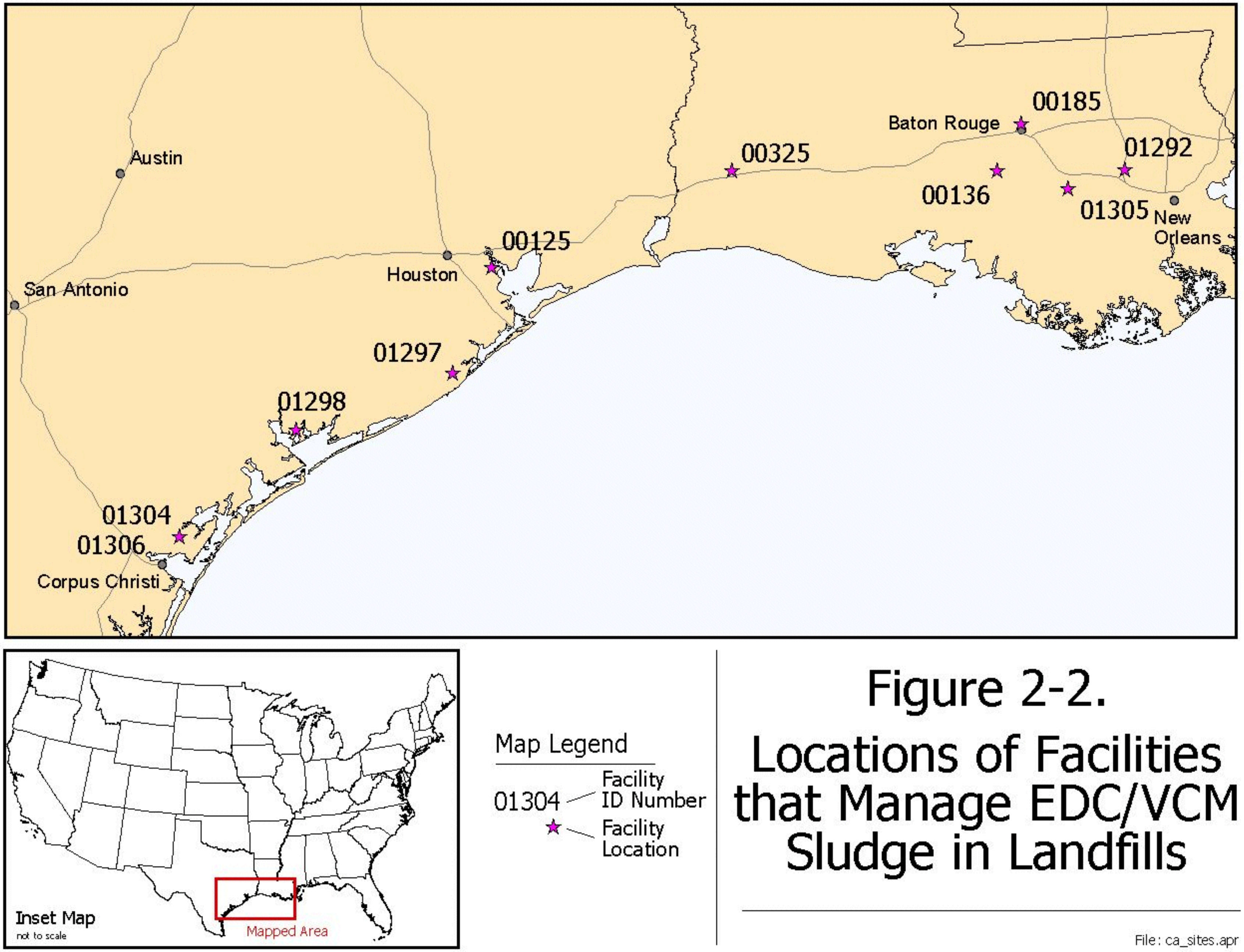

Figure 2-2.

Locations of Facilities that Manage EDC/VCM Sludge in Landfills

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Although the probabilistic risk analysis allows us to evaluate all of the landfill locations, in order to conduct the deterministic risk analysis we had to select a central tendency landfill location and a high end landfill location. Central tendency and high end values generally are 50th and 90th percentile values, respectively, but it is difficult to identify true central tendency and high end facility locations because there are numerous parameters associated with each location. Most locations are likely to have a mix of high end, central tendency, and low end characteristics; however, it is important to select specific locations for analysis, rather than selecting high end parameters from all the locations (thus creating a hypothetical location). In the latter case, it would be possible to create scenarios that actually do not exist at any location. In many cases, conditions that favor high end exposure for one pathway may have the opposite effect on another pathway. For example, locations with high precipitation may produce high end results for leaching and overland flow pathways, but will tend to produce lower end results for air transport pathways. Other conditions (for example, soil type, geology, topography, depth to groundwater) also affect fate and transport of contaminants.

For this analysis, we concluded that precipitation and infiltration are the parameters most likely to influence contaminant concentrations in groundwater, which is the pathway of greatest concern for landfills. Therefore, we based our selection of a high end location primarily on these two parameters. In order to select facility locations for modeling, we arrayed the values for several meteorological parameters corresponding to each of the facilities and sorted the data by precipitation ([note that the facility in Gregory, Texas is listed twice because this facility is a joint venture between two companies] Table 2-7). Windspeed was not highly variable among the facilities. We did not consider it an important variable for selecting facility locations because the air dispersion model is not highly sensitive to small changes in windspeed.

We selected Baton Rouge and Houston to represent high end and central tendency locations, respectively. Baton Rouge is appropriate as a representative high end facility location based on relatively high rainfall, infiltration, runoff, and erosion. Rainfall, runoff, infiltration, and erosivity values for Houston were at or near central tendency values (mean or median) reported for all facility locations. Therefore, we considered Houston an appropriate central tendency location.

EPA did not collect or have access to data that characterize the design and operation of the specific landfills where chlorinated aliphatics facilities dispose of their EDC/VCM sludges.

Table 2-7. Meteorological Data used to Select EDC/VCM Landfill Locations for Deterministic Analyses

Met Station	Site		Facility Number	Precipitation (cm/yr)	Evapotranspiration (cm/yr)	Runoff (cm/yr)	Infiltration (cm/yr)[a]	Temperature (K)	Windspeed (m/s)	Erosivity (1/yr)
Baton Rouge	Geismar	LA	01292	146.1	73.7	23.6	49	293	4.1	520
Baton Rouge	Plaquemine	LA	00136	146.1	73.7	23.6	49	293	4.1	520
Baton Rouge	Baton Rouge	LA	00185	146.1	73.7	23.6	49	293	4.1	520
Baton Rouge	Convent	LA	01305	146.1	73.7	23.6	49	293	4.1	520
Lake Charles	Lake Charles	LA	00325	140.5	74.6	19.1	47	293	4.6	57
Houston	Freeport	TX	01297	119.1	65.7	13.7	40	294	4.1	414
Houston	La Porte	TX	00125	119.1	65.7	13.7	40	294	4.1	414
Victoria	Point Comfort	TX	01298	91.2	55.2	5.6	30	295	5.1	286
Corpus Christi	Gregory	TX	01304	74.2	48.2	1.3	25	295	6.2	271
Corpus Christi	Gregory	TX	01306	74.2	48.2	1.3	25	295	6.2	271
			Mean	120.3	65.2	14.9	40	293.8	4.7	379

a For the purposes of selecting locations, infiltration rates were calculated as precipitation minus evapotranspiration and runoff as reported by the meteorological stations. Actual infiltration rates used in the groundwater models were calculated using the Hydrologic Evaluation of Landfill Performance (HELP) model and may be different from those reported here.

Consequently, we used national databases to establish the design and operating parameters for the municipal landfills. We based our assumptions regarding landfill area on the distribution of areas of municipal Subtitle D landfills reported by respondents to a 1986 survey conducted by EPA's Office of Solid Waste (DPRA, 1993). For our probabilistic analysis, we were able to use the entire distribution of landfill areas, omitting those areas that would not be reasonably large enough to accept the EDC/VCM sludge quantities in our analysis. For our deterministic analysis, we selected the 50th percentile value of the municipal landfill area distribution, 60,705 m^2, to represent our central tendency landfill area (DPRA, 1993). Selecting the high end landfill area for nongroundwater and groundwater pathways was more problematic. For nongroundwater pathways, the larger the landfill, the lower the effective waste concentration due to dilution of waste; therefore, a small landfill results in higher concentrations of constituents transported offsite via air or overland flow pathways. For groundwater, a larger landfill results in greater risk because the dilution/attenuation of the leachate plume is smaller when the plume is larger, even given the dilutional effect of having a greater amount of non-EDC/VCM waste in the landfill. A high end landfill area for nongroundwater pathways corresponds to the 10th percentile value (8,094 m^2); however, a landfill this small could not accommodate the EDC/VCM waste quantity generated over a 30-year period. Therefore, we evaluated only a central tendency landfill area (60,705 m^2) for nongroundwater pathways. For groundwater pathways, our high end landfill area was 420,888 m^2, corresponding to the 90th percentile of the municipal landfill distribution.

EPA has not collected information from municipal solid waste owners and operators on the depths of their landfills. The only available source of landfill depth information is data collected from municipal landfill permits in the state of Texas. For our deterministic analysis, we set landfill depth at 11 m, the 50th percentile depth in the Texas database. For the probabilistic analysis, the landfill depths ranged from approximately 2 to 61 meters, based on this same distribution.

EDC/VCM Sludges in an Onsite Land Treatment Unit

The land treatment unit waste management scenario that we evaluated represents a combination of site-specific and generic parameters. One facility (Georgia Gulf) reported in their §3007 survey response that they manage their EDC/VCM sludges in a land treatment unit located at their facility in Plaquemine, Louisiana. We used the primary characteristics of Georgia Gulf's land treatment unit, as available, to establish the general land treatment unit waste management scenario. Specifically, the Plaquemine location is the location we evaluated in our risk analysis (the representative meteorological station is Baton Rouge, LA). The total quantity of sludge managed by the facility in the land treatment unit is 1750 Mtons/year. The quantity of that sludge that we calculated as attributable to EDC/VCM production is 624 Mtons/year. As was the case for the landfill analysis, because our EDC/VCM sludge analytical data are derived from dedicated EDC/VCM sludge samples, we assumed that the land treatment unit receives 624 Mtons of dedicated EDC/VCM sludge per year. We assumed that the land treatment unit is the same size as the unit operated by Georgia Gulf, 687,990 m^2.

We did not obtain site-specific information that describes the operation of Georgia Gulf's land treatment unit. We assumed that the land treatment unit operates for 40 years, at which time it ceases to receive EDC/VCM sludge, but that it continues to release contaminants into the environment for an additional 40 years. We assumed that there are no runoff/runon controls in place at the land treatment unit to mitigate the release of sludge via runoff and erosion. Land treating often involves tilling the waste into the soil, and we assumed that the EDC/VCM sludge is tilled to a depth of 0.2 m (U.S.EPA 1990). Land treatment units commonly are unlined (allowing releases to groundwater) and uncovered (allowing releases to air).

2.1.4.3 Methyl Chloride Sludges

Our review of the §3007 survey responses found that methyl chloride sludges are managed in an onsite nonhazardous waste landfill located in Carrollton, Kentucky. The volume of wastewater treatment sludge disposed at the facility has been reduced by about 80 percent since 1993 due to use of the sludge as a raw material by cement kilns. The facility reports that they currently generate 776 Mtons of wastewater treatment sludge annually, and we assume, based on the quantities of methyl chloride wastewaters generated, that approximately 142 Mtons of this sludge results from methyl chloride production. Because the sludge, as generated, is not dedicated, and we conducted our risk analysis using the sample data for the single nondedicated methyl chloride sludge sample, we also conducted the risk analysis using the nondedicated (total) methyl chloride sludge quantity (776 Mtons/yr).

The facility disposes of their wastewater treatment sludge in an onsite permitted solid waste landfill. In addition to the wastewater treatment sludge, construction and demolition debris are disposed of in the landfill. The facility's permit allows construction of a 20-acre landfill (approximately 600 feet x 1,500 ft). However, the landfill is being constructed in phases and only four phases, covering an area of about 500 ft x 600 ft, have been used to date. The first two phases were 300 ft x 300 ft, and the third and fourth phases totaled 300 ft x 200 ft. Cells within each phase vary in size, but we assumed for our analysis that the average size is 100 ft × 100 ft with a depth of 6ft. After a cell has been filled, it is covered with 1 ft of sand and gravel. The cell walls are about 4 to 6 ft thick and are constructed with drums containing cured silicone. The base of the landfill is about 10 ft below land surface and has a 24-inch-thick clay liner and a leachate collection system. The landfill will be completed at about 60 ft above grade, with the first 30 ft constructed on a 1:4 slope. Elevations above 30 ft will be constructed on a 1:10 slope. The landfill has run-on controls, but daily cover is not applied. Based on the landfill size, and the volumes of waste currently generated, it would take over 400 years to complete the landfill. However, we estimated that the landfill would have an active life of 90 years, which corresponds to the estimated time to fill the unit to ground level. Based on the average landfill cell size and annual wastewater treatment sludge quantity, we assumed that one cell would be filled each year and that about 40 percent of the total waste disposed in the landfill would be nondedicated methyl chloride wastewater treatment sludge.

2.2 Description of Human Receptors and Exposure Pathways

The following sections describe the human receptors evaluated in our risk analyses. For each receptor we establish the pathways by which the receptor potentially could be exposed to contaminants in chlorinated aliphatics wastes.

2.2.1 Identification of Human Receptors

EPA determined that the following receptors reasonably represent the types of individuals that could be exposed to contaminants in chlorinated aliphatics wastes, and were the receptors evaluated in our risk analyses:

- Adult Resident
- Child Resident
- Home Gardener (adult)
- Adult Farmer
- Child of Farmer
- Fisher (adult)

These receptors reflect the range of possible individual exposures for direct and indirect exposure pathways. The farmer and the child of the farmer are the most sensitive subpopulations that we evaluated because our assumptions regarding the activity patterns and dietary habits of the farmer and child of the farmer result in the greatest number of exposure pathways.

The following subsections briefly describe the receptor scenarios that EPA evaluated in this risk analysis. The parameters (and data sources) we used to describe these receptors are provided in Appendix K.

Adult and Child Resident

We assume that an adult and child reside on a 5100 square meter lot (approximately 1.25 acres) located near the waste management unit. The residential receptors inhale vapors and particulate matter that are dispersed in the ambient air. We assume that household water is supplied to the residential receptors by a domestic groundwater well that is located near their home. The receptors drink water that comes from the well. The adult resident also is exposed to contaminants by inhaling vapors that are emitted from the water used in his/her house (for example, for showering) and through dermal contact while bathing. We did not evaluate risk or hazard for the child resident from dermal contact with contaminated water or inhaling vapors emitted from the water. We assume that young children are more likely to take baths than showers. Furthermore, the skin permeability constants developed by EPA may not be appropriate for children (U.S. EPA 1992). The residential receptors do not ingest foods that are grown in the vicinity of their home, however they do incidentally ingest surface soil from their yard.

Home Gardener

We assume that the residential receptor may have a home garden. The home gardener grows fruit, exposed vegetables (vegetables with edible parts that are exposed at land surface), and root vegetables in a plot that is 5100 square meters. Approximately 23 percent of the exposed vegetables, 11 percent of the root vegetables, and 12 percent of the fruits eaten by the gardener are grown in his/her garden. The gardener's other characteristics and activities are the same as those of the adult resident.

Fisher

EPA assumes that the residential receptor described above may be a recreational angler who obtains 32 percent of the fish in his/her diet from a stream located near the waste management unit. The fisher's other characteristics and activities are the same as those of the adult resident. EPA did not request that survey respondents report the distance between the unit in which they manage their wastewater treatment sludges and the nearest surface water body, consequently EPA does not have any data to describe that distance. Additionally, EPA does not have general information on the distances of nonhazardous landfills from surface water bodies. EPA did, however, ask the §3007 survey respondents to provide EPA the location of the closest water body to the facility boundary. This information indicated that a number of facilities in this industry are located adjacent to surface water bodies. We assumed that the stream in which the recreational angler fishes is located 102m from the waste management unit. If the fisher scenario had generated risks exceeding those of the other scenarios evaluated, EPA may have chosen to reevaluate our assumptions regarding the distance to surface water (see Section 5 for a summary of significant risk results for the fisher scenario).

Adult Farmer and Child of Farmer

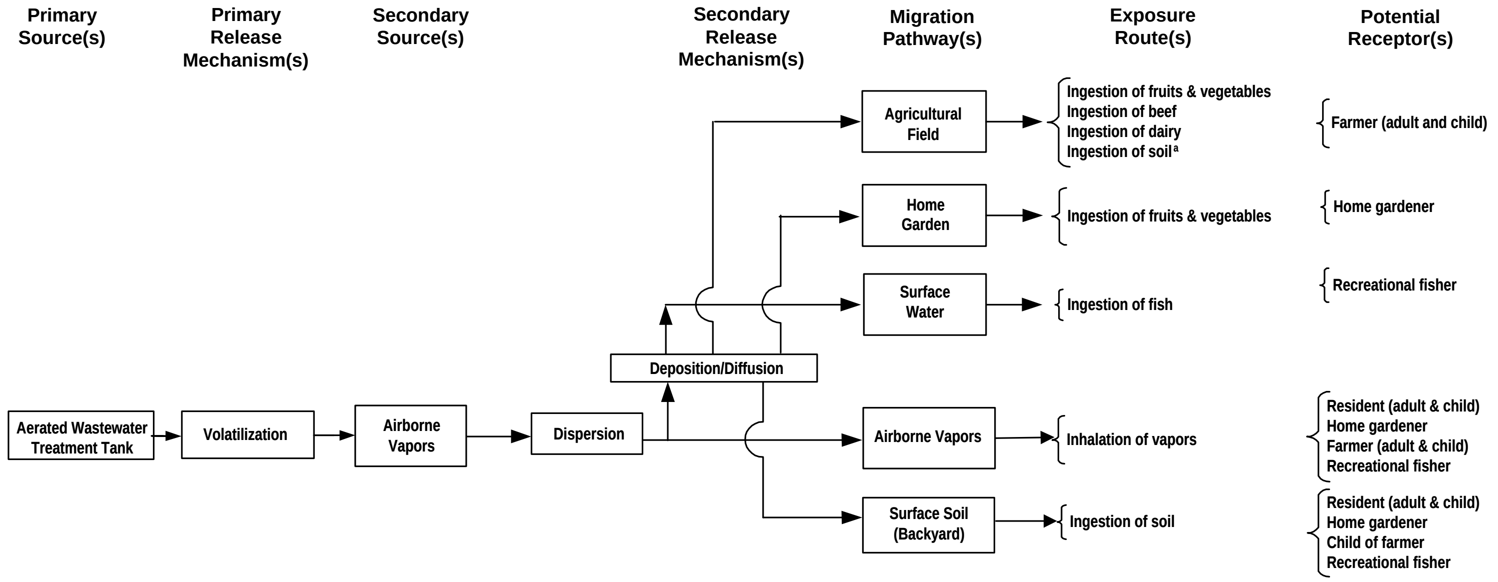
We assume that a farmer raises fruits, exposed vegetables, root vegetables, beef cattle, and dairy cattle in a 2,000,000 square meter agricultural field near the waste management unit. Approximately 42 percent of the exposed vegetables, 17 percent of the root vegetables, 33 percent of the fruits, 49 percent of the beef, and 25 percent of the dairy products eaten by the farmer and the child of the farmer are grown/raised on the farmer's agricultural plot. We assume that the farmer incidentally ingests soil from the agricultural field, and that the child of the farmer incidentally ingests soil from his/her yard. The farmer's and child's exposure to groundwater via ingestion, inhalation, and dermal contact are the same as that for the adult resident and child of the resident.

2.2.2 Identification of Exposure Pathways for Human Receptors

In the previous sections, we characterized the contaminant sources and the potential receptors. In order for human exposure to occur, there must be mechanisms by which contaminants are released from the source and be transported in the environment to a medium (air, food, soil) to which a receptor may be exposed. This section describes how we established exposure pathways for each of the receptors. Section 3.0 describes how we estimated the concentrations of contaminants at the receptor's point of exposure using contaminant fate and transport models.

Figures 2-3, 2-4, and 2-5 depict the pathways by which receptors may be exposed to contaminants released from wastewaters managed in onsite tanks, and wastewater treatment sludges managed in landfills and onsite land treatment units, respectively. "Exposure pathway" is a broad term that encompasses the contaminant source, contaminant release and transport mechanism(s), contaminant exposure points, and contaminant exposure routes (for example, inhalation, ingestion, and dermal contact). Exposure pathways are "direct" when a receptor is exposed directly to the contaminated source or media, such as air or groundwater. Exposure pathways also are "indirect" when a contaminant is released into one medium (for example, air), but subsequently is partitioned to other media, such as water, soil, or food, to which the receptor is exposed.

EPA determined that releases from all of the waste management units could occur through release of vapor emissions to the air. In addition, for the land treatment unit and the landfill, EPA determined that releases could occur through leaching of the waste into the subsurface. We assumed that the wastewater would entrain any particulate matter such that particulates would not be released from tanks. For the land treatment unit, releases could occur through release of particulate emissions to the air and runoff and erosion of waste from the unit. EPA did not evaluate particulate emissions from the landfills because the moisture content of the sludges (41 to 74 percent moisture and described in sampling logs as having a mud-like consistency [U.S.EPA 1999]) would prevent generation and release of particulates to the air in the time between placement of the waste in the landfill and application of daily cover or a new day's waste. EPA also assumed that runon/runoff controls would prevent releases from the landfills due to erosion and runoff.



Notes: → Complete pathway evaluated
[a] Adult farmer only

Figure 2-3. Exposure Pathways - Chlorinated Aliphatics Wastewater Managed in Onsite Tanks

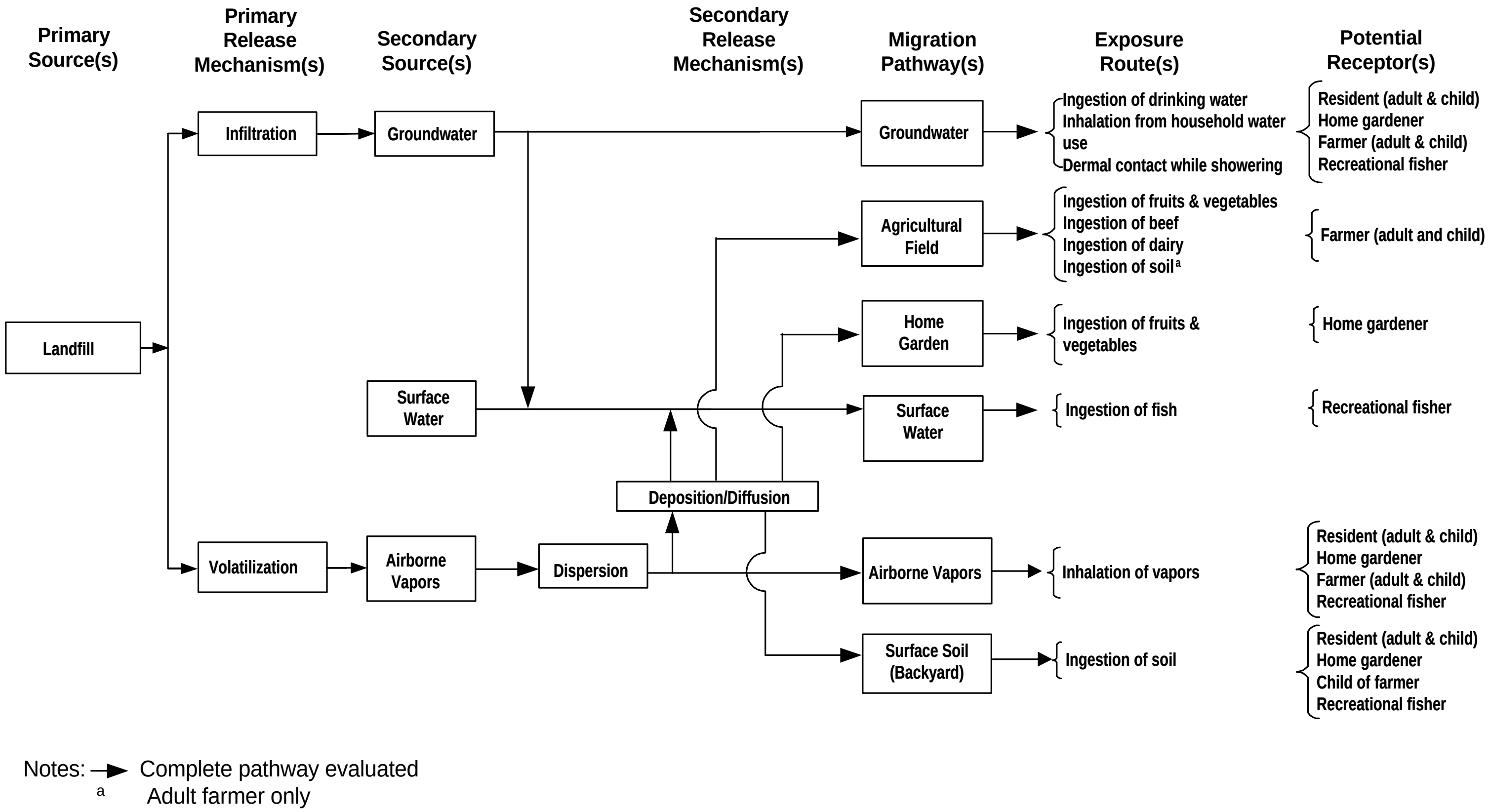
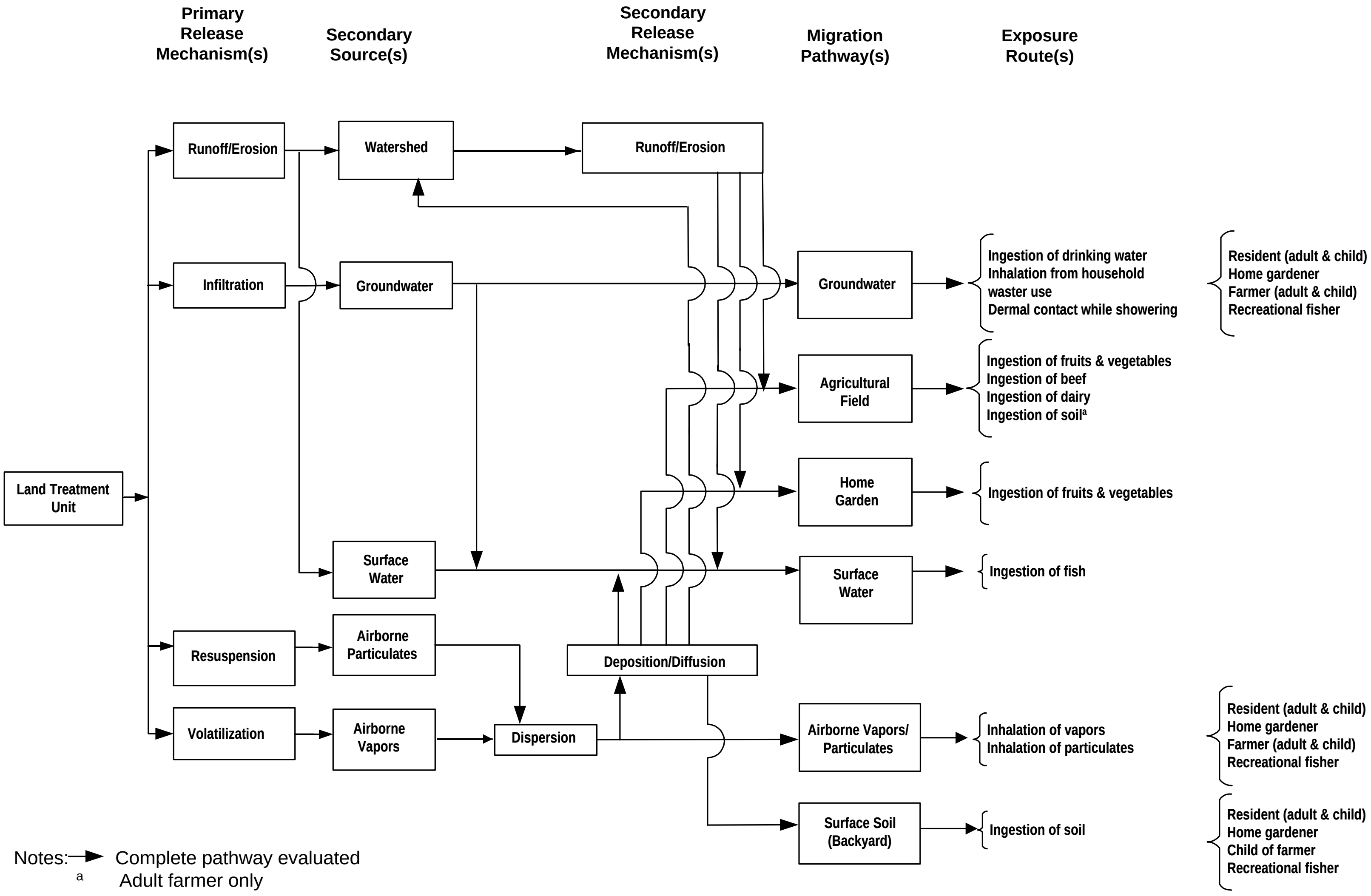


Figure 2-4. Exposure Pathways - Wastewater Treatment Sludges Managed in Landfills



Notes: → Complete pathway evaluated
[a] Adult farmer only

Figure 2-5. Exposure Pathways - Wastewater Treatment Sludge Managed in an On-Site Land Treatment Unit

EPA also evaluated the mechanisms and pathways by which contaminants might be transported to the points where receptors are exposed. The mechanisms and pathways evaluated are as follows:

- Eroded waste can be transported by runoff and deposited onto the soil and into surface water bodies.

- Leachate can migrate through the unsaturated (vadose) zone to the saturated zone, where contaminants are transported in groundwater to drinking water wells and to points of discharge to surface water bodies.

- Vapor emissions can remain dispersed in the air, or can be deposited through wet and dry deposition. Specifically, EPA models:

  - The concentration of vapor phase contaminants in air,
  - The diffusion of vapor phase contaminants into plants,
  - The diffusion of vapor phase contaminants into surface water,
  - Wet deposition of vapors onto soils and surface water (for example, due to wash-out [scavenging] by precipitation).
  - Dry deposition of vapors onto soils (for example, due to density).

  Although we do not evaluate wet and dry deposition of vapors onto plants, we do assume that wet and dry deposition of vapors onto soils increases the contaminant concentrations in the soil and results in additional uptake of contaminants into plants via soil-to-plant uptake.

- Particulate emissions can remain dispersed in the air or be deposited through wet deposition (in precipitation) or dry deposition (particle settling). We assume that particulates may be deposited onto soil and surface water through both wet and dry deposition, and onto plants through dry deposition.

Individuals indirectly may come into contact with contaminants in fruits and vegetables when contaminant vapors diffuse into vegetation, contaminated particles are deposited on vegetation, or when contaminants are taken up by vegetation from the soil. Additional indirect exposures can occur through the ingestion of contaminated fish, or home-raised beef and dairy products. Contamination of beef and dairy products occurs when cattle ingest contaminated forage, silage, grain, and surface soil. Contamination of fish occurs through contamination of the surface water body. Contaminants enter the surface water body through vapor diffusion, vapor and particle deposition, overland runoff/erosion, and groundwater discharge.

Exposure to groundwater occurs through the use of water from drinking water wells, and exposure via nongroundwater pathways occurs through runoff/erosion and releases to air. Therefore, “distance to receptor” for groundwater exposure pathways actually is the distance to the drinking water well that the receptor is using (the “receptor well”). “Distance to receptor” for nongroundwater pathways is the distance to the residence where the receptor is inhaling air or contacting soil, the distance to the garden where the receptor is growing fruits and vegetables, or the distance to the field where the receptor is growing crops or raising livestock. Consequently, we use different databases to establish “distance to receptor,” depending on whether we are evaluating a groundwater or a nongroundwater pathway.

For analysis of the nongroundwater pathway (air pathways and erosion/runoff) risks in the deterministic analysis we assumed that the receptors live at either 75 m (high end) or 300 m (central tendency) from the waste management unit. The distance of 250 ft (approximately 75 m) is based on the actual measured distance to the nearest resident for the worst-case facility evaluated in the risk assessment conducted to support the “Hazardous Waste Treatment, Storage, and Disposal Facilities - Organic Air Emissions Standards for Process Vents and Equipment Leaks Final Rule” (55 FR 25454), and was used as distance to the nearest resident for that rulemaking. In the same risk assessment, EPA identified the receptor distance of 1000 ft (approximately 300 m) as the median distance in a random sample of distances to the nearest residence. For the probabilistic analysis, we assume the receptors live either 50, 75, 100, 200, 300, 500, or 1000 m from the waste management unit. For air pathway analyses, we always assume that the receptors live along the centerline of the area most greatly impacted by air releases from the waste management units.

For deterministic analyses we assume that a receptor well is located 102 m (high end) or 430 m (central tendency) from the waste management unit, and that the receptor well is located on centerline of the contaminant plume (high end) or halfway between the centerline and the edge of the contaminant plume (central tendency). The 102 m distance is the 10th percentile value in the distribution of distances derived from EPA’s survey of municipal solid waste landfill facilities completed in 1986 (DPRA 1993). The 430 m value is the 50th percentile value in that same distribution. For the Monte Carlo analysis, the distance from the waste management unit to the receptor well is based on the complete distribution of distances to receptor well reported by the survey respondents, and ranges from 0.02 m (the location of the closest reported well was 0 m) to 1604 m (the maximum distance for which EPA requested receptor well information was 1 mile). For the Monte Carlo analysis we assume that the receptor well can be located anywhere within the contaminant plume. We base the value for the depth to the well intake on a database developed by the American Petroleum Institute (API 1989). For the deterministic analyses, the depth to the well intake was 4.57 m. For the probabilistic analyses, the depth to the well intake was based on API's distribution for the geographic region of interest. We assume, however, that the practical limit to the depth that a residential drinking water well extends into an aquifer is 10 m.

2.3 Identification of Constituents of Potential Concern (COPCs)

Based on the results of the analysis of waste samples and the evaluation of the contaminant exposure scenarios, EPA developed a list of "constituents of potential concern" (COPCs) for the chlorinated aliphatics wastewaters, EDC/VCM sludges, and methyl chloride sludges. The COPCs, presented in Table 2-8, are the constituents which were the subject of EPA's risk analysis. EPA developed the COPC list by taking the complete list of detected constituents and removing the following constituents:

- Constituent groups (for example, TOC, oil and grease, total PeCDF).
- For chlorinated aliphatics wastewaters and EDC/VCM sludges, constituents which were detected in only one of the samples, and the constituent concentration in that sample was an estimated ("J") value.
- Constituents that are essential nutrients and only pose risk at very high concentrations (that is, calcium, iron, magnesium, manganese, potassium, and sodium).
- For evaluations of risks or hazards that result from the emission of vapors into the air (the air vapor pathway), we excluded all metals, except mercury, from the COPC list. We excluded metals from the evaluation of air vapor pathway risks because metals, with the exception of mercury, are not volatile at ambient temperatures. Metals that we eliminated from the COPC list for the air vapor pathway were retained on the COPC list for the other pathways.

Table 2-8. COPCs for Chlorinated Aliphatics Wastewaters, EDC/VCM Sludges, and Methyl Chloride Sludges

Constituent	CAS No.	Chlorinated Aliphatics Wastewaters	EDC/VCM Sludges				Methyl Chloride Sludges	
			Groundwater Pathways		Nongroundwater Pathways			
		Non-Groundwater Pathways	LTU	Landfill	LTU	Landfill	Groundwater Pathways	Non-Groundwater Pathways
Volatile Organics								
1,2-Dichloroethane	107062	X	g	X	X	X		
2-Chloro-1,3-butadiene	126998	X						
Acetone	67641	X	g	g	X	X	g	X
Allyl chloride	107051	X	g		X	X		
Bromoform	75252	j*	g					
Carbon disulfide	75150	X	g	g	X	X	g	
Chlorobenzene	108907	X						
Chloroethane	75003	X						
Chloroform	67663	X	g	X	X	X		
cis-1,2-Dichloroethylene	156592	X						
cis-1,3-Dichloropropene	10061015			j*				
Chlorodibromomethane	124481	j*	g					
Ethylbenzene	100414	X						
2-Hexanone	591786				j	j		
Methyl ethyl ketone	78933	X	g	g	X	X		
Methylene chloride	75092	j*	g	X	X	X	g	X
Styrene	100425	X						
Tetrachloroethylene	127184	X	g,j		j*	j*		
trans-1,2-Dichloroethylene	156605	j*						
Trichloroethylene	79016	X	g,j		j*	j*		
Vinyl Chloride	75014		g,j		j*	j*		
Vinyl Acetate	108054		g		X	X		

(continued)

Table 2-8. (continued)

Constituent	CAS No.	Chlorinated Aliphatics Wastewaters	EDC/VCM Sludges				Methyl Chloride Sludges	
		Non-Groundwater Pathways	Groundwater Pathways		Nongroundwater Pathways		Groundwater Pathways	Non-Groundwater Pathways
			LTU	Landfill	LTU	Landfill		
Semivolatile Organics								
4-Aminobiphenyl	92671	j						
Benzoic acid	65850	**X**	g,j	g	j*	j*		
Benzyl alcohol	100516	**X**					g	
Bis(2-chloroethyl)ether	111444	**X**	**X**	**X**	**X**	**X**		
Bis(2-chloroisopropyl)ether	39638329	**X**						
2-(2-Chloroethoxy)ethanol	628897		**X**	**X**				
Diethyl phthalate	84662	**X**						
Dimethyl phthalate	131113	j						
Bis(2-ethylhexyl)phthalate	117817	j*	g		**X**	**X**		
Hexachlorobenzene	118741	j*	g,j		j*	j*		
4-Methyl-2-pentanone	108101			g				
2-Methylphenol	95487	**X**						
4-Methylphenol	106445	**X**		g				
Di-n-octyl phthalate	117840	j*						
Pentachlorophenol	87865	**X**						
Phenol	108952	**X**						
2,4,5-Trichlorophenol	95954	**X**						
2,4,6-Trichlorophenol	88062	**X**						
Metals								
Aluminum	7429905	v	**X**		**X**	v		v
Arsenic	7440382	v	**X**	**X**	**X**	v	**X**	v

(continued)

X = Constituent of Potential Concern.
g = Constituent screened out of the groundwater pathway analysis.
v = Constituent screened out of the nongroundwater pathway analysis (only the vapor air pathway was evaluated).
e = Essential nutrient.
j = All values were non-detect, with the exception of one "J"-qualified (estimated) value; "*" indicates that although screened out, the constituent was retained in the analysis inadvertently.

Table 2-8. (continued)

Constituent	CAS No.	Chlorinated Aliphatics Wastewaters	EDC/VCM Sludges				Methyl Chloride Sludges	
		Non-Groundwater Pathways	Groundwater Pathways		Nongroundwater Pathways		Groundwater Pathways	Non-Groundwater Pathways
			LTU	Landfill	LTU	Landfill		
Barium	7440393	v	g		**X**	v		
Beryllium	7440417	v						
Cadmium	7440439	v	g	**X**	**X**	v		
Calcium	7440702	v	e	e	e	v	e	v
Chromium	7440473	v	g		**X**	v		v
Cobalt	7440484	v	g	g	**X**	v		
Copper	7440508	v	**X**	**X**	**X**	v	**X**	v
Iron	7439896	v			e	v		v
Lead	7439921	v	**X**		**X**	v		v
Magnesium	7439954	v	e	e	e	v	e	v
Manganese	7439965	v	g	e	e	v	g	v
Mercury	7439976	**X**						
Molybdenum	7439987	v	g	**X**	**X**	v		
Nickel	7440020	v	g	**X**	**X**	v		v
Potassium	7440097	v	e	e	e	v		
Sodium	7440235	v	e		e	v		
Vanadium	7440622	v	g		**X**	v		
Zinc	7440666	v	g	g	**X**	v	g	v
Dioxins/Furans								
1,2,3,4,6,7,8-HpCDD	35822469	**X**	g		**X**	**X**		**X**
1,2,3,4,6,7,8-HpCDF	67562394	**X**	g	**X**	**X**	**X**		**X**
1,2,3,4,7,8,9-HpCDF	55673897	**X**	g	**X**	**X**	**X**		
1,2,3,4,7,8-HxCDD	39227286	**X**	g		**X**	**X**		

(continued)

X = Constituent of Potential Concern.
g = Constituent screened out of the groundwater pathway analysis.
v = Constituent screened out of the nongroundwater pathway analysis (only the vapor air pathway was evaluated).
e = Essential nutrient.
j = All values were non-detect, with the exception of one “J”-qualified (estimated) value; “*” indicates that although screened out, the constituent was retained in the analysis inadvertently.

Table 2-8. (continued)

Constituent	CAS No.	Chlorinated Aliphatics Wastewaters	EDC/VCM Sludges				Methyl Chloride Sludges	
		Non-Groundwater Pathways	Groundwater Pathways		Nongroundwater Pathways		Groundwater Pathways	Non-Groundwater Pathways
			LTU	Landfill	LTU	Landfill		
1,2,3,6,7,8-HxCDD	57653857	**X**	g		**X**	**X**		
1,2,3,7,8,9-HxCDD	19408743	**X**	g		**X**	**X**		
1,2,3,4,7,8-HxCDF	70648269	**X**	g		**X**	**X**		
1,2,3,6,7,8-HxCDF	57117449	**X**	g		**X**	**X**		
1,2,3,7,8,9-HxCDF	72918219	**X**	g		**X**	**X**		
2,3,4,6,7,8-HxCDF	60851345	**X**	g		**X**	**X**		
2,3,4,7,8-PeCDF	57117314	**X**	g		**X**	**X**		
1,2,3,7,8-PeCDF	57117416		g		**X**	**X**		
2,3,7,8-TCDD	41903575	**X**	g		**X**	**X**		
2,3,7,8-TCDF	51207319	**X**	g		**X**	**X**		
OCDD	3268879	**X**	g	g	**X**	**X**		**X**
OCDF	39001020	**X**	g	**X**	**X**	**X**		**X**

X = Constituent of Potential Concern.
g = Constituent screened out of the groundwater pathway analysis.
v = Constituent screened out of the nongroundwater pathway analysis (only the vapor air pathway was evaluated).
e = Essential nutrient.
j = All values were non-detect, with the exception of one "J"-qualified (estimated) value; "*" indicates that although screened out, the constituent was retained in the analysis inadvertently.

- For evaluations of risks or hazards that result from release of contaminants to groundwater (the groundwater pathway), we excluded all constituents which pass a screening analysis that predicts the risk associated with drinking the "leachate" from the waste. We describe the screening analysis in more detail below. Any constituents that we eliminated from the COPC list for the groundwater pathway were retained on the list for the other pathways.

To determine if we could eliminate constituents from evaluation for groundwater pathway risks, we conducted a screening analysis that maximizes risk or hazard from the direct ingestion of waste leachate. We conducted this screening analysis as follows:

- For carcinogens, we calculated the carcinogenic risk for a 70 kilogram (kg) adult who ingests 1.4 liters/day (L/day) of waste leachate 350 days/year for 58.4 years. 70 kg is the generally accepted mean body weight for an adult; 1.4 L/day is the mean drinking water ingestion rate for an adult; 350 days/year, which accounts for the receptor being elsewhere on vacation for 2 weeks/year, is the exposure frequency; and 58.4 years is the 95th percentile exposure duration for farmers (U.S.EPA 1997a,b,c).

- For noncarcinogens, we calculated non-cancer hazard quotients (HQs) for a 21.4 kg child who ingests 0.74 L/day of waste leachate 350 days per year for 9 years. 21.4 kg is the mean body weight for children 1 to 10 years in age; 0.74 L/day is the mean drinking water ingestion rate for 1 to 10 year old children; 350 days/year, which accounts for the receptor being elsewhere on vacation for 2 weeks/year, is the exposure frequency; and 9 years represents an exposure duration for a child whose exposure begins at age 1 and ends at age 10 (U.S.EPA 1997a,b,c).

We retained in our groundwater pathway analysis all constituents for which the adult's carcinogenic risk exceeded 1×10^{-6} or for which the child's HQ exceeded 1. For the landfill waste management scenarios, the leachate concentrations we evaluated for the screening analysis are the maximum detected TCLP concentrations. EPA developed the TCLP analysis to simulate the concentrations of contaminants in municipal landfill leachate. For the land treatment unit waste management scenario, we predicted the leachate concentrations using a waste partitioning analysis that is described in Section 3.1. The results of the groundwater pathway screening analysis are presented in Appendix B. Some of the sample data for three of the TCLP constituents in EDC/VCM sludges were qualified with the "B" qualifier, indicating that these constituents also were detected in sample blanks (Section 2.1.3). The constituents that carried the “B” qualifiers were acetone, 4-methyl-2-pentanone, and methylene chloride. Acetone and 4-methyl-2-pentanone were screened out of the groundwater pathway analysis. The two samples with the highest methylene chloride concentrations were not “B”-qualified (Table 2-2), therefore, we retained the “B”-qualified data in the analysis as they were reported.

Table 2-8 indicates that one constituent, arsenic, is a COPC for exposure to methyl chloride sludges via the groundwater pathway. However, EPA did not evaluate arsenic in methyl chloride sludges further. EPA estimated that the risk associated with drinking leachate from the methyl chloride sludge landfill for 58 years is $5x10^{-5}$ (that is, the cancer risk is 5 in 100,000) due to arsenic (Appendix B, Table B-3). The Agency views the arsenic risk results from the screening analysis as marginal. Assuming that the landfill leachate is diluted and attenuated in the aquifer by a factor of at least 5 before it reaches the receptor well (a reasonable assumption for an unlined landfill),[3] the predicted risk becomes less than $1x10^{-5}$, the typical risk identified by EPA as sufficient to require the waste to be listed as hazardous (see 59 FR 66072). In addition, for the methyl chloride sludge is managed at a single facility in a landfill that is lined with a 24-inch clay liner and has a leachate collection system. The landfill is located onsite and has significant remaining capacity, such that we have no reason to assume that the facility will not continue to manage its wastewater treatment sludges from the production of methyl chloride in this manner. Consequently, we expect the actual potential risk from the methyl chloride sludge will be much lower than the risk level predicted by the screening analysis.

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[3] Our groundwater fate and transport modeling for arsenic in leachate derived from an unlined EDC/VCM landfill resulted in dilution/attenuation factors (DAFs) ranging from 13.5 to 92 (see Table 2.2 and Appendix H, Table H.3-4). The DAF is the ratio of the concentration of a constituent in landfill leachate to the concentration of the constituent at the receptor well.

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3.0 ESTIMATING EXPOSURE POINT CONCENTRATIONS

EPA conducts contaminant fate and transport modeling and indirect exposure modeling to determine what the concentrations of contaminants will be in the media that the receptor comes into contact with (for example, groundwater, air, soil, food items). These concentrations are called “exposure point concentrations” (that is, they are the contaminant concentrations at the point at which the receptor is exposed). There are a number of computer-based models and sets of equations that EPA uses to predict exposure point concentrations. In the following sections, we briefly discuss these models and equations and their application in the risk analysis. Detailed descriptions of the methods are provided in Appendices D and E . Appendix C contains the physical and chemical properties used to model the fate and transport of contaminants in the environment. Appendix C also contains the biological transfer factors that we used to estimate the uptake of contaminants by plants and animals. The exposure point concentrations for the contaminants that produced significant risk results (described in Section 5) are provided in Appendix H.

3.1 Contaminant Partitioning in Landfills and in the Land Treatment Unit (Partitioning Model)

For the landfills and the land treatment unit, EPA uses a series of “partitioning” equations to determine how much contaminant mass is retained in the waste management unit and how much is released into the environment. The partitioning equations are based upon equations presented in a series of articles by Jury et al. (Jury et al. 1983, 1984, and 1990).

EPA used the partitioning equations to estimate the mass of a contaminant that will be lost from the EDC/VCM sludge land treatment unit due to volatilization into the air, leaching into the subsurface, erosion and runoff, and degradation. For the land treatment unit, EPA used equations documented in EPA’s “Compilation of Air Pollutant Emission Factors (AP- 42)” (U.S. EPA 1995a) to estimate particulate emissions resulting from wind erosion and tilling activities. Emissions from wind erosion were modeled for particles that are 10 microns (PM10) or smaller because: (1) this is the size range of respirable particulates that is of concern to us from the standpoint of evaluating inhalation risks and (2) the emission of larger particles (PM30) is relatively insignificant from this process. Emissions from tilling activities were modeled for both smaller (PM10) and larger (PM30) particle size ranges.

For the landfill scenarios (the EDC/VCM sludge landfill and the methyl chloride sludge landfill), EPA used the partitioning equations to determine how much of the contaminant mass would be lost due to volatilization into the air; we assumed that the remainder of the mass would be available to leach into the subsurface. As illustrated in Figure 3-1, the model accounts for volatilization losses that occur (1) prior to the landfill being covered with daily cover or daily waste addition, (2) through the daily cover or daily waste addition, and (3) through the cap that is placed on the landfill after closure. For the landfill, we used toxicity characteristic leaching procedure (TCLP) analytical results (rather than the partitioning equations) to predict leachate concentration. Appendix D.1 provides a complete description of the partitioning model.

3.2 Contaminant Emissions from Wastewater Treatment Tanks (CHEMDAT8)

EPA modeled vapor emissions from aerated biological wastewater treatment tanks using the CHEMDAT8 model (U.S. EPA 1994c). The resulting emission estimates were used in conjunction with the dispersion modeling results (Section 3.3) to estimate constituent-specific air concentrations and deposition rates. Use of CHEMDAT8 requires specifying parameters relating to tank characteristics, waste characteristics, contaminant physical and chemical properties, and location-specific meteorological conditions (for example, windspeed and temperature). We discussed these parameters in Section 2. CHEMDAT8 accounts for most of the competing removal pathways that might limit air emissions, including adsorption, biodegradation, and hydrolysis. Chemicals that sorb to solids or decompose due to either biodegradation or hydrolysis have lower potential for emission to the air.

In modeling the emissions of the dioxin congeners in wastewaters using CHEMDAT8, we considered the concentrations of the congeners relative to their solubility limits in water. Because dioxins are strongly hydrophobic (octanol/water partition coefficients [K_{ow}s] ranging from 3.4×10^6 to 6.3×10^8), we expect that the dioxins in the wastewaters would be preferentially sorbed onto the suspended solids in the wastewater influent, and that they are unlikely to exist in the dissolved phase in excess of solubility limits. Consequently, for both the deterministic and probabilistic analyses, we modeled wastewater emissions at the solubility limit for three congeners with sample concentrations that exceeded their respective solubility limits: 1,2,3,4,6,7,8-HpCDF, OCDD. This reduced the central tendency TEQ emissions for wastewaters by roughly a factor of 2 to produce the central tendency estimate shown in

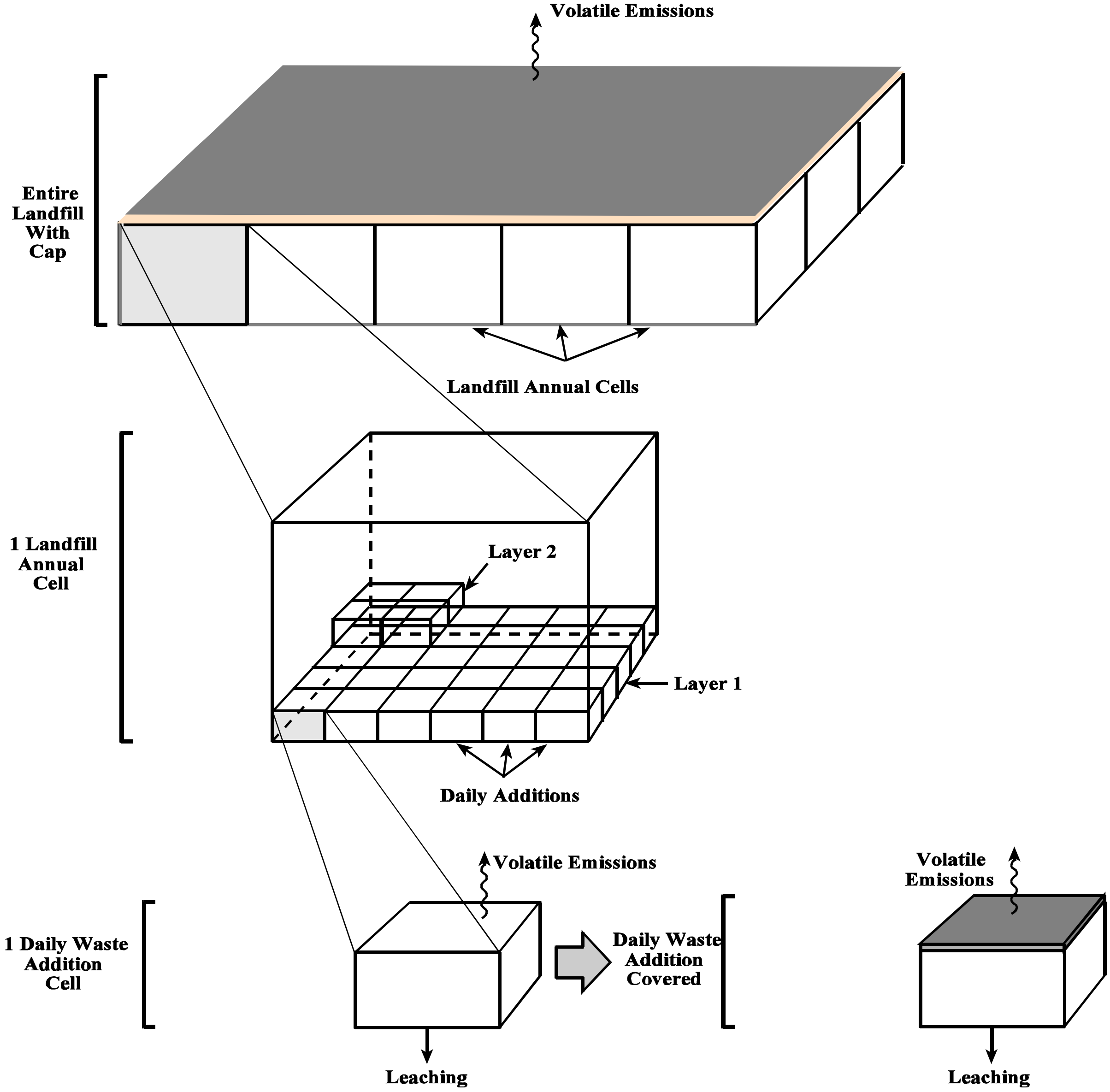

Figure 3-1. Schematic View of Landfill

Table 3-1a. The high end estimate shown in Table 3-1b was reduced by about a factor of 7 from the TEQ emissions that were calculated using sample data that were not adjusted for dioxin solubilities (see Table 2-1 for central tendency and high end wastewater concentrations). Using the emissions model, we estimate that, of the total annual mass of dioxin influent to the modeled tank, only 0.1 to 0.4 percent of the dioxin mass would be emitted into the air in the vapor phase. Appendix D.3 provides a complete description of our CHEMDAT8 modeling procedures.

3.3 Air Dispersion and Deposition (ISCST3)

We used EPA's Industrial Source Complex Short Term Model (Version 3; ISCST3; U.S. EPA 1995b) to estimate the dispersion and deposition of vapors emitted from the chlorinated aliphatics wastewater treatment tank, the EDC/VCM sludge municipal landfill, the methyl chloride sludge onsite industrial landfill, and the EDC/VCM sludge land treatment unit. EPA also used ISCST3 to estimate the dispersion and deposition of particulate emissions from the land treatment unit. Vapor emissions from the landfills and the land treatment unit, and particulate emissions from the land treatment unit, were estimated using the partitioning and emissions models discussed in Section 3.1. Vapor emissions from the wastewater treatment tank were estimated using the CHEMDAT8 model discussed in Section 3.2. ISCST3 was used to estimate the:

- air concentration of vapors,
- air concentration of particulates,
- wet deposition of vapors onto soils and surface water,
- wet deposition of particulates onto soils and surface water, and
- dry deposition of particulates onto soils, surface water, and plants.

Because ISC currently cannot be used to model dry vapor deposition, we estimated dry deposition of vapors onto soils using an algorithm for the dry deposition of particles (from the ISCST users' manual), with a default deposition velocity of 0.2 centimeters per second (cm/s). Applying this approach to dioxins implicitly assumes that these compounds behave as fine aerosols and, therefore, are amenable to modeling using a dry deposition algorithm for particles. EPA decided that this was a reasonable alternative to use for all organic constituents

Table 3-1a. Estimated Central Tendency Dioxin Congener Emissions from Wastewater Tanks

Congener	TEF	PL-01 Concentration (ng/L)	Annual Quantity (g/yr)[a]	Annual TEQ Quantity (g/yr)	Annual Emissions (Mg/yr)	Annual Emissions (g/yr)	Annual TEQ Emissions (g/yr)
1,2,3,4,6,7,8-HpCDD	0.01	0.310	0.100	0.001	1.42E-10	1.42E-04	1.42E-06
1,2,3,4,6,7,8-HpCDF	0.01	4.60	1.481	0.015	6.92E-09	6.92E-03	6.92E-05
1,2,3,4,7,8,9-HpCDF	0.01	0.830	0.267	0.003	4.10E-09	4.10E-03	4.10E-05
1,2,3,4,7,8-HxCDD	0.1	0	0	0	0	0	0
1,2,3,4,7,8-HxCDF	0.1	0.610	0.196	0.020	2.07E-09	2.07E-03	2.07E-04
1,2,3,6,7,8-HxCDD	0.1	0	0	0	0	0	0
1,2,3,6,7,8-HxCDF	0.1	0.280	0.090	0.009	4.19E-10	4.19E-04	4.19E-05
1,2,3,7,8,9-HxCDD	0.1	0	0	0	0	0	0
1,2,3,7,8,9-HxCDF	0.1	0.076	0.024	0.002	1.85E-10	1.85E-04	1.85E-05
2,3,4,6,7,8-HxCDF	0.1	0.120	0.039	0.004	2.92E-10	2.92E-04	2.92E-05
2,3,4,7,8-PeCDF	0.5	0	0	0	0	0	0
2,3,7,8-TCDD	1	0	0	0	0	0	0
2,3,7,8-TCDF	0.1	0	0	0	0	0	0
OCDD	0.001	6.50	2.093	0.002	7.79E-14	7.79E-08	7.79E-11
OCDF	0.001	140	45.072	0.045	5.52E-11	5.52E-05	5.52E-08
		Total	**49.26**	**0.10**		**0.014**	**0.00041**
					% Total	**0.03%**	**0.41%**

[a] Based on central tendency annual waste quantity of 321,946 m^3 or Mtons.

Note: The TEQ emissions estimates presented in this table are based on the solubility limits for 1,2,3,4,6,7,8-HpCDF, OCDD, and OCDF (see Appendix C), and the sample concentrations in the PL-01 and GL-02 samples for the other congeners.

Table 3-1b. Estimated High End Dioxin Congener Emissions from Wastewater Tanks

Congener	TEF	G (ng/L) Concentration (ng/L)	Annual Quantity (g/yr)[a]	Annual TEQ Quantity (g/yr)[a]	Annual Emissions (Mg/yr)	Annual Emissions (g/yr)	Annual TEQ Emissions (g/yr)
1,2,3,4,6,7,8-HpCDD	0.01	0.880	0.283	0.00283	4.03E-10	4.03E-04	4.03E-06
1,2,3,4,6,7,8-HpCDF	0.01	43.0	13.844	0.138	6.92E-09	6.92E-03	6.92E-05
1,2,3,4,7,8,9-HpCDF	0.01	12.0	3.863	0.0386	6.92E-09	6.92E-03	6.92E-05
1,2,3,4,7,8-HxCDD	0.1	0.052	0.017	0.0017	7.25E-11	7.25E-05	7.25E-06
1,2,3,6,7,8-HxCDD	0.1	0.091	0.029	0.0029	1.80E-08	1.80E-02	1.80E-03
1,2,3,7,8,9-HxCDD	0.1	0.110	0.035	0.0035	2.65E-10	2.65E-04	2.65E-05
1,2,3,4,7,8-HxCDF	0.1	5.30	1.706	0.171	1.80E-09	1.80E-03	1.80E-04
1,2,3,6,7,8-HxCDF	0.1	1.20	0.386	0.0386	3.20E-10	3.20E-04	3.20E-05
1,2,3,7,8,9-HxCDF	0.1	0	0	0	0	0	0
2,3,4,6,7,8-HxCDF	0.1	0.430	0.138	0.0138	1.05E-09	1.05E-03	1.05E-04
2,3,4,7,8-PeCDF	0.5	0.210	0.068	0.034	5.96E-10	5.96E-04	2.98E-04
2,3,7,8-TCDD	1	0.017	0.0055	0.0055	6.30E-11	6.30E-05	6.30E-05
2,3,7,8-TCDF	0.1	0.082	0.026	0.0026	5.77E-10	5.77E-04	5.77E-05
OCDD	0.001	6.90	2.221	0.00222	7.79E-14	7.79E-08	7.79E-11
OCDF	0.001	6,000	1931.676	1.932	5.52E-11	5.52E-05	5.52E-08
		Total	**1954.3**	**2.387**		**0.037**	**0.003**
					% Total	**0.002%**	**0.11%**

[a] Based on central tendency annual waste quantity of 321,946 m^3 or metric tons.

Note: The TEQ emissions estimates presented in this table are based on the solubility limits for 1,2,3,4,6,7,8-HpCDF, OCDD, and OCDF (see Appendix C), and the sample concentrations in the PL-01 and GL-02 samples for the other congeners.

until suitable algorithms for dry vapor deposition become available in the ISC. This method tends to overstate dry deposition of lipophilic organics because the dry deposition of vapor may be negligible for dioxins and a significant fraction of the dioxin mass, approximately 25% to 75%, typically is bound to particles (Trapp and Matthies 1998; Koester and Hites 1992). However, plant concentrations that reflect dry vapor deposition to soil are within one percent of the concentrations predicted excluding this mechanism, that is, modeling only the air-to-plant transfer, wet deposition of vapors to soil, and wet and dry deposition of particulates. This is expected since the uptake of dioxins via the soil-to-plant pathway is considered negligible (U.S. EPA 1993). Appendix D.3 provides a complete description of our air dispersion and deposition modeling procedures.

3.4 Overland Transport (USLE)

The Universal Soil Loss Equation (USLE) is an empirical erosion model originally designed to estimate long-term average soil erosion losses to a nearby surface water body from an agricultural field having uniform slope, soil type, vegetative cover, and erosion-control practices. We used a modified form of the USLE to estimate the mass of soil lost per year per unit area from the land treatment unit and deposited directly onto an adjacent receptor site (agricultural field, residential lot, home garden) and into a nearby stream. Although we evaluated constituent losses from overland transport only for the land treatment unit, we assumed that constituents deposited on adjacent soils from the tanks or landfills via air dispersion and deposition are subject to transport by soil erosion and runoff to intervening soils and nearby water bodies. This overland transport mechanism depletes the contaminant mass deposited from air onto soil at the receptor site.

Because the basic USLE equation only estimates soil erosion to surface water bodies, EPA assumes the receptor location is between the land treatment unit and the surface water body. The area including the land treatment unit, the receptor site, and the intervening area is considered for the purposes of the analysis to be an independent, discrete drainage subbasin that is at steady-state. The soil erosion load from the subbasin to the surface water body is estimated using a distance-based sediment delivery ratio, and the sediment not reaching the surface water body is considered to be deposited evenly over the area of the subbasin. Thus, using mass balance equations, EPA estimates contaminant contributions to the surface water body and the intervening soil.

Contaminated particles are transported from the land treatment unit to receptor sites via air deposition as well as runoff/erosion. Mass balance considerations are applied for each area of interest (for example, the buffer area between source and receptor site, receptor site, or surrounding area). Consequently, the respective air deposition value for each area of interest is included in the mass balance evaluation. The air deposition over the entire subbasin area is considered uniform and equal to the air deposition modeled for the receptor site. Appendix D.2 provides a complete description of the overland transport model.

3.5 Groundwater Fate and Transport (EPACMTP)

Precipitation that migrates through the waste management unit generates leachate that infiltrates to the bottom of the waste management unit and migrates into the unsaturated zone. The contaminants dissolved in the leachate are transported in the aqueous phase through the unsaturated zone to the underlying saturated zone and then downgradient to a receptor (drinking water) well or surface water body located at a specified distance from the boundary of the waste management unit (Figure 3-2). We used EPA's Composite Model for Leachate Migration with Transformation Products (EPACMTP; U.S. EPA, 1996a, 1996b, 1996c, 1997a) to model the subsurface fate and transport of contaminants that leach from the EDC/VCM sludge land treatment unit or from the EDC/VCM sludge landfill and migrate to a residential drinking water well or discharge from groundwater to surface water. As discussed in Section 2.3, we did not conduct groundwater pathway modeling for the methyl chloride sludge landfill.

EPACMTP accounts for the following processes affecting contaminant fate and transport: advection, hydrodynamic dispersion, equilibrium linear or nonlinear sorption by the soil and aquifer solids, and contaminant hydrolysis. In the event that the hydrolysis daughter products are hazardous and their chemical properties are known, the model also accounts for the formation and subsequent fate and transport of the daughter products. We account for sorption of organic constituents by means of a partition coefficient (k_d) that we calculate as the product of the constituent-specific organic carbon partition coefficient (k_{oc}) and the fraction organic carbon (f_{oc}) in the soil and aquifer. For metals whose sorption is linearly related to

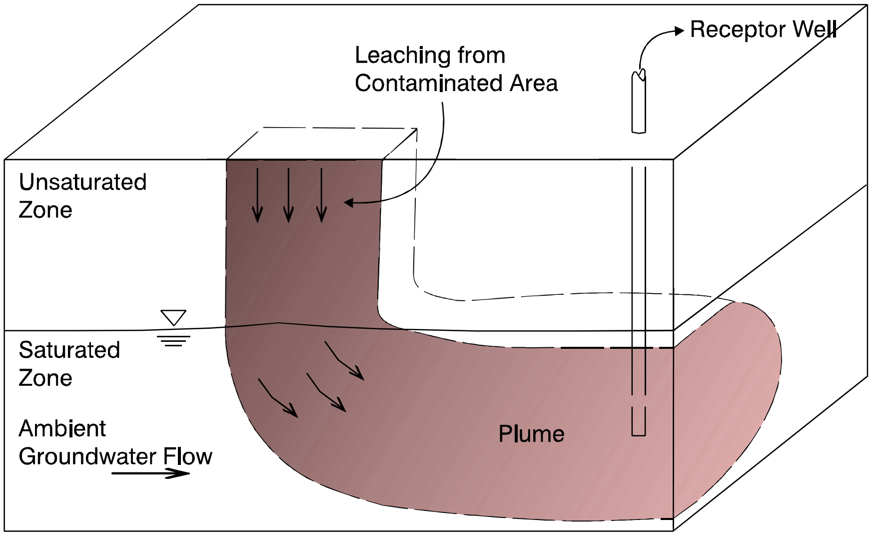

Figure 3-2. Schematic View of Groundwater Plume

concentration (arsenic and molybdenum), we account for sorption by incorporating into the analysis empirical sorption isotherms developed by EPA (Loux et al. 1990). For metals whose sorption is non-linearly related to concentration (nickel and manganese), we account for sorption by incorporating into the analysis sorption isotherms developed using the MINTEQA2 geochemical speciation model (Allison et al. 1991).

The primary transport mechanisms in the subsurface are downward movement along with infiltrating water in the unsaturated zone and movement along with (ambient) groundwater flow in the saturated zone. The advective movement in the unsaturated zone is one-dimensional, while the saturated zone module accounts for three-dimensional flow and transport. The model also considers mixing due to hydrodynamic dispersion in both the unsaturated and saturated zones. In the unsaturated zone, flow is gravity-driven and prevails in the vertically downward direction. Therefore, we model flow in the unsaturated zone as one-dimensional in the vertical direction. We also assume that transverse dispersion (both mechanical dispersion and molecular diffusion) is negligible in the unsaturated zone. This assumption is based on the fact that lateral migration due to transverse dispersion is negligible compared with the horizontal dimensions of waste management units. In addition, this assumption is environmentally protective because it allows the leading front of the contaminant plume to arrive at the water table with greater peak concentration in the case of a finite source.

As described in Section 2.1.4, we assumed that the EDC/VCM sludge landfill is a Subtitle D municipal landfill that has an earthen cover, but no liner or leachate collection system. The leachate flux through the landfill is the result of infiltration of ambient precipitation through the landfill cover. We calculated the leachate flux rates used in the analysis using the Hydrologic Evaluation of Landfill Performance (HELP) model (US EPA 1997a and 1997c; U.S. EPA, 1994a and 1994b). We calculated the net infiltration rate using a water balance approach which considers, among other factors, precipitation, evapotranspiration, and surface runoff. We assumed that each constituent initially leaches out of the landfill at a concentration equal to the TCLP concentration in the EDC/VCM sludge, with a gradual decrease in leaching concentration over time due to depletion of the source (U.S. EPA 1996a). Assuming that all of the constituent mass in the waste eventually may leach out with linear equilibrium partitioning of the constituent into the aqueous phase, the leaching concentration follows an exponential decrease with time.

Our landfill analysis was based on two simplifying assumptions. First, we assumed that contaminant leaching from the landfill does not occur until after the landfill closes (that is, after 30 years). EPA made this assumption because of complexities associated with linking the output of the landfill partitioning equations (discussed in Appendix D.1) and EPACMTP. Second, for the purpose of evaluating groundwater pathway risks, we assumed that there are no contaminant losses due to mechanisms other than leaching after the landfill has been closed (for the purpose of evaluating air pathway risks, we assumed that air releases from the landfill continue after closure). This effectively overestimates the total mass of volatile contaminants that would leach to groundwater because it does not allow volatilization losses from the landfill to deplete the total contaminant mass in the landfill in the years after closure. EPA determined that if volatile constituents caused significant risk via the groundwater pathway, we would have to re-evaluate our methodology for conducting the landfill analysis. This situation did not occur. Therefore, the total amount of constituent mass in the landfill available for leaching is given by 30 times the annual waste quantity disposed of in the landfill minus the volatilization losses that occurred prior to closure. Table 3-2 shows the waste concentrations we used in the

Table 3-2. Contaminant Losses Due to Volatilization During the Active Life of EDC/VCM Landfill

Contaminant	Parameter at High End	Waste Volume (m^3)	Waste Conc. (mg/kg)	Constituent Mass before Volatilization (mg)	Constituent Mass Volatilized (mg)	Constituent Mass after Volatilization (mg)	Waste Conc. after Volatilization (mg/kg)
Chloroform [b]	None (Central Tendency)	15,202	0.55	8.95E+06	8.79E+04	8.86E+06	0.545
	X-well	15,202	0.55	8.95E+06	8.79E+04	8.86E+06	0.545
	LF Area	15,202	0.55	8.95E+06	8.79E+04	8.86E+06	0.545
	Site Location	15,202	0.55	8.95E+06	8.79E+04	8.86E+06	0.545
	Leachate Concentration	15,202	0.55	8.95E+06	8.79E+04	8.86E+06	0.545
	Y-well	15,202	0.55	8.95E+06	8.79E+04	8.86E+06	0.545
	Waste Volume	50,579	0.55	2.98E+07	2.92E+05	2.95E+07	0.545
Methylene Chloride [b]	None (Central Tendency)	15,202	0.07	1.14E+06	1.38E+04	1.12E+06	0.069
	X-well	15,202	0.07	1.14E+06	1.38E+04	1.12E+06	0.069
	Y-well	15,202	0.07	1.14E+06	1.38E+04	1.12E+06	0.069
	Site Location	15,202	0.07	1.14E+06	1.37E+04	1.12E+06	0.069
	Leachate Concentration	15,202	0.07	1.14E+06	1.38E+04	1.12E+06	0.069
	Waste Volume	50,579	0.07	3.79E+06	4.58E+04	3.74E+06	0.069
	LF Area	15,202	0.07	1.14E+06	1.38E+04	1.12E+06	0.069
1,2 Dichloroethane [b]	None (Central Tendency)	15,202	0.53	8.62E+06	7.20E+04	8.55E+06	0.526
	X-well	15,202	0.53	8.62E+06	7.20E+04	8.55E+06	0.526

(continued)

Table 3-2. (continued)

Contaminant	Parameter at High End	Waste Volume (m^3)	Waste Conc. (mg/kg)	Constituent Mass before Volatilization (mg)	Constituent Mass Volatilized (mg)	Constituent Mass after Volatilization (mg)	Waste Conc. after Volatilization (mg/kg)
	LF Area	15,202	0.53	8.62E+06	7.20E+04	8.55E+06	0.526
	Site Location	15,202	0.53	8.62E+06	7.19E+04	8.55E+06	0.526
	Leachate Concentration	15,202	0.53	8.62E+06	7.20E+04	8.55E+06	0.526
	Y-well	15,202	0.53	8.62E+06	7.20E+04	8.55E+06	0.526
	Waste Volume	50,579	0.53	2.87E+07	2.40E+05	2.84E+07	0.526
cis -1,3 Dichloropropene [b]	None (Central Tendency)	15,202	0.0125	2.03E+05	8.10E+02	2.03E+05	0.013
	X-well	15,202	0.0125	2.03E+05	8.10E+02	2.03E+05	0.013
	Y-well	15,202	0.0125	2.03E+05	8.10E+02	2.03E+05	0.013
	LF Area	15,202	0.0125	2.03E+05	8.10E+02	2.03E+05	0.013
	Waste Volume	50,579	0.0125	6.77E+05	2.70E+03	6.74E+05	0.013
	Leachate Concentration	15,202	0.0125	2.03E+05	8.10E+02	2.03E+05	0.013
	Site Location	15,202	0.0125	2.03E+05	8.10E+02	2.03E+05	0.013
Bis(2-chloroethyl)ether [c]	None (Central Tendency)	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	Site Location	15,202	0.487	7.92E+06	5.68E+03	7.92E+06	0.487
	X-well	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	LF Area	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	Y-well	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	Waste Volume	50,579	0.487	2.64E+07	1.91E+04	2.63E+07	0.487

(continued)

Table 3-2. (continued)

Contaminant	Parameter at High End	Waste Volume (m^3)	Waste Conc. (mg/kg)	Constituent Mass before Volatilization (mg)	Constituent Mass Volatilized (mg)	Constituent Mass after Volatilization (mg)	Waste Conc. after Volatilization (mg/kg)
	Leachate Concentration	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
2(2-Chloroethoxy)ethanol[a, c]	None (Central Tendency)	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	Site Location	15,202	0.487	7.92E+06	5.68E+03	7.92E+06	0.487
	X-well	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	LF Area	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	Y-well	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	Leachate Concentration	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	Waste Volume	50,579	0.487	2.64E+07	1.91E+04	2.63E+07	0.487
p-Dioxane[a, c]	None (Central Tendency)	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	X-well	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	LF Area	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	Site Location	15,202	0.487	7.92E+06	5.68E+03	7.92E+06	0.487
	Y-well	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	Leachate Concentration	15,202	0.487	7.92E+06	5.74E+03	7.92E+06	0.487
	Waste Volume	50,579	0.487	2.64E+07	1.91E+04	2.63E+07	0.487

[a] Degradation product of bis(2-chloroethyl)ether.

[b] Based on dry-weight concentrations.

[c] Based on wet-weight concentrations.

groundwater pathway analysis, corrected for volatilization losses. We calculated the mass of contaminants that volatilize during the operational life of the landfill using the partitioning equations described in Section 3.1. The data in Table 3-2 are reported for various scenarios evaluated in the one-parameter high end sensitivity analysis (see Section 5). A density of 1.07 grams per cubic centimeter (g/cm^3) was used in the computation in Table 3-2.

As described in Section 3.1, the partitioning equations for land treatment units were used to generate the leachate concentration that is used as input to EPACMTP. In addition, the inputs common to both the partitioning equations and EPACMTP (including area of the waste management unit, site location, and infiltration rate) were fully synchronized in the probabilistic analyses in order to preserve mass balance and input parameter correlations. For the land treatment unit scenario, the leachate concentration given by the partitioning equations was held constant over a time period of 80 years. The value of 80 years was calculated based on the assumption that the active life of the land treatment unit is 40 years, and that after cessation of sludge application, the generation of leachate would continue for no more than 40 additional years. Based upon EPACMTP sensitivity analyses conducted for the Hazardous Waste Identification Rule (HWIR), water added to the water balance through the application of sludge-type wastes does not significantly impact the soil water balance over the modeling period of 10,000 years (U.S. EPA 1996a). Therefore, the net infiltration rate for a land treatment unit was assumed the same as the HELP-modeled ambient regional recharge rate (U.S. EPA 1997a and 1997c; U.S. EPA, 1994a and 1994b).

For both the landfill and land treatment unit scenarios, we evaluated groundwater discharge to surface water by calculating the total contaminant mass flux at the downgradient location that represents the intersection of the contaminant plume with the surface water body. We assumed that the surface water body fully penetrates the aquifer and the groundwater plume fully intersects the surface water body. We calculated the total contaminant mass flux to surface water by multiplying the groundwater flux with the net contaminant mass across the entire plume cross section. Appendix D.4 provides a complete description of the groundwater model.

3.6 Surface Water Fate and Transport (IEM)

EPA assumed that fish are exposed to waste constituents in surface water. Specifically, we assumed that fish are exposed to contaminants dissolved in the water column, contaminants sorbed to suspended solids in the water column, and contaminants associated with the bed sediment in the surface water body. The method used to estimate how management of chlorinated aliphatics wastewaters and wastewater treatment sludges impacts surface water is based on the indirect exposure methodology (IEM) presented in the "Addendum to Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions" (U.S. EPA 1993). The model accounts for six ways in which contaminants may enter the surface water body: (1) contaminants may be sorbed to eroded soils that enter the surface water body, (2) contaminants may be dissolved in runoff that enters the surface water body, (3) contaminants may be bound to airborne particles that are deposited on the surface water body, (4) vapor phase contaminants in air may be deposited on the surface water body in precipitation (that is, wet deposition of vapor phase contaminants), (5) vapor phase contaminants in air may enter the surface water body through direct diffusion from the air, and (6) contaminants in groundwater may discharge into the surface water body. The model also accounts for processes that remove contaminants from the surface water body including: (1) volatilization of contaminants that are dissolved in surface water, and (2) burial of contaminants in the sediment at the bottom of the surface water body. Appendix D.5 provides a complete description of the surface water model.

3.7 Indirect Exposure Equations

EPA uses a series of "indirect exposure equations" to quantify the concentrations of contaminants that pass from contaminated environmental media to the receptor indirectly. For example, contaminants that are transported in air may be deposited on plants or onto the soil where they may accumulate in forage, grain, silage or soil that subsequently is consumed by beef cattle and dairy cattle. Receptors the may ingest contaminated beef [1] and dairy products. Similarly, contaminants may be transported in groundwater to domestic wells where the

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[1] For some dioxin congeners, our analysis did not use the recommended factor for converting the milk biotransfer factor to a beef biotransfer factor (5.4) and, instead, conversion factors ranging from 3 to 8 were used in estimating the beef biotransfer factor. Correcting this error would result in less than a 1% difference in the TCDD TEQ concentrations calculated for beef.

groundwater is extracted and used for showering. In addition to the dermal exposure to shower water, the water vapor generated in the shower may be inhaled by the receptor. The indirect exposure equations allow EPA to calculate exposure point concentrations for these pathways and routes of exposure. The indirect exposure equations used by EPA to conduct the chlorinated aliphatics wastewater, EDC/VCM sludge, and methyl chloride sludge risk assessments are presented in Appendix E.

3.8 Evaluations of Environmental Fate and Transport (Special Cases of Mercury and Chromium)

This section presents a simplified approach for evaluating the fate and transport of mercury and chromium in the environment. Evaluation of mercury and chromium is technically complex because both mercury and chromium exist in the environment as more than one species, and each of the species has its own toxicity. We conducted our initial evaluations of mercury and chromium using the environmentally protective assumption that the most toxic form of the contaminant is the predominant species found at the point of exposure. We relied on the principle that if we could establish that risks and hazards due to mercury and chromium are not of concern for the most toxic forms of these metals, then further analysis that included the less toxic forms would not be necessary.

Mercury. Mercury exists in the environment in three different forms: elemental (free) mercury, inorganic mercury compounds (mercury that occurs as a compound with other inorganic elements), and organic mercury compounds (most commonly, methylmercury). Mercury was detected in one of the six chlorinated aliphatics wastewater samples at a concentration of 0.0008 mg/L. No data were available regarding the form(s) of mercury present in the wastewater. The environmental fate and transport of mercury is highly complex as described in the Mercury Report to Congress (U.S. EPA 1997b). Because the waste water data did not indicate that mercury is a prominent component of chlorinated aliphatics wastewaters, and because the current maximum contaminant level (MCL) for mercury is more than an order of magnitude greater than the sample concentration (the MCL is 0.005 mg/L), an environmentally protective screening approach was adopted to address mercury risk.[2] The purpose of the screening approach was to ensure that even a worst case scenario for mercury emissions from

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[2] Mercury is a major component of VCM-A watewaters and sludges; however, VCM-A wastewaters and sludges are not addressed in this analysis.

tanks would not be associated with unacceptable health risks. As described below, the screening approach was based on relatively protective assumptions regarding the dominant form of mercury in tanks and in the environment. These assumptions tend to maximize tank releases of mercury and subsequent uptake and accumulation of mercury in the food chain. The resulting risk estimates associated with mercury release from wastewaters should be considered to be at the extreme tail of the possible risk distribution.

EPA modeled the mercury in the wastewater tanks as elemental mercury. Because elemental mercury is highly volatile, we assumed that 100 percent of the mercury is released into the vapor phase, resulting in an upper bound for possible mercury emissions from tanks. The mercury species found in wastewater tanks are likely to include other forms of mercury such as inorganic mercury (for example, divalent mercury) and, depending upon the fraction of organic carbon and presence of microbial activity, possibly methylmercury and ethyl mercury. We calculated emission rates of elemental mercury from the tank using CHEMDAT8 and modeled downwind air dispersion of vapors using ISCST3. We based direct inhalation exposure on the predicted vapor concentration of elemental mercury at various distances from the emission source; we evaluated all other exposures as though the exclusive form of mercury was methylmercury, the most toxic form of mercury. In effect, we assumed that the form of mercury in contact with soil, vegetation, surface water, etc., is methylmercury.

Limiting the ingestion exposure to methylmercury (versus divalent mercury) is not strictly protective from the standpoint of plant uptake. The air-to-plant biotransfer factors for divalent mercury are higher than those for methylmercury (roughly by an order of magnitude) and, as a result, estimates of plant concentrations may be somewhat lower under the assumption that methylmercury is the dominant species at the point of contact with vegetation.[3] However, this effect is mitigated by the fact that the reference dose (RfD[4]) for divalent mercury is three times higher than the RfD for methylmercury, that is, the two assumptions essentially cancel each other. Thus, for the exposure pathways relevant to the farmer's ingestion of contaminated plant, beef, and dairy products, the risk estimates produced using methylmercury biotransfer factors are slightly lower than those that would be produced using divalent mercury (roughly by

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[3] The Mercury Report to Congress (U.S. EPA 1997b) points out that methylmercury typically constitutes only 1-3% of the mercury in soil and that a significant contribution to plant loading is driven by the mercury species in ambient air.

[4] Very simply, an RfD is the "acceptable dose" of a non-carcinogenic contaminant, such that the lower a contaminant's RfD, the lower the dose of the contaminant required to produce non-cancer health effects.

a factor of 3). In contrast, the risk estimates for the ingestion of contaminated fish are more than an order of magnitude higher for methylmercury than they would be if divalent mercury were assumed to be the predominant species. Recognizing these uncertainties, the screening results of mercury indicated that all hazard quotients were well below the target value of 1 (Appendix H.1) and, therefore, the potential for adverse effects associated with mercury releases from wastewater tanks is considered negligible.

Chromium. In the environment, chromium commonly occurs in one of two oxidation states: trivalent chromium (Cr^{+3}) and hexavalent chromium (Cr^{+6}). Although at high concentrations trivalent chromium may be associated with non-cancer health effects, in small amounts trivalent chromium is an essential nutrient that helps the body use sugar, protein, and fat (U.S. EPA 1998). Hexavalent chromium is much more toxic than trivalent chromium, such that EPA classifies hexavalent chromium as a known human carcinogen (due to inhalation exposure) and believes that ingestion of hexavalent chromium causes non-cancer health effects (U.S. EPA 1998). The RfD for trivalent chromium is 500 times greater than the RfD for hexavalent chromium.

In our analysis of the EDC/VCM wastewater treatment sludge, we modeled the fate and transport of chromium using a distribution coefficient (K_d) for total chromium (Cr_T), or the combined total of trivalent and hexavalent chromium. This is an accepted practice due to the unavailability of analytical results to calculate K_d values for a specific species of an element (in this case, hexavalent chromium). However, to ensure that the analysis is protective of human health, we assumed that exposure point concentrations of chromium were in the form of hexavalent chromium, thus maximizing the receptor's exposure to the most toxic chromium species. As discussed in Section 5, even given these protective assumptions, our estimates of the risks and hazards from exposure to hexavalent chromium in EDC/VCM sludge were not significant.

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4.0 EXPOSURE AND TOXICITY ASSESSMENTS

The following sections describe the exposure and toxicity assessments we conducted to support the chlorinated aliphatics risk assessment. Exposure assessment is the determination or estimation of the magnitude, frequency, duration, and route of exposure. The purpose of a toxicity assessment is to weigh available evidence regarding the potential for contaminants to cause adverse effects in exposed individuals. Our exposure and toxicity assessments are discussed in Sections 4.1 and 4.2, respectively.

4.1 Exposure Assessment

Exposure is the condition that occurs when a contaminant comes into contact with the outer boundary of the body, such as the skin, mouth, and nostrils. Once EPA establishes the concentrations of contaminants at the points of exposure, we can estimate the magnitude of each receptor’s exposure, or the dose of contaminant. The dose is the amount of the contaminant that crosses the outer boundary of the body and is available for absorption at internal exchange boundaries (lungs, gut, skin) (U.S. EPA 1992a). For example, given an exposure to a carcinogen through ingestion of contaminated drinking water, dose is a function of the concentration of the contaminant in drinking water (the exposure point concentration), as well as certain “exposure factors,” such as how much drinking water the receptor consumes each day (the intake rate), the number of years the receptor is exposed to contaminated drinking water (the exposure duration), how often the receptor is exposed to contaminated drinking water (the exposure frequency), the body weight of the receptor, and the period of time over which the dose is averaged.

EPA’s primary source of exposure factors is the *Exposure Factors Handbook* published by EPA in August 1997 (U.S. EPA 1997a). The *Exposure Factors Handbook* is the data source for most human exposure model inputs. It summarizes data on human behaviors and characteristics related to human exposure from relevant key studies and provides recommendations and associated confidence estimates on the values of exposure factors. EPA carefully reviewed and evaluated data quality before inclusion in the *Exposure Factors Handbook.* EPA’s evaluation criteria included peer review, reproducibility, pertinence to the United States, currency, adequacy of the data collection period, validity of the approach, representativeness of the population, characterization of the variability, lack of bias in study design, and measurement error (U.S. EPA 1997a). For many factors, percentile data that can be used to develop distributions are provided in the handbook.

For deterministic analyses, EPA selected high end and central tendency values for each of the exposure factors. The high end values typically were set at 90th percentile values and the central tendency values typically were set at 50th percentile values. For probabilistic risk analyses, EPA developed probability distribution functions (PDFs) for each of the exposure factors from the values in the *Exposure Factors Handbook*. Appendix K presents the exposure factors that we used in our deterministic and probabilistic risk analyses.

For most variables for which PDFs were developed, *Exposure Factors Handbook* data were analyzed to fit selected parametric models. *Exposure Factors Handbook* data include sample sizes and estimates of the following parameters for specific receptor types and age groups: mean, standard deviation, standard error, and percentiles corresponding to a subset of the following probabilities–0.01, 0.02, 0.05, 0.10, 0.15, 0.25, 0.50, 0.75, 0.85, 0.90, 0.95, 0.98, 0.99, and 1.00. We used maximum likelihood estimation to fit selected parametric models (gamma, lognormal, Weibull, and generalized gamma) to the exposure factor data. The chi-square measure of goodness of fit then was used to choose the best distribution. In addition, parameter uncertainty information (that is, for mean, standard deviation, scale, and shape) was derived using the asymptotic normality of the maximum likelihood estimate or a regression approach. These data were used in Monte Carlo exposure modeling for the probabilistic risk assessment, as discussed in Appendix F.2.

Probabilistic risk analyses involve “sampling” values from PDFs and using the values to calculate risk. In some cases distributions are infinite, and there is a probability, although very small, that very large or very small values might be selected from the distributions. EPA acknowledges that for many exposure parameters, selecting extremely large or extremely small values is unrealistic (for example, the range of adult body weights is not infinite), and that maximum and minimum values must be imposed on the distributions. In a number of cases, EPA does not know the maximum and minimum values for certain exposure factors. For example, although surveys may ascertain body weights for a large sample of individuals, it is very unlikely that the individual in the population with the highest body weight was part of that sample. In these cases we developed maxima and minima using a combination of available data and professional judgement. The following sections describe the rationale we used to select the central tendency and high end values for the deterministic analysis, the maximum and minimum exposure parameter values that we used in the probabilistic analyses, and the parametric models (gamma, lognormal, and Weibull) for those exposure factors that were varied in the probabilistic analysis.

4.1.1 Intake Rates

This section presents the basis for the intake rates that we used for soil, food items, and drinking water. Both adult and child intake rates for water, soil, and food items are derived from data in the *Exposure Factors Handbook* (U.S. EPA 1997a).

The *Exposure Factors Handbook* reports exposure factors for child receptors for various age ranges. For the deterministic analysis, we calculated time-weighted average child exposure factors for children between the ages of 1 and 19 by developing intake rates specific to the four age groups presented in the *Exposure Factors Handbook*: age 1-2; age 3-5; age 6-11; and age 12-19. For the probabilistic analysis described in Appendix F, we combined the two youngest age groups and developed input data for three child cohorts: age 1-5 (Child 1), age 6-11 (Child 2), and age 12-19 (Child 3). These three child cohorts were developed to make the most appropriate use of available data and to be consistent with other EPA initiatives in which child risks are being evaluated. The intake rates in the *Exposure Factors Handbook*, expressed on a gram (or Liter) per kilogram body weight per day (g/kg/d) basis, were multiplied by the number of years in the age range, summed together, and divided by the total number of years of exposure. Intakes that were not expressed on a body weight basis (soil ingestion and inhalation) were divided by the average body weights for the age ranges over the exposure period. Tables 4-1 and 4-2 present the central tendency and high end intake rates, respectively, used in the deterministic analysis of child risks.

For the probabilistic analyses, we set the maximum intake rates for food items at the 100th percentile value of the distribution reported in the *Exposure Factors Handbook*. We did not establish any minimum intake values. The 100th percentile values represent maximum values reported in the surveys and provide a reasonable limit on intake rates. Although intake rates for some individuals could be higher than these rates, such individuals are expected to represent a very small fraction of the population and their intake rates are unlikely to impact the risk results for the percentiles of the Monte Carlo distribution that are the focus of EPA's concern.

Table 4-1. Time-weighted Average (TWA) Intake Rates for Central Tendency Child Receptor

Parameter	Units	Data source[a]	Age Group 1-2	Age Group 3-5	Age Group 6-11	Age Group 12-19	Calculated TWA	TWA Units	Comments
Soil ingestion rate	kg/d	Table 4-23	0.0001	0.0001	0.00005	0.00005	2.70E-06	kg/kg/d	
Drinking water ingestion rate	L/kg/d	Tables 3-7 & 3-30	0.0305			0.0163	2.38E-02	L/kg/d	ages 1 - 10 and 11 - 19
Fruit ingestion rate	g DW/kg/d	Table 13-61	0.432	0.271	0.165	0.0907	1.79E-04	kg DW/kg/d	WW concentrations in EFH converted to DW
Vegetable ingestion rate	g DW/kg/d	Table 13-63	0.166	0.102	0.0566	0.0577	7.58E-05	kg DW/kg/d	WW concentrations in EFH converted to DW
Root ingestion rate	g WW/kg/d	Table 13-65	0.92	0.462	0.523	0.565	5.73E-04	kg WW/kg/d	
Root ingestion rate	g DW/kg/d	calculated	0.131	0.066	0.074	0.080	8.16E-05	kg DW/kg/d	WW concentrations in EFH converted to DW
Dairy ingestion rate	g WW/kg/d	Table 11-2	23.48	19.52	11.88	5.29	1.15E-02	kg WW/kg/d	
Dairy ingestion rate	g DW/kg/d	calculated	5.635	4.685	2.851	1.270	2.77E-03	kg DW/kg/d	WW concentrations in EFH converted to DW
Beef ingestion rate	g WW/kg/d	Tables 11-3 & 13-36	1.339	1.162	2.11	1.51	1.63E-03	kg WW/kg/d	
Beef ingestion rate	g DW/kg/d	calculated	0.380	0.330	0.599	0.429	4.62E-04	kg DW/kg/d	WW concentrations in EFH converted to DW
Inhalation rate	m^3/d	Myers et al. 1998		5.4	8.6	12	3.15E-01	m^2/kg/d	ages 0 - 3, 4 - 10, 11 - 18
Average body weight for soil ingestion	kg	Tables 7-6 & 7-7	12.15	17.13	29.48	56.61		kg	used to calculate TWA for soil ingestion
Average body weight for inhalation	kg	Tables 7-6 & 7-7		12.13	24.91	53.55		kg	used to calculate TWA for inhalation

DW = dry weight
WW = wet weight

[a] Tables are found in the *Exposure Factors Handbook (EFH)*

Table 4-2. Time-weighted Average (TWA) Intake Rates for High End Child Receptor

Parameter	Units	Data source[a]	Age Group 1-2	Age Group 3-5	Age Group 6-11	Age Group 12-19	Calculated TWA	TWA Units	Comments
Soil ingestion rate	kg/d	Table 4-23	0.0004	0.0004	0.00005	0.00005	8.06E-06	kg/kg/d	
Fruit ingestion rate	g DW/kg/d	Table 13-63	0.942	0.305	0.2834	0.2068	3.24E-04	kg DW/kg/d	WW concentrations in EFH converted to DW
Vegetable ingestion rate	g DW/kg/d	Table 13-65	10.4	4.73	5.59	3.32	5.00E-03	kg DW/kg/d	WW concentrations in EFH converted to DW
Root ingestion rate	g WW/kg/d	calculated	1.481	0.674	0.796	0.473	7.13E-04	kg WW/kg/d	
Root ingestion rate	g DW/kg/d	Table 11-2	45.72	39.54	25.38	12.75	2.44E-02	kg DW/kg/d	WW concentrations in EFH converted to DW
Dairy ingestion rate	g WW/kg/d	calculated	10.973	9.490	6.091	3.060	5.87E-03	kg WW/kg/d	
Dairy ingestion rate	g DW/kg/d	Tables 11-3 & 13-36	2.783	3.163	11.4	3.53	5.88E-03	kg DW/kg/d	WW concentrations in EFH converted to DW
Beef ingestion rate	g WW/kg/d	calculated	0.790	0.898	3.238	1.003	1.67E-03	kg WW/kg/d	
Beef ingestion rate	g DW/kg/d	Myers et al., 1998, Table 5-2		12.4	12.6	18.3	5.45E-01	kg DW/kg/d	WW concentrations in EFH converted to DW
Inhalation rate	m^3/d	Tables 7-6 & 7-7	12.15	17.13	29.48	56.61		m^2/kg/d	ages 0 - 3, 4 - 10, 11 - 18
Average body weight for soil ingestion	kg	Tables 7-6 & 7-7		12.13	24.91	53.55		kg	used to calculate TWA for soil ingestion
Average body weight for inhalation	kg	Tables 7-6 & 7-7		12.13	24.91	53.55		kg	used to calculate TWA for inhalation

DW = dry weight
WW = wet weight

[a] Tables are found in the *Exposure Factors Handbook (EFH)*

Soil Ingestion. Ingestion of contaminated soil is a pathway common to all receptors. Only three studies have investigated soil ingestion by adults. We used 50 mg/d as the soil ingestion rate for the adult receptors for both the deterministic and the probabilistic analyses. A high end soil ingestion rate is not available for adults because of high uncertainty. For the child receptor, we used ingestion rates of 100 mg/d (central tendency) and 400 mg/d (high end) for children at age 5 and younger. The adult soil ingestion rate was used for children older than age 5.

In the probabilistic analysis, soil intake rate is a random variable only for children. For children, Tables 4-9 and 4-10 of the *Exposure Factors Handbook* provide a range of soil ingestion rates based on a study of 64 children ages 1 to 4. Table 4-10 provides a projected intake rate of 1 to ~2,300 mg/d over a 365-day study period (U.S. EPA 1997a). We used a value of 2,300 mg/d as the maximum soil intake rate for children. Although the *Exposure Factors Handbook* recommends a 10,000 mg/d soil intake rate for a pica child, this estimate is for acute exposure only. Over the long-term, the 2,300 mg/d estimate is considered more appropriate, and the study on which this value is based included a pica child. Table 4-9 of the *Exposure Factors Handbook* presents an overall maximum soil intake rate of 7,700 mg/d based on the average of all children in the study and 8 different tracers. This estimate was not used as a maximum value because the data correspond to only an 8-day study period.

Fruits and Vegetables Ingestion**.** Ingestion of contaminated home-grown fruits and vegetables is a potential pathway of exposure to adult farmers and home gardeners and their children. Consumption rate data of home-grown exposed fruit, exposed vegetables, and root vegetables by these receptors were obtained from the *Exposure Factors Handbook*. Examples of exposed fruits are apples, peaches, pears, and berries. Above-ground exposed vegetables include tomatoes, green leafy vegetables (for example, lettuce, cabbage, kale), cucumber, summer squash, peppers, broccoli, okra, and snap beans. Common root vegetables include carrots, onions, potatoes, and beets.

Above-ground exposed fruits and vegetables may be contaminated by air deposition directly onto the plant (dry deposition of particulate-bound contaminants), vapor phase transfer of contaminants into the plant, and contaminant uptake through the roots. Contamination of root vegetables occurs by only the latter process. Plant uptake factors (Appendix C) may be based on the dry weight or wet weight of plants; therefore, intake rates are provided on either a dry weight and wet weight basis to be consistent with the plant uptake factors. For example, intakes of root vegetables are expressed as dry weights for most metals and wet weights for organics. Similarly, beef and dairy intakes are based on dry weights for most metals but wet weights for other constituents (Appendix K). We derived dry weight intake rates by multiplying the wet weight rates by the percent moisture content of selected fruits and vegetables as reported in the *Exposure Factors Handbook*. Based on recommendations in the *Exposure Factors Handbook*, we used an average moisture content of 0.85 for exposed fruits, 0.86 for root vegetables, and 0.91 for exposed vegetables.

Although farmers and home gardeners grow much of their food, they generally do not grow all of their food. Therefore, we considered the fraction of their diet that may be contaminated. Specifically, the *Exposure Factors Handbook* provides recommendations on the percent of the total diet of gardeners and farmers that is home grown. These factors are listed in Appendix K. We assumed that residents without home gardens purchase all of their food from supermarkets and that none of the produce or beef and dairy products originate from areas in the immediate vicinity of a chlorinated aliphatics facility.

Beef and Dairy Ingestion. The farmer is the only receptor who is assumed to ingest beef and dairy products from cattle raised in the immediate vicinity of a chlorinated aliphatics facility. Appendix K provides beef and dairy ingestion rates on both a dry weight and wet weight basis. As with fruits and vegetables, it was necessary to consider the fraction of the total beef and dairy in the farmer's diet that consists of products raised in the immediate vicinity of a chlorinated aliphatics facility. These factors are listed in Appendix K.

Fish ingestion. Fish ingestion rates were based on a recreational angler who catches and eats some fish from a stream impacted by contaminants released from the waste management units. The *Exposure Factors Handbook* currently recommends 8 g/d and 25 g/d as central tendency and high end intake factors for recreational freshwater anglers. These values were derived as averages from three studies. The high end value in this case is a 95th percentile value. We derived fish ingestion rates for the probabilistic analysis from Table 10-64 of the *Exposure Factors Handbook* (U.S. EPA 1997a). This table provides data (in g/d) for adult freshwater anglers in Maine. The fraction of fish intake that is home caught is 0.325 for households that fish (Table 13-71, U.S. EPA 1997a).

Drinking Water Ingestion. Use of groundwater from a contaminated well downgradient from a waste management unit is a pathway common to all receptors. For the deterministic analysis, the groundwater ingestion rate for adult receptors was set at 1.4 L/d, based on Table 3-30 of the *Exposure Factors Handbook* (U.S. EPA 1997a). For children, a central tendency, age-adjusted, time-weighted average ingestion rate of 0.02 L/kg/d was used (Table 4-1). The sensitivity analysis for input parameters for the groundwater ingestion pathway (Appendix D.4) indicated that the ingestion rate was not one of the two most sensitive parameters; consequently, the high end groundwater ingestion rate is not shown in Table 4-2. For the probabilistic analysis, we obtained drinking water intake data from Table 3-6 of the *Exposure Factors Handbook*. Data (in mL/d) are provided by age group. For the probabilistic analysis, we set the maximum drinking water intake rate at twice the 99th percentile value, which for adults is about 8 L/d, or 0.06 L/kg-d. We did not set a minimum value since there are some individuals who drink only bottled water.

4.1.2 Inhalation Rates

The *Exposure Factors Handbook* reports recommended inhalation values by age, gender, activity pattern, and outdoor workers; however, it does not provide high end values in most cases. Myers et al. (1998) conducted a study for EPA to evaluate distributions of data from the *Exposure Factors Handbook*. Based on this study, we set the central tendency and high end inhalation rates for adults at the 50th percentile (0.67 m^3/hr) and 90th percentile (0.98 m^3/hr) inhalation rates for adult males age 18 to 60 (Myers et al. 1998). As presented in Tables 4-1 and 4-2, we calculated age-adjusted, time-weighted central tendency and high end inhalation rates for children.

For the probabilistic analysis, we estimated adult inhalation rates by fitting a lognormal distribution to inhalation data analyzed by Myers et al. (1998). We used a population mean of 13.3 m^3/d and a standard deviation of 3.99 m^3/d to estimate inhalation rates. We set the maximum inhalation rate at twice the average 99th percentile values. We set the minimum value at one half the average resting inhalation rate. Based largely on professional judgment, we used a minimum inhalation rate of 5 m^3/d and a maximum inhalation rate of 51 m^3/d (derived from data from table 5-6 of U.S. EPA 1997a and Myers et al. 1998, respectively). We considered the average resting inhalation rate a low end rate, one that might apply to sedentary individuals. Dividing this rate by 2 provides a reasonable estimate of a minimum inhalation rate for the population. The maximum inhalation rate is derived from the 99th percentile of average adult daily inhalation rates and includes all activity levels. Multiplying the 99th percentile of average daily inhalation rates by 2 provides an estimate of the maximum value; this may overestimate the maximum inhalation rate because it is unlikely that someone would breathe at a high activity level for 24 hours per day.

4.1.3 Body Weights

We used only central tendency values for body weights in the deterministic analysis. The recommended average value for adults is 70 kg (U.S. EPA 1997a). For children, the intake rates reported in the *Exposure Factors Handbook* are expressed as unit intake per kg body weight per day (Tables 4-1 and 4-2). As a result, children's body weights were not needed for the deterministic or probabilistic analyses. For the probabilistic analysis, body weights for adults were varied based on a lognormal distribution, with a mean of 71.2 kg and a standard deviation of 13.3 kg. Percentile data from the *Exposure Factors Handbook* were combined for all adult ages and genders and were used as the basis of fitting a lognormal distribution. The maximum value was set at twice the 95th percentile body weight for men and the minimum was set at half the 5th percentile body weight for women, producing a range of 24 kg to 205 kg for adult body weights.

4.1.4 Dermal Factors

The dermal exposure to contaminants that occurs in the shower was evaluated only for adults. We assumed that children under the age of 12 are more inclined to take baths rather than showers. In addition, the skin permeability constants developed by EPA may not be appropriate for children primarily because of the differences in the surface-to-volume ratio between children and adults and, to a lesser degree, because of the differences in physiological characteristics of skin (U.S. EPA 1992b). Consequently, this pathway was not assumed relevant for children.

Central tendency values for adults used for the deterministic analysis came from the *Exposure Factors Handbook* (U.S. EPA 1997a) and U.S. EPA (1992b). We used an average adult skin surface area of 20,000 cm^2 (U.S. EPA 1992b), an average shower duration of 10 minutes (U.S. EPA 1997a), and contaminant-specific skin permeability constants (U.S. EPA 1992b). Dermal exposure factors were not highly sensitive parameters; therefore, we did not evaluate high end values in our analysis. Because the results of our deterministic analysis indicated that the risks from dermal exposures were insignificant, we did not evaluate risks via the dermal route of exposure in our probabilistic analyses.

4.1.5 Exposure Duration, Exposure Frequency, Lifetime, and Averaging Time

Exposure duration refers to the amount of time that a receptor is exposed to a contaminant source. For this risk analyses, we assume that exposure duration corresponds to the receptor's residence time in the same house. Exposure durations used in this risk assessment are shown in Appendix K. For the deterministic analysis, we used residence times of 9 years (50th percentile) and 30 years (95th percentile) for the resident, home gardener, and fisher as recommended in the *Exposure Factors Handbook*. We based residence times for the farmer on the 50th percentile (10 years) and 90th percentile (48.3 years) values, the residence times presented for farm populations. Residence times for the child receptors (7.3 and 15.3 years) were based on averaging the 50th percentile and 90th percentile values reported for children at ages 3, 6, 9, 12, 15, and 18. One exception was made for soil ingestion for children. The statistics for soil ingestion rates for children are based on ages less than 7; therefore, we used 6 years as the central tendency exposure duration when evaluating soil ingestion by children.

Exposure frequency is frequency at which the receptor is exposed to the contaminated source during the exposure duration. We assume that all receptors are exposed to the contaminant source 350 days per year. This value is based on an assumption that individuals are away from their homes (for example, on vacation) approximately 2 weeks out of the year.

Averaging time is the period of time over which a receptor's dose is averaged. When evaluating carcinogens, dose is averaged over the lifetime of the individual, assumed to be 70 years. When evaluating non-carcinogens, we average dose over the period of exposure since noncancer effects may become evident during less-than-lifetime exposure durations if toxic thresholds are exceeded.[1]

4.1.6 Background Exposures

In certain cases, EPA performs a risk assessment on wastes that contain contaminants that also are present in the environment as a result of both natural processes and anthropogenic activities. Under these circumstances, receptors potentially receive a

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[1] EPA recognizes that the benchmarks for noncancer effects were developed for exposures over the lifetime of the individual. However, noncancer benchmarks for subchronic exposures are currently unavailable from EPA-approved sources.

"background" exposure that may be greater than the exposure resulting from release of contaminants from the waste. Section 5 discusses significant risk results for arsenic and dioxin, two constituents to which individuals are likely to receive background exposure. Tables 4-3 and 4-4 provide 1) background contaminant intakes for arsenic and dioxin and 2) background concentrations of arsenic in groundwater, and dioxin in soil, food items, and air. The tables compare the background intakes and concentrations to corresponding values for EDC/VCM sludges and chlorinated aliphatics wastewaters.

4.2 Toxicity Assessment

The purpose of the dose-response assessment is to determine the most sensitive health effects associated with the COPCs and to attempt to express the relationship between dose and effect in quantitative terms. These quantitative terms are known as health benchmarks and include:

- Cancer slope factors (CSFs) for oral exposure carcinogenic contaminants;
- Reference doses (RfDs) for oral exposure to noncarcinogenic contaminants;
- Inhalation CSFs, derived from Unit Risk Factors (URFs), for inhalation exposure to carcinogenic contaminants;
- RfCs for inhalation exposure to noncarcinogenic contaminants; and
- Toxicity Equivalency Factors (TEFs) to express the toxicity of specific dioxin congeners in terms of the toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD).

Health benchmark values are available from a number of sources. For the chlorinated aliphatics wastewater, EDC/VCM sludge, and methyl chloride sludge risk assessments, EPA established an order of preference for the sources of health benchmarks. The order of preference is as follows (from most preferred to least preferred): (1) the Integrated Risk Information System (IRIS) online database of verified health benchmarks (U.S. EPA 1998); (2) the Health Effects Assessment Summary Tables (HEAST; U.S. EPA 1997b); (3) EPA's National Center for Environmental Assessment (NCEA) provisional values, and (4) benchmarks developed by the California Environmental Protection Agency (CalEPA 1997). The specific health benchmarks used in the analysis, and their sources, are presented in Appendix C.

Table 4-3. Comparison of Concentrations and Intake Rates of Arsenic from EDC/VCM Wastewater Treatment Sludges to Background Levels of Exposure

Estimates of Arsenic Exposure Point Concentrations in Groundwater and Arsenic Intake Rates EDC/VCM Wastewater Treatment Sludges	
High End and Central Tendency arsenic exposure point concentrations for groundwater (concentration of groundwater in a receptor well)	***EDC/VCM Landfill*** High End: 1.4 µg/L Central Tendency: 0.2 µg/L ***EDC/VCM Land Treatment Unit*** High End: 0.5 µg/L Central Tendency: 0.2 µg/L
High End and Central Tendency arsenic intake (based on ingestion rate of 1.4 liters of water per day)	***EDC/VCM Landfill*** High End: 2 µg/d Central Tendency: 0.3 µg/d ***EDC/VCM Land Treatment Unit*** High End: 0.7 µg/d Central Tendency: 0.3 µg/d
Background Arsenic Concentration in Groundwater and Arsenic Intake Rates	
Maximum Contaminant Level (MCL, allowable concentration in public drinking water systems)	50 µg/L [Note: This value is currently being reevaluated.]
World Health Organization Guideline for drinking water	10 µg/L
Concentration of arsenic in U.S. groundwaters, based on analysis of samples from 30,000 locations (Welch 1999)	Median: approximately 1 to 5 µg/L (dependent on geographic location) 90th percentile: approximately 10 µg/L
Air Force Background Levels of Arsenic in Groundwater (Hunter 1998) • 2996 wells sampled (sample size = 7259) • Samples collected at 107 Air Force Bases	Detection Frequency: 32% Median concentration: Not Detected 95% upper tolerance limit: 44 µg/L 99% upper tolerance limit: 171 µg/L
Estimated dietary intake of inorganic arsenic (U.S. EPA 1997c)	14 µg/d
1992 EPA projections of the number of drinking water systems with groundwater sources impacted by various arsenic MCL options (U.S. EPA 1997d)	• Approximately 150 CWS and NCNTWS with ground water sources would exceed 50 µg/L • Approximately 4,500 CWS and NCNTWS with ground water sources would exceed 5 µg/L.

CWS = community water systems
NCNTWS = non-community, non-transient water systems

Table 4-4. Comparison of Background Concentrations and Intake Rates of Dioxin to Concentrations and Intake Rates Associated with Chlorinated Aliphatics Wastes

	Average Background		**Chlorinated Aliphatics Wastes (Adult Farmer Scenario)**			
			Chlorinated Aliphatics Wastewater		**EDC/VCM Land Treatment Unit**	
Dioxin Source	**Source: “Estimating Exposure to Dioxin-Like Compounds” (U.S. EPA 1994a)**	**Source: “Dioxin Factsheet: Background Environmental Levels and Exposure” (U.S. EPA 1999a)**	**Central Tendency**	**High End**	**Central Tendency**	**High End**
TEQ in soil	8 ng/kg (n=95)	Rural: 1 to 6 ng/kg Urban: 7 to 20 ng/kg	0.0011 ng/kg	0.04 ng/kg	5.4 ng/kg	61 ng/kg
TEQ in beef	0.29 ng/kg [0.48 ng/kg]* (n=14)	0.17 ng/kg	0.011 ng/kg	0.75 ng/kg	0.13 ng/kg	1.4 ng/kg
TEQ in dairy	***Milk*** 0 ng/kg [0.07 ng/kg] (n=2) ***Other Dairy*** 0.35 ng/kg [0.36 ng/kg] (n=5) ***Egg*** 0.0004 ng/kg [0.13 ng/kg] (n=8)	***Milk*** 0.03 ng/kg ***Other Dairy*** 0.1 ng/kg ***Egg*** Not Reported	0.0032 ng/kg	0.20 ng/kg	0.030 ng/kg	0.32 ng/kg
TEQ in air	0.0949 pg/m^3 (n=84)	Rural: 0.002 to 0.02 pg/m^3 Urban: 0.02 to 0.2 pg/m^3	0.00032 pg/m^3	0.013 pg/m^3	NA	NA
Dioxin Intake	mean: 0.062 ng/d [0.119 ng/d]	0.040 ng/kg-day due to dioxins/furans	0.0012 ng/d*	0.074 ng/d*	0.012 ng/d*	0.13 ng/d*

* Based on central tendency intake rates (and stated assumptions regarding the fraction of a farmer’s food that is home-grown).

ED = Exposure Duration
n = number of samples
NA = not calculated
[ ] = Values in [brackets] based on ND = 0.5DL

4.2.1 Noncarcinogens

EPA uses RfDs and RfCs to evaluate noncancer effects for ingestion and inhalation exposures, respectively, and defines RfD and RfC as “an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime” (U.S. EPA 1989b). RfDs are expressed in milligrams of chemical intake per kilogram body weight per day (mg/kg/d) and RfCs are expressed as milligrams of chemical per cubic meter of air (mg/m^3).[2]

The RfD and RfC are the primary benchmarks used to evaluate noncarcinogenic hazards posed by environmental exposures to chemicals and are based on the “threshold” approach, which is based on the theory that there is a “safe” exposure level (the threshold) that must be exceeded before a toxic effect occurs. RfDs and RfCs do not provide true dose-response information in that they are estimates of an exposure level or concentration that is believed to be below the threshold level or no observed adverse effects level (NOAEL). It also is important to understand that all RfDs are not necessarily equivalent expressions of toxicity. The degree of uncertainty and confidence levels in the various RfDs vary a great deal and are based on different toxic effects. RfDs and RfCs that have been verified by an intra-Agency workgroup are listed in IRIS.

RfDs and RfCs are derived from the highest NOAEL for the most sensitive effect identified in human epidemiological studies or from subchronic or chronic studies in laboratory animals. If a NOAEL is not identified in any of the available studies, the lowest observed adverse effect level (LOAEL) is used. If the studies reported dose levels as parts per million (ppm) in the diet or water, the dose levels are converted to mg/kg/d based on the consumption level and body weights of the test subjects. It is generally assumed that dose levels expressed on a mg/kg/d basis are equivalent in humans and animals; therefore, dose adjustments are not necessary unless chemical-specific pharmacokinetic data indicate that a dose adjustment is appropriate. NOAELs and LOAELs are adjusted ($NOAEL_{adj}$ or $LOAEL_{adj}$) for inhalation exposure protocols that are not continuous (i.e., less than 7 days per week or 24 hours per

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[2] EPA does not develop an expression of dose where we use Reference Concentrations (RfCs) to estimate noncancer hazard for the inhalation exposure route. In this situation, EPA calculates noncancer hazard from concentration of the contaminant in air and the RfC, without considering exposure factors (inhalation rate, body weight) other than those inherent in the RfC.

day). Differences in respiratory rates and respiratory physiology between humans and laboratory animals are well recognized; therefore, NOAELs and LOAELs identified from inhalation studies are converted to the human equivalent concentration ($\text{NOAEL}_{\text{HEC}}$ or $\text{LOAEL}_{\text{HEC}}$) before deriving the RfC. The RfC methodology is described in detail in U.S. EPA (1994b).

Once an appropriate NOAEL or LOAEL has been identified, the characteristics and the quality of the database are examined and the NOAEL or LOAEL is divided by uncertainty factors and modifying factors to derive the RfD or RfC. Factors of 10 are most commonly used as uncertainty factors (Table 4-5). An uncertainty factor of 3 may be used if appropriate pharmacokinetic data (or a model) are available. The default value for the modifying factor is 1. All uncertainty factors and modifying factors are multiplied together to derive the total uncertainty factor, with 3,000 being the maximum recommended value (U.S. EPA 1994b).

Table 4-5. Standard Uncertainty and Modifying Factors

Description	Rationale	Values
Interspecies variation	Extrapolation from animal data to humans	3 to 10
Intraspecies variation	Accounts for sensitive individuals (e.g., children, elderly, asthmatics)	1 to 10
Subchronic to chronic	A subchronic study was used to derive a chronic RfD or RfC	3 to 10
LOAEL to NOAEL	A LOAEL was used instead of a NOAEL	1 to 10
Incomplete database	Lack of data for critical endpoints (e.g., reproductive and developmental)	1 to 10
Modifying factor	Accounts for additional uncertainties per professional judgment	1 to 10

Most toxicity values are based on the administered dose rather than the amount absorbed. Therefore, in some cases, it is necessary to adjust toxicity values to ensure that they match the exposure estimates. In other words, exposures may be based on an “administered” or “applied” dose, or they may be based on an absorbed dose. Typically, ingestion exposure estimates are based on an administered dose and dermal exposure estimates consider the amount absorbed through the skin. EPA has not developed toxicity values for dermal exposures. Instead, RfDs and CSFs are adjusted, if necessary, to represent an absorbed dose. This is accomplished by multiplying the RfD by the measured or predicted absorption efficiency of the chemical from the gastrointestinal tract or by dividing the CSF by the absorption efficiency (U.S. EPA 1989a). The following default oral absorption efficiencies were used: 80% for VOCs, 50% for SVOCs, and 20% for metals (U.S. EPA 1995). As noted in Section 4.1.4, suitable skin permeability constants for dermal exposure for children are currently unavailable.

4.2.2 Carcinogens

Measures of carcinogenic potency, the CSFs and URFs, may be derived from a number of statistically- and/or biologically-based models. Traditionally, the linearized multistage model has been the default model for extrapolating cancer slope factors for low doses; however, other models also have been used. Although several models may provide a good fit to the experimental data, the slope factors at low doses may be different by up to several orders of magnitude depending upon which model is used. EPA's proposed cancer risk guidelines propose significant changes to the default methodology (U.S. EPA 1996). Although the new methodology has been used to develop some benchmarks listed in IRIS (for example, for PCBs), all of the cancer benchmarks used in this report are based on the linearized multistage model.

CSFs and URFs are used to evaluate cancer risks for ingestion and inhalation exposures, respectively. Unlike RfDs and RfCs, CSFs and URFs do not represent "safe" exposure levels, rather, they are derived mathematically as the 95 percent upper confidence limit of the slope of the linear portion of the dose-response curve, that is, they relate levels of exposure with a probability of effect or risk. The CSF is expressed in units of $(mg/kg/d)^{-1}$ and the URF is expressed in units of $(\mu g/m^3)^{-1}$. For this risk assessment, we converted URFs into inhalation CSFs for the purpose of calculating risk. Appendix E provides the equation used to convert URFs to inhalation CSFs.

4.2.3 Dioxin TEFs

EPA assigns the 17 dioxin and furan congeners individual toxicity equivalency factors (TEFs). TEFs are estimates of the toxicity of dioxin-like compounds relative to the toxicity of 2,3,7,8–TCDD, which is assigned a TEF of 1.0. There currently are at least two TEF schemes used internationally by scientists. We used the TEFs identified as the I-TEFs (International-TEFs) to conduct the chlorinated aliphatics risk assessment because, until very recently, this is the TEF scheme EPA used by scientists for the last 10 years (U.S. EPA 1989b). The I-TEFs are presented in Table 4-6. The I-TEFs are based on a limited database of in vivo and in vitro toxicity testing (U.S. EPA 1989b). EPA currently is in the process of developing a recommendation to use a newer TEF scheme established by the World Health Organization (WHO) (Van den Berg et al. 1998). As shown in Table 4-6, the WHO-TEFs differ from the I-TEFs for only three dioxin and furan compounds: 1,2,3,7,8-PeCDD (pentachlorodibenzo-p-dioxin), OCDD (octachlorodibenzo-p-dioxin), and OCDF (octachlorodibenzofuran). 1,2,3,7,8-PeCDD was not detected in dedicated chlorinated aliphatic wastewaters, dedicated EDC/VCM sludges, or methyl chloride sludges. Consequently, the difference in the I-TEF and the WHO-TEF for 1,2,3,7,8-PeCDD has no impact on the results of the risk analyses presented in Section 5. However, because of the TEF differences for OCDD and OCDF, the decision to use the I-TEFs rather than the WHO-TEFs may result in differences in the TCDD TEQ concentrations calculated for a given chlorinated aliphatics waste sample. Nevertheless, because OCDD and OCDF contribute a small portion to the actual risk attributable to dioxin compounds, the decision to use either the I-TEFs or the WHO-TEFs has negligible impact on the overall risk results. Appendix H provides separate risk results for each of the dioxin congeners detected in the wastewaters and sludges evaluated.

4.2.4 Contaminants For Which No Toxicity Values Are Available

EPA was unable to obtain toxicity values for three contaminants that were COPCs in this risk assessment. Although these contaminants were carried through EPA's exposure assessment, the lack of toxicity values compelled us to drop them from the risk assessment. The contaminants and the wastes in which they were detected are discussed below.

Aluminum. Aluminum was detected in the following wastes:

- Chlorinated aliphatics wastewater (maximum concentration of 44.6 mg/L);
- EDC/VCM sludge (maximum total concentration of 29,500 mg/kg); and
- Methyl chloride sludge (maximum total concentration of 1,930 mg/kg; maximum TCLP concentration of 2.4 mg/L).

Hunter (1998) evaluated an Air Force database of background soil and groundwater data. He reported that the 95% upper tolerance limit of aluminum background concentrations in groundwater was 44 mg/L and in soils was 23,700 mg/kg. The concentrations of aluminum

Table 4-6 . Toxicity Equivalency Factors (TEFs) for Dioxin Compounds

Compound	I-TEF	WHO-TEF
2,3,7,8-TCDD	1	same
1,2,3,4,5,7,8,9-OCDD	0.001	0.0001
1,2,3,7,8,9-HxCDD	0.1	same
1,2,3,4,6,7,8-HpCDD	0.01	same
1,2,3,4,6,7,8,9-OCDF	0.001	0.0001
1,2,3,4,7,8-HxCDD,	0.1	same
1,2,3,7,8-PeCDD,	0.5	1
2,3,7,8-TCDF	0.1	same
1,2,3,4,7,8,9-HpCDF	0.01	same
2,3,4,7,8-PeCDF	0.5	same
1,2,3,7,8-PeCDF	0.05	same
1,2,3,6,7,8-HxCDF	0.1	same
1,2,3,6,7,8-HxCDD	0.1	same
2,3,4,6,7,8-HxCDF	0.1	same
1,2,3,4,6,7,8-HpCDF	0.01	same
1,2,3,4,7,8-HxCDF	0.1	same
1,2,3,7,8,9-HxCDF	0.1	same

detected in chlorinated aliphatics wastes are within the range of reported background concentrations in soils and groundwater.

Copper. Copper was detected in the following wastes:

- Chlorinated aliphatics wastewater (maximum concentration of 33.5 mg/L);
- EDC/VCM sludge (maximum total concentration of 4,080 mg/kg; maximum TCLP concentration of 22.3 mg/L); and
- Methyl chloride sludge (maximum total concentration of 643 mg/kg; maximum TCLP concentration of 5.3 mg/L) .

Hunter (1998) reported that the 95% upper tolerance limit of copper concentrations in groundwater was 0.086 mg/L and in soils was 53 mg/kg. The concentrations of copper in chlorinated aliphatics wastes exceed these concentrations.

Lead. Lead was detected in the following wastes:

- Chlorinated aliphatics wastewater (maximum concentration of 0.12 mg/L);
- EDC/VCM sludge and (maximum total concentration of 13 mg/kg); and
- Methyl chloride sludge (maximum total concentration of 7 mg/kg).

Hunter (1998) reported that the 95% upper tolerance limit of background lead concentrations in groundwater was 0.047 mg/L and in soils was 54 mg/kg. EPA has established an action level for lead in drinking water of 0.015 mg/L (U.S. EPA 1999b). The concentration of lead in chlorinated aliphatics wastewater exceeds this concentration, as well as the background concentrations reported by Hunter (1998). EPA recommends that soil lead levels not exceed 400 mg/kg, a concentration above which soils may cause elevated blood lead levels that pose a health risk to children (Goldman and Fields 1998). The lead concentrations in the EDC/VCM and methyl chloride sludges are well below this level.

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5.0 RISK CHARACTERIZATION

Estimates of dose and estimates of toxicity (the health benchmarks) are combined to calculate individual excess lifetime carcinogenic risk estimates and non-cancer HQs for the COPCs in chlorinated aliphatic wastewaters, EDC/VCM sludge, and methyl chloride sludge. Specifically, we estimate cancer risk resulting from exposure to a carcinogenic contaminant by multiplying the contaminant's CSF by our estimate of contaminant dose. We calculate a receptor's ingestion hazard quotient resulting from exposure to a noncarcinogenic contaminant by dividing our estimate of contaminant dose by the RfD. We calculate a receptor's inhalation hazard quotient by dividing the concentration of the contaminant in air by the RfC. Appendix E provides the equations that we use to calculate cancer risk and non-cancer hazard quotients. Appendix G provides an example of our calculations using the case of a farmer's exposure to a carcinogen and a noncarcinogen. Appendix H provides risk results for all of the COPCs.

EPA typically decides to list a waste when, for a given receptor, the sum of the excess lifetime cancer risk estimates for the COPCs equals 1×10^{-5} or more or when the sum of the noncancer HQs (for the same health endpoint or target organ) equals 1 or more. The contaminants with the highest risk estimates or HQs, that is, those that generate risk in the "listable" risk range, are the contaminants of concern (COCs) in the waste. None of the contaminants that we evaluated for noncancer health effects produced HQs greater than 1, nor did the sum of contaminant HQs exceed 1. EPA determined that it was not appropriate to sum carcinogenic risk estimates or noncancer hazard quotients for contaminants included in both groundwater and nongroundwater pathways. Estimated risks due to nongroundwater pathways occur during the operating or post-closure life of the unit (that is, due to releases to air and runoff/erosion) while risks via the groundwater pathways are not projected to occur for hundreds, or even thousands, of years due to long times required for contaminant migration in groundwater. Therefore, risks resulting from these two pathways would not apply to the same individual.

This section presents our estimates of individual risk for each of the COCs and provides an overview of the toxicity of each of the COCs. In addition, this section presents a discussion of population risk. Lastly, this section provides an analysis of the sources of uncertainty in our risk assessment.

5.1 Individual Risk

The primary goal of our risk assessment is to estimate individual risk. We calculated individual risk in two ways – using a deterministic approach and using a probabilistic (Monte Carlo) approach. A deterministic risk analysis produces a point estimate of risk or hazard for each receptor based on using a single value for each parameter in the analysis. Using probabilistic analysis, we calculate risk or hazard by allowing some of the parameters to have more than one value, and therefore produce a distribution of risk or hazard for each receptor. This section discusses these risk assessment methods and their results.

5.1.1 Conducting the Deterministic Risk Analysis

EPA conducts both “central tendency” and “high end” deterministic risk assessments to attempt to quantify the risk or hazard for the “average” receptor in the population (the central tendency risk) and the risk or hazard for individuals in small, but definable “high end” segments of the population (the high end risk). For central tendency deterministic risk analyses, we set all parameters at their central tendency values. For the chlorinated aliphatics risk assessments, the central tendency values are either mean (average) or 50th percentile (median) values.

We use high end deterministic risk analysis to predict the risks and hazards for those individuals exposed at the upper range of the distribution of exposures. EPA’s Guidance For Risk Characterization (U.S.EPA 1995) advises that “conceptually, high end exposure means exposure above about the 90th percentile of the population distribution, but not higher than the individual in the population who has the highest exposure”, and recommends that “... the assessor should approach estimating high end by identifying the most sensitive variables and using high end values for a subset of these variables, leaving others at their central values.” For the chlorinated aliphatics high end deterministic risk analyses, EPA set two parameters at their high end values (generally 90th percentile values), and set all other parameters at their central tendency values. We used a “sensitivity analysis” to identify the two parameters that we set at high end. A sensitivity analysis is an iterative procedure in which an analysis is performed by alternately setting different parameters at high end to identify the parameters that most influence the analysis’ outcome. EPA compares the different results generated by the sensitivity analysis and selects the two high end parameters to which the analysis was “most sensitive”, that is, the two parameters that are expected to generate the greatest estimate of risk or hazard.

Groundwater Pathway Sensitivity Analyses

We evaluated groundwater pathway risks for the EDC/VCM sludge landfill and the EDC/VCM sludge land treatment unit. The purpose of the sensitivity analyses conducted for these waste management scenarios was to determine the two most sensitive parameters to set at high end in the two-parameter high end deterministic risk analysis. The parameters that we evaluated in the groundwater pathway sensitivity analyses are as follows:

- Exposure duration
- Distance to the receptor well (“x-well”)
- Leachate concentration
- Distance of the receptor well from the centerline of the plume (“y-well”)
- Waste quantity (for the landfill analysis only)
- Waste management unit area (for the landfill analysis only)
- Waste management unit location (for the landfill analysis only)

We evaluated fewer parameters in the sensitivity analysis for the land treatment unit than for the landfill because the land treatment unit represents one waste volume disposed in one management unit at one location. Thus, for the land treatment unit, we only evaluated one value each for waste volume, waste management unit area, and waste management unit location.

For groundwater pathways, we accomplish the sensitivity analysis in two steps. First, we perform a one-parameter sensitivity analysis for all COPCs (identified in Section 2). The sensitivity analysis for each contaminant consists of a central tendency analysis (all parameters are set at their central tendency values) and a series of analyses in which one of the parameters in the analysis alternately is set at its high end value. For each analysis we calculate the concentration of the contaminant at the receptor well, which equates to contaminant risk. We then determine the relative sensitivities of the parameters we evaluated by ranking the parameters according to the calculated receptor well concentration values. The parameter that we rank most sensitive is considered one of the two most sensitive (that is, one of the two high end) parameters. In conducting the one-parameter sensitivity analysis, the exposure point concentration (EPC) in groundwater is the maximum 9-year average receptor well concentration over the 10,000-year EPACMTP simulation time. The central tendency EPC is the 9-year average receptor well concentration because 9 years is the central tendency exposure duration for the adult resident.

Next, we determine what the other most sensitive parameter is. Specifically, the other most sensitive parameter is either the second-most sensitive parameter we identified in the one-parameter sensitivity analysis, or exposure duration (30 years). Exposure duration is evaluated only for carcinogens because exposures are averaged over a lifetime of 70 years. (When exposure duration is one of the high end parameters in our analysis, we designate the groundwater EPC as the maximum 30-year average receptor well concentration over the 10,000-year simulation time.) We know which parameter to select because increasing exposure duration from 9 to 30 years will increase risk by a factor of 3.33 (30/9), assuming that the maximum 30 year average receptor well concentration is close to the maximum 9 year average receptor well concentration, which is typically the case for the COCs in this assessment. Therefore, we select the second-most sensitive parameter in the one-parameter sensitivity analysis only if the second parameter is more sensitive than exposure duration. In other words, if the receptor well concentration predicted using the second-most sensitive parameter in the sensitivity analysis is greater than 3.33 times the receptor well concentration predicted by setting all parameters at their central tendency values, then the second-most sensitive parameter is selected as the other high-end parameter – otherwise, exposure duration is selected as the other high-end parameter. We then perform the two high-end parameter deterministic risk analysis using the most sensitive parameter from the sensitivity analysis along with either the second most sensitive parameter from the sensitivity analysis or a 30-year exposure duration (and a 30-year receptor well concentration averaging period).

The results of the groundwater pathway sensitivity analyses are provided in Appendices H.2 (Table H.2-7) for the EDC/VCM land treatment unit and H.3 (Table H.3-3) for the EDC/VCM landfill. Tables H.2-9 and H.3-5 provide the groundwater pathway deterministic risk results for the land treatment unit and landfill scenarios, respectively. The peak concentration time (y) values in Tables H.2-8 and H.3-4 represent the time it took the peak concentration of contaminants to arrive at the receptor well. However, when the contaminant breakthrough curve at the receptor well exhibits a broad flat peak, the reported time may not necessarily correspond to the earliest arrival of the peak. Moreover, it may be possible for the contaminant concentration to reach levels of potential concern, in terms of risk, long before the peak concentration arrives at the receptor well. The data indicate that peak concentrations for cis-1,3-dichloropropene, OCDD, OCDF, 1,2,3,4,7,8,9-HpCDF, and 1,2,3,4,6,7,8-HpCDF in landfills do not reach the receptor within the 10,000-year simulation time frame used in the risk analysis.

We made three unrelated errors in calculating deterministic groundwater pathway risks for the EDC/VCM sludge landfill and the methyl chloride sludge landfill. These errors are as follows:

- In certain cases we used contaminant concentrations expressed on a dry weight basis in the analysis rather than the correct wet weight concentrations (see Section 2.1.3). The dry weight concentrations are greater than the wet weight concentrations because the dry weight concentrations reflect the same contaminant mass in a smaller sludge volume (a volume for which the water content has been removed). Appendix I provides the contaminant concentrations in the samples reported on a dry weight basis.

- In certain cases we mistakenly used the wrong groundwater temperature in the analysis. Specifically, we used a groundwater temperature of 15 ^{o}C for locations where the average groundwater temperature should have been 22.5^{o}C. The lower temperature decreases the rates of hydrolysis of contaminants that hydrolyze in groundwater, resulting in overestimates of receptor well concentrations.

- In certain cases we mistakenly calculated groundwater EPCs based on the 1-year maximum receptor well concentration. The 1-year maximum concentration is sometimes greater than the 9-year average receptor well concentration that we should have used, depending upon the movement of the contaminant plume.

Because each of these errors has the impact of increasing our estimates of risk, we did not recalculate risks to correct the errors in those cases where the erroneous risk estimates were below our “listable” risk range (an HQ of 1 or greater, or a cancer risk of 1 x 10^{-5} or greater). In those cases where the erroneous risk results were within our “listable” risk range, we corrected the errors and recalculated the risk results. The tables presented in Appendix H include footnotes that indicate the concentration (wet weight or dry weight), groundwater temperature, and maximum concentration (1-year, 9-years, or 30-years) used in the analysis that generated the results.

Nongroundwater Pathway Sensitivity Analyses

For nongroundwater pathways, EPA conducts both one-parameter and two-parameter sensitivity analyses. We conduct the two-parameter sensitivity analysis in the same manner as the one-parameter sensitivity analysis, except that we alternately select two of the parameters, set them at their high end values (we set the other parameters at their central tendency values), and then calculate risk. Our risk estimate is the highest double high end parameter result. This methodology differs somewhat from the groundwater pathway sensitivity analysis methodology, for which we conduct only single-parameter sensitivity analyses. The reason for this difference is that it is not practical to run the complex groundwater model for every two parameter combination.

We evaluated nongroundwater pathway risks for all of the waste management scenarios (wastewater tank, EDC/VCM sludge landfill, EDC/VCM sludge land treatment unit, and methyl chloride sludge landfill). The parameters that we varied in the nongroundwater pathway sensitivity analyses are as follows:

- Exposure duration
- Distance to receptor
- Contaminant concentration (not evaluated for the methyl chloride landfill analysis)
- Site (meteorological) location (for the EDC/VCM landfill and tank analyses only)
- Waste volume or quantity (for the EDC/VCM landfill and tank analyses only)
- Inhalation rate
- Soil intake (for child receptors only)
- Fish intake (for fisher only)
- Exposed vegetable intake (for gardener, farmer, and child of farmer receptors only)
- Root vegetable intake (for gardener, farmer, and child of farmer receptors only)
- Fruit intake (for gardener, farmer, and child of farmer receptors)
- Beef intake (for farmer and child of farmer only)
- Dairy intake (for farmer and child of farmer only)

As was the case for the groundwater pathways, we did not evaluate site location or waste quantity in the land treatment unit or the methyl chloride landfill sensitivity analyses because under these scenarios there is only one value for each of these two parameters. Moreover, because there is only one sample of methyl chloride sludge, there is only one concentration value for each contaminant, and consequently the parameter “contaminant concentration” has only one value.

For the nongroundwater pathways, media concentrations are estimated at steady state conditions and reflect peak media concentrations (soil, food items) that occur during or following the operational life of the waste management unit. The results of the nongroundwater pathway sensitivity analyses are combined with the nongroundwater pathway deterministic risk results, and are provided in Appendices H.1 (Tables H.1-3 through H.1-6) for the wastewater tank, H.2 (Tables H.2-3 and H.2-4) for the EDC/VCM sludge land treatment unit, H.3 (Tables H.3-1 and H.3-2) for the EDC/VCM sludge landfill, and H.4 (Tables H.4-1 and H.4-2) for the methyl chloride sludge landfill. As was the case for certain groundwater pathway risk analyses, for some of the nongroundwater pathway analyses we mistakenly used contaminant concentrations expressed on a dry weight basis rather than the correct wet weight concentrations. Because this error increased our risk estimates, we only corrected our analyses and recalculated risk in cases where the risk estimates were above our “listable” risk range. The risk analyses that are based on dry weight data instead of wet weight data are noted in Appendix H, and Appendix I provides the sample analytical results presented on a dry weight basis.

5.1.2 Conducting the Probabilistic Risk Analysis

Probabilistic analyses are used to support the results of the deterministic risk analyses and to allow us to quantify individual risk at selected percentiles of the risk distribution (for example, 50th percentile, 90th percentile, 95th percentile). We conducted deterministic risk analyses for all of the receptor types and all of the COPCs identified in Section 2. We conducted probabilistic risk analyses for those combinations of receptor, contaminant, and pathway (groundwater or nongroundwater) for which risk or hazard estimated using a deterministic analysis exceeded the following criteria: a cancer risk of $1x10^{-6}$ or a hazard quotient of 1. As mentioned previously, none of the contaminants generated HQs greater than 1. Moreover, although some groundwater pathway risks exceeded $1x10^{-6}$, groundwater pathway risks were dominated by the exposure via groundwater ingestion. Groundwater pathway risks due to the dermal or inhalation exposure routes were generally 2 to 3 orders of magnitude less than ingestion risks and were not evaluated in the probabilistic risk analyses. One exception that we made is that we did not conduct a probabilistic risk analysis for chloroform in chlorinated aliphatics wastewaters. The maximum chloroform risk was $3x10^{-6}$ for chlorinated aliphatics wastewaters via direct inhalation of vapors downwind from the wastewater tank, and we did not believe that risk was high enough to warrant the resources needed for a full probabilistic analysis.

In a probabilistic analysis, each parameter may have more than one value. EPA develops PDFs that describe the full range of values that the various input parameters may have. Some of the parameters in the probabilistic analysis are set as constant values because 1) there are insufficient data to develop a PDF; 2) EPA made assumptions to simplify the analysis in cases where such simplifications would improve the efficiency of the analysis without influencing the results; 3) site-specific constants are available; or 4) the analysis has been shown not to be sensitive to the value of the parameter, that is, even if the parameter varies, the resulting risk estimate does not vary significantly. Appendix K provides the input parameters used in the probabilistic analysis. In the probabilistic analysis, risk is approximated through repetitive calculation of the fate and transport and exposure equations and models using input parameters randomly selected from the PDFs. The result of the probabilistic analysis is a distribution of the risks or hazards for each of the receptors. Appendix F provides a detailed description of the probabilistic risk assessment procedures.

For each iteration of the probabilistic risk analysis for nongroundwater pathways, we randomly selected:

- One of the samples (to establish a starting contaminant concentration for the constituent evaluated),
- A receptor distance,
- The fraction organic carbon in the waste (for the EDC/VCM sludges only),
- A waste quantity,
- The area of the waste management unit (for the EDC/VCM landfill only),
- The location of the waste management unit (which established the environmental setting of the unit),
- Body weight of the receptor, as applicable (intakes of food items were normalized to body weight),
- The receptor's age at the time of first exposure (the "starting age" for the analysis for child receptors only),
- The receptor's exposure duration, and
- Applicable intake and inhalation rates for the receptor.

For groundwater pathways, we first selected a groundwater concentration from the PDF generated by groundwater fate and transport modeling (see Appendix F.1), and then we selected a receptor body weight, exposure duration, and intake rate. For dioxins, each of the congeners was included as a separate contaminant in the fate and transport modeling and the exposure modeling.

For each iteration of our probabilistic analysis of adult exposure, we randomly selected an exposure duration for the adult, which could range from 1 to 50 years. We then randomly selected a body weight, applicable ingestion rates, and an inhalation rate for the adult. We did not assume that an adult's body weight is correlated with their ingestion or inhalation rates, nor did we assume that their ingestion rate for one food item (for example, fruits) is correlated with their ingestion rates of other food items (for example, beef). For example, the model randomly may have assigned an adult receptor a 20th percentile body weight, an 80th percentile beef ingestion rate, a 10th percentile fruit ingestion rate, and a 70th percentile inhalation rate. Although it is reasonable to assume that intake rates of some food items may be correlated with body weight, sufficient data are not presented in the *Exposure Factors Handbook* to derive appropriate correlation coefficients. Therefore, the options are to assume either no correlation of the exposure factors or perfect correlation of the exposure factors. For the following reasons, we chose to assume no correlation of the exposure factors: 1) some people are known to eat relatively large amounts of certain foods and relatively small amounts of other types of foods; and 2) because the *Exposure Factors Handbook* normalizes intakes of food items to body weight, any effects on the analysis of correlations between intake rates and body weight are minimized.

We conducted probabilistic risk analyses for the child of a farmer and a child resident. For our probabilistic analysis of child exposure, we divided the child age range, 1 to 19 years, into 3 different age cohorts, 1 to 5, 6 to 11, and 12 to 19. Children under the age of 1 were not included in the analysis for two reasons: most of the exposure pathways were not applicable to infants (for example, beef intake), and any exposure a child would receive as an infant likely would be small compared to the child's total exposure.

We randomly selected a starting age for the child. Then, we randomly selected an exposure duration ranging from 1 to 30 years. Consequently, the probabilistic risk estimates for the "child" receptor actually correspond to an individual who initially is exposed to the contaminant source as a child in a given age cohort, but then whose exposure duration may or may not extend into adulthood (to a maximum age of 19 years [maximum age at exposure] + 30 years [maximum exposure duration] = 49 years). This is somewhat different from the deterministic analysis, for which we use exposure factors "averaged" over the child's age range, 1 to 19. After selecting the child's exposure duration, we selected intake rates for the child. We used the intake rates corresponding to the starting age for all subsequent years until the child aged into the next cohort. Once the child aged into the next cohort, we selected new intake rates and body weights; however, we kept the initial exposure duration the same (i.e., new exposure durations were not selected each time a child aged into the next cohort). We maintained correlations of body weights and correlations of intake rates as the child aged from one cohort to the next. That is, if the body weight or an intake rate was selected as the 75th percentile value for the age 1-5 cohort, then the body weights or intake rates for the ages 6 to 11 and 12 to 19 cohorts also would be approximately 75th percentile values. As was the case for the adult receptors, body weights were not correlated with intake rates, nor were intake rates for different parameters correlated with each other (e.g., the body weight could be the 25th percentile, beef intake could be the 40th percentile, and vegetable intake could be the 90th percentile).

For the EDC/VCM land treatment unit and landfill scenarios, we ran ten thousand realizations of the probabilistic risk analysis for each receptor. For the chlorinated aliphatics wastewater tank scenario, because of the large number of variables, we ran nearly 8 million realizations of the probabilistic risk analysis for each receptor. These realizations resulted in a PDFs for each constituent and pathway we evaluated. For dioxins, we evaluated each of the congeners as separate constituents in the fate and transport modeling and exposure modeling, however, we expressed dioxin risks as a total 2,3,7,8-TCDD TEQ.

5.1.3 Risk Results

The following sections provide summaries of deterministic and probabilistic risk results for those contaminants for which a receptor's excess lifetime cancer risk was 1×10^{-6} or greater. For the deterministic risk results, we identify the two parameters we set at high end to estimate risk. For groundwater pathways, we also identify our estimate of the travel time required for the peak concentration of the contaminant to reach the receptor well. Section 5.1.3.1 presents the risk results for the chlorinated aliphatics wastewaters; Section 5.1.3.2 presents the risk results for the EDC/VCM sludges; and Section 5.1.3.3 presents the risk results for the methyl chloride sludges.

5.1.3.1 Chlorinated Aliphatic Wastewaters

Tables 5-1a, 5-1b, and 5-2 summarize risk estimates greater than $1x10^{-6}$ for receptors exposed to contaminants in chlorinated aliphatics wastewaters managed in on-site aerated biological wastewater treatment tanks. Table 5-1a presents the deterministic summary and Table 5-1b presents the probabilistic summary for dioxin (expressed as the 2,3,7,8-TCDD TEQ) risk estimates for nongroundwater pathways. The high-end deterministic risk estimate for the farmer is $2x10^{-5}$. In all cases, we estimated that the highest risks occur for the farmer. Most of this risk is due to ingestion of beef and dairy products. The high end parameters resulting in the highest risk for the farmer are contaminant concentration and distance to receptor. Because we were concerned that concentrations of contaminants occurring in beef and dairy products may be overestimated when distance to receptor (that is, distance to cattle) is at the high end distance (75 m), we reviewed the results for the second highest risk estimate. Specifically, the two high end parameters “contaminant concentration” and “exposure duration” produce an equivalent estimate of high end risk ($2x10^{-5}$) for the farmer. The farmer’s high end deterministic risk falls slightly below the 90th percentile probabilistic risk estimate (the 80th percentile risk estimate is $1x10^{-5}$ [Appendix H.1]). Table 5-2 summarizes the deterministic risks for chloroform via the direct inhalation exposure route. Table 5-3 presents the deterministic wastewater risk results for dioxin by pathway.

5.1.3.2 EDC/VCM Sludges

This section summarizes risk estimates greater than $1x10^{-6}$ for receptors exposed to contaminants in EDC/VCM sludges managed in an on-site land treatment unit and in a municipal landfill. Appendix H.2 presents complete risk results for the land treatment unit and Appendix H.3 provides complete risk results for the landfill.

Table 5-1a. Excess Lifetime Dioxin Cancer Risk, Tank Scenario, Deterministic Results, Nongroundwater Pathways

Receptor	HE	CT	High End Parameters
Farmer	2E-05	4E-07	Contaminant Concentration and Exposure Duration
Child of Farmer	7E-06	3E-07	Contaminant Concentration and Waste Quantity
Home Gardener	2E-08	1E-09	Contaminant Concentration and Distance to Receptor
Adult Resident/Fisher	2E-09	1E-11	Contaminant concentration and Meteorological Location
Child of Resident	7E-09	4E-11	

Table 5-1b. Excess Lifetime Dioxin Cancer Risk, Tank Scenario, Probabilistic Results, Nongroundwater Pathways

Receptor	Percentile				
	50th	**90th**	**95th**	**97.5th**	**100th**
Farmer	2E-07	5E-05	1E-04	3E-04	2E-02
Child of Farmer					
Age 1-5	2E-07	4E-05	1E-04	2E-04	8E-03
Age 6-11	2E-07	4E-05	1E-04	2E-04	9E-03
Age 12-18	2E-07	4E-05	1E-04	2E-04	1E-02

Table 5-2. Excess Lifetime Chloroform Cancer Risk, Tank Scenario, Deterministic Results, Nongroundwater Pathways (Direct Inhalation of Vapor)

Receptor	HE	CT	HE Parameters
Farmer	3E-06	8E-08	Exposure Duration and Distance To Receptor
Child of Farmer/Child Resident	2E-06	8E-08	Contaminant concentration and Distance to Receptor
Home Gardener/Fisher/Adult Resident	2E-06	8E-08	

Table 5-3. Carcinogenic Risks for the Tank Scenario

	TCDD TEQ		
	Central Tendency	**High End**	**High end parameters**
Adult Farmer			
Nongroundwater Pathways			
Inhalation of Vapors	NA	NA	
SUM - Inhalation	NA	NA	
Ingestion of Soil (risk attributable to air pathway)	2E-11	5E-10	HY
Ingestion of Beef (risk attributable to air pathway)	2E-07	8E-06	HY
Ingestion of Milk (risk attributable to air pathway)	2E-07	9E-06	HY
Ingestion of Root Vegetables (risk attributable to air pathway)	2E-12	8E-11	HY
Ingestion of Aboveground Vegetables (risk attributable to air pathway)	6E-10	2E-08	HY
Ingestion of Fruits (risk attributable to air pathway)	4E-09	1E-07	HY
SUM - Ingestion	4E-07	2E-05	HY
Child of Farmer			
Nongroundwater Pathways			
Inhalation of Vapors	NA	NA	
SUM - Inhalation	NA	NA	
Ingestion of Soil (risk attributable to air pathway)	4E-11	6E-10	HQ
Ingestion of Beef (risk attributable to air pathway)	1E-07	4E-06	HQ
Ingestion of Milk (risk attributable to air pathway)	1E-07	4E-06	HQ
Ingestion of Root Vegetables (risk attributable to air pathway)	1E-12	2E-11	HQ
Ingestion of Aboveground Vegetables (risk attributable to air pathway)	3E-10	4E-09	HQ
Ingestion of Fruits (risk attributable to air pathway)	3E-09	4E-08	HQ
SUM - Ingestion	3E-07	7E-06	HQ

Double High End Combinations

HQ - Waste concentration, waste quantity
HY - Waste concentration, exposure duration
NA - Not applicable (all receptor/pathway risk < 10^{-6}).

EDC/VCM Sludges Managed in an On-Site Land Treatment Unit

Tables 5-4a, 5-4b, 5-5a, 5-5b, 5-6a, 5-6b, 5-7a, and 5-7b summarize risk estimates greater than 1×10^{-6} for receptors exposed to contaminants from EDC/VCM wastewater treatment sludges managed in an on-site land treatment unit. In all cases, we estimated that the highest risks occur for the farmer. Tables 5-4a and 5-4b present dioxin (expressed as the 2,3,7,8-TCDD TEQ) risk estimates for the land treatment unit nongroundwater pathways. The high end deterministic risk estimate for the farmer is 2×10^{-4} (Table 5-4a), which also corresponds to the 95th percentile probabilistic risk estimate (Table 5-4b). Tables 5-5a and 5-5b present arsenic risk estimates for the land treatment unit groundwater pathways. The high end deterministic risk estimate for the farmer is 1×10^{-5} (Table 5-5a), which is between the 97.5th percentile probabilistic risk estimate ($6x10^{-6}$) and the 100th percentile probabilistic risk estimate ($5x10^{-5}$) (Table 5-5b). EPA estimates that the groundwater pathway risks in Table 5-5a would occur thousands of years in the future, whereas the dioxin nongroundwater pathway risks in Table 5-4a would occur during the assumed operating life of the land treatment unit.

Table 5-4a. Excess Lifetime Dioxin Cancer Risk, Land Treatment Unit Scenario, Deterministic Results, Nongroundwater Pathways

Receptor	HE	CT	High End Parameters
Farmer	2E-04	4E-06	Exposure Duration and Contaminant Concentration
Child of Farmer	8E-05	3E-06	Contaminant concentration and Beef Intake
Home Gardener	2E-06	6E-08	Exposure Duration and Contaminant Concentration
Fisher	2E-05	6E-07	
Adult Resident	2E-06	5E-08	
Child Resident	5E-06	1E-07	Contaminant Concentration and Soil Intake

Table 5-4b. Excess Lifetime Dioxin Cancer Risk, Land Treatment Unit Scenario, Probabilistic Results, Nongroundwater Pathways

Receptor	Percentile				
	50th	90th	95th	97.5th	100th
Farmer	7E-06	1E-04	2E-04	4E-04	2E-03
Home Gardener	5E-08	2E-06	2E-06	4E-06	2E-05
Resident	3E-08	1E-06	2E-06	2E-06	7E-06
Fisher	2E-07	3E-06	8E-06	2E-05	4E-04
Child Resident					
Age 1-5	1E-07	2E-06	4E-06	6E-06	1E-04
Age 6-11	5E-08	1E-06	2E-06	2E-06	6E-06
Age 12-18	3E-08	8E-07	1E-06	1E-06	4E-06
Child of Farmer					
Age 1-5	9E-06	1E-04	2E-04	3E-04	1E-03
Age 6-11	7E-06	9E-05	2E-04	2E-04	1E-03
Age 12-18	5E-06	7E-05	1E-04	2E-04	1E-03

Table 5-5a. Excess Lifetime Arsenic Cancer Risk, Land Treatment Unit Scenario, Deterministic Results, Groundwater Pathways

Receptor	HE	CT	High End Parameters
Farmer	1E-05	8E-07	Leachate Concentration and Exposure Duration
Child of Farmer/Child Resident	3E-06	6E-07	
Adult Resident/Gardener/Fisher	6E-06	7E-07	

Time for Peak concentration to reach receptor is approximately 1500 years

Table 5-5b. Excess Lifetime Arsenic Cancer Risk, Land Treatment Unit Scenario, Probabilistic Results, Groundwater Pathways

Receptor	Percentile				
	50th	90th	95th	97.5th	100th
Farmer	9E-08	2E-06	4E-06	6E-06	5E-05
Adult Resident/Gardener/Fisher	7E-08	1E-06	3E-06	5E-06	3E-05
Child Resident					
Age 1-5	6E-08	1E-06	2E-06	4E-06	5E-05
Age 6-11	5E-08	1E-06	2E-06	3E-06	4E-05
Age 12-18	4E-08	9E-07	2E-06	3E-06	4E-05
Child of Farmer					
Age 1-5	1E-07	2E-06	4E-06	6E-06	4E-05
Age 6-11	8E-08	2E-06	3E-06	4E-06	2E-05
Age 12-18	6E-08	1E-06	2E-06	4E-06	2E-05

Tables 5-6a, 5-6b, 5-7a, and 5-7b present risk results for the land treatment unit that exceed 1x10^{-6} but are below 1x10^{-5}. Table 5-6a presents arsenic risks that occur via nongroundwater pathways. The high end deterministic risk for the farmer is 6x10^{-6} (Table 5-6a), which corresponds to the 95th percentile probabilistic risk estimate (Table 5-6b). Tables 5-7a and 5-7b present hexavalent chromium risks that occur via nongroundwater pathways. The high end deterministic risk for the farmer is 2x10^{-6} (Table 5-7a), also the 95th percentile probabilistic risk estimate (Table 5-7b). Hexavalent chromium is a carcinogen only via inhalation exposure. Ingestion of hexavalent chromium never exceeded the RfD.

Table 5-8 summarizes the deterministic risk results for arsenic, hexavalent chromium, and dioxin by pathway.

Table 5-6a. Excess Lifetime Arsenic Cancer Risk, Land Treatment Unit Scenario, Deterministic Results, Nongroundwater Pathways

Receptor	HE	CT	High End Parameters
Farmer	6E-06	5E-07	Exposure Duration and Contaminant Concentration
Child of Farmer	2E-06	4E-07	
Home Gardener	9E-07	1E-07	
Fisher	3E-07	3E-08	Exposure Duration and Fish Intake
Adult Resident	3E-07	4E-08	Exposure Duration and Contaminant Concentration
Child Resident	1E-06	1E-07	Contaminant Concentration and Soil Intake

Table 5-6b. Excess Lifetime Arsenic Cancer Risk, Land Treatment Unit Scenario, Probabilistic Results, Nongroundwater Pathways

Receptor	Percentile				
	50th	90th	95th	97.5th	100th
Farmer	6E-07	4E-06	6E-06	8E-06	2E-05
Home Gardener	1E-07	7E-07	1E-06	2E-06	9E-06
Resident	3E-08	2E-07	3E-07	4E-07	1E-06
Child Resident					
Age 1-5	6E-08	5E-07	8E-07	1E-06	2E-05
Age 6-11	4E-08	2E-07	3E-07	3E-07	9E-07
Age 12-18	3E-08	1E-07	2E-07	2E-07	6E-07
Child of Farmer					
Age 1-5	6E-07	3E-06	4E-06	6E-06	2E-05
Age 6-11	4E-07	2E-06	3E-06	4E-06	2E-05
Age 12-18	3E-07	2E-06	3E-06	4E-06	1E-05

Table 5-7a. Excess Lifetime Chromium VI Cancer Risk, Land Treatment Unit Scenario, Deterministic Results, Nongroundwater Pathways, Inhalation

Receptor	HE	CT	High End Parameters
Farmer	2E-06	2E-07	Exposure Duration and Contaminant Concentration
Child of Farmer/Child Resident	1E-06	2E-07	Contaminant Concentration and Distance to Receptor
Adult Resident/Gardener/Fisher	1E-06	1E-07	Exposure Duration and Contaminant Concentration

Table 5-7b. Excess Lifetime Chromium VI Cancer Risk, Land Treatment Unit Scenario, Probabilistic Results, Nongroundwater Pathways, Inhalation

Receptor	Percentile				
	50th	90th	95th	97.5th	100th
Farmer - Inhalation	1E-07	1E-06	2E-06	3E-06	1E-05
Gardener, Resident, Fisher - Inhalation	8E-08	8E-07	1E-06	2E-06	9E-06

Table 5-8. Carcinogenic Risks for the Land Treatment Unit Scenario

	Arsenic			Chromium VI			TCDD TEQ		
	Central Tendency	High End	High end parameters	Central Tendency	High End	High end parameters	Central Tendency	High End	High end parameters
Adult Resident									
Groundwater Pathways									
Ingestion of Drinking Water	7E-07	6E-06	YQ	NA	NA		NA	NA	
Inhalation of Volatiles in the Rest of the House (non-bathroom)	NA	NA		NA	NA		NA	NA	
Inhalation of Volatiles While Showering	NA	NA		NA	NA		NA	NA	
Inhalation of Volatiles after Showering	NA	NA		NA	NA		NA	NA	
SUM - Inhalation	NA	NA		NA	NA		NA	NA	
Dermal Contact While Showering	<10-8	7E-08	YQ	NA	NA		NA	NA	
SUM - Dermal and Ingestion	7E-07	6E-06	YQ	NA	NA		NA	NA	
Nongroundwater Pathways									
Inhalation of Vapors	NA	NA		NA	NA		NA	NA	
Inhalation of Dust	NA	NA		1E-07	1E-06	YH	NA	NA	
SUM - Inhalation	NA	NA		1E-07	1E-06	YH	NA	NA	
Ingestion of Soil (risk attributable to air pathway)	<10-8	<10-8	YH	NA	NA		<10-8	<10-8	YH
Ingestion of Soil (risk attributable to erosion and runoff)	4E-08	3E-07	YH	NA	NA		5E-08	2E-06	YH
SUM - Ingestion	4E-08	3E-07	YH	NA	NA		5E-08	2E-06	YH
Child Resident									
Groundwater Pathways									
Ingestion of Drinking Water	6E-07	3E-06	YQ	NA	NA		NA	NA	
Inhalation of Volatiles in the Rest of the House (non-bathroom)	NA	NA		NA	NA		NA	NA	
Inhalation of Volatiles While Showering	NA	NA		NA	NA		NA	NA	
Inhalation of Volatiles after Showering	NA	NA		NA	NA		NA	NA	
SUM - Inhalation	NA	NA		NA	NA		NA	NA	
Dermal Contact While Showering	NA	NA		NA	NA		NA	NA	

(continued)

Table 5-8. (continued)

	Arsenic			Chromium VI			TCDD TEQ		
	Central Tendency	High End	High end parameters	Central Tendency	High End	High end parameters	Central Tendency	High End	High end parameters
SUM - Dermal and Ingestion	6E-07	3E-06	YQ	NA	NA		NA	NA	
Nongroundwater Pathways									
Inhalation of Vapors	NA	NA		NA	NA		NA	NA	
Inhalation of Dust	NA	NA		2E-07	1E-06	HM	NA	NA	
SUM - Inhalation	NA	NA		2E-07	1E-06	HM	NA	NA	
Ingestion of Soil (risk attributable to air pathway)	<10-8	<10-8	HK	NA	NA		<10-8	<10-8	HK
Ingestion of Soil (risk attributable to erosion and runoff)	1E-07	9E-07	HK	NA	NA		1E-07	5E-06	HK
Dermal Contact with Soil	NA	NA		NA	NA		NA	NA	
SUM - Ingestion	1E-07	9E-07	HK	NA	NA		1E-07	5E-06	HK
Additional Pathways for Home Gardener (In addition to Adult Resident)									
Nongroundwater Pathways									
Ingestion of Soil (risk attributable to air pathway)	<10-8	<10-8	YH	NA	NA		<10-8	<10-8	YH
Ingestion of Soil (risk attributable to erosion and runoff)	4E-08	3E-07	YH	NA	NA		5E-08	2E-06	YH
Ingestion of Root Vegetables (risk attributable to air pathway)	<10-8	<10-8	YH	NA	NA		<10-8	<10-8	YH
Ingestion of Root Vegetables (risk attributable to erosion and runoff)	<10-8	3E-08	YH	NA	NA		<10-8	9E-08	YH
Ingestion of Aboveground Vegetables (risk attributable to air pathway)	<10-8	<10-8	YH	NA	NA		<10-8	4E-08	YH
Ingestion of Aboveground Vegetables (risk attributable to erosion and runoff)	3E-08	3E-07	YH	NA	NA		<10-8	7E-08	YH
Ingestion of Fruits (risk attributable to air pathway)	<10-8	<10-8	YH	NA	NA		<10-8	1E-07	YH
Ingestion of Fruits (risk attributable to erosion and runoff)	3E-08	2E-07	YH	NA	NA		<10-8	2E-07	YH

(continued)

Table 5-8. (continued)

	Arsenic			Chromium VI			TCDD TEQ		
	Central Tendency	High End	High end parameters	Central Tendency	High End	High end parameters	Central Tendency	High End	High end parameters
SUM - Ingestion	1E-07	9E-07	YH	NA	NA		6E-08	2E-06	YH
Additional Pathway for Fisher (In addition to Adult Resident)									
Nongroundwater Pathways									
Ingestion of Fish (risk attributable to SW contaminated via air pathway)	NA	NA		NA	NA		NA	NA	
Ingestion of Fish (risk attributable to SW contaminated via erosion and runoff)	3E-08	3E-07	YD	NA	NA		6E-07	2E-05	YH
SUM - Ingestion	3E-08	3E-07	YD	NA	NA		6E-07	2E-05	YH
Groundwater Pathways									
Ingestion of Fish	<10-8	<10-8	YQ	NA	NA		NA	NA	
SUM (for receptor, groundwater pathways)	7E-07	6E-06	YQ	NA	NA		NA	NA	
Adult Farmer									
Groundwater Pathways									
Ingestion of Drinking Water	8E-07	1E-05	YQ	NA	NA		NA	NA	
Inhalation of Volatiles in the Rest of the House (non-bathroom)	NA	NA		NA	NA		NA	NA	
Inhalation of Volatiles While Showering	NA	NA		NA	NA		NA	NA	
Inhalation of Volatiles after Showering	NA	NA		NA	NA		NA	NA	
SUM - Inhalation	NA	NA		NA	NA		NA	NA	
Dermal Contact While Showering	<10-8	1E-07	YQ	NA	NA		NA	NA	
SUM - Dermal and Ingestion	8E-07	1E-05	YQ	NA	NA		NA	NA	
Nongroundwater Pathways									
Inhalation of Vapors	NA	NA		NA	NA		NA	NA	
Inhalation of Dust	NA	NA		2E-07	2E-06	YH	NA	NA	
SUM - Inhalation	NA	NA		2E-07	2E-06	YH	NA	NA	
Ingestion of Soil (risk attributable to air deposition)	<10-8	<10-8	YH	NA	NA		<10-8	<10-8	YH

(continued)

Table 5-8. (continued)

	Arsenic			Chromium VI			TCDD TEQ		
	Central Tendency	High End	High end parameters	Central Tendency	High End	High end parameters	Central Tendency	High End	High end parameters
Ingestion of Soil (risk attributable to erosion and runoff)	5E-08	6E-07	YH	NA	NA		8E-08	4E-06	YH
Ingestion of Beef (risk attributable to air deposition)	<10-8	3E-08	YH	NA	NA		1E-06	6E-05	YH
Ingestion of Beef (risk attributable to erosion and runoff)	1E-07	1E-06	YH	NA	NA		8E-07	4E-05	YH
Ingestion of Milk (risk attributable to air deposition)	<10-8	5E-09	YH	NA	NA		1E-06	6E-05	YH
Ingestion of Milk (risk attributable to erosion and runoff)	2E-08	2E-07	YH	NA	NA		5E-07	2E-05	YH
Ingestion of Root Vegetables (risk attributable to air deposition)	<10-8	3E-09	YH	NA	NA		<10-8	<10-8	YH
Ingestion of Root Vegetables (risk attributable to erosion and runoff)	7E-08	9E-07	YH	NA	NA		<10-8	5E-07	YH
Ingestion of Aboveground Vegetables (risk attributable to air deposition)	<10-8	8E-09	YH	NA	NA		<10-8	2E-07	YH
Ingestion of Aboveground Vegetables (risk attributable to erosion and runoff)	1E-07	1E-06	YH	NA	NA		<10-8	5E-07	YH
Ingestion of Fruits (risk attributable to air deposition)	<10-8	2E-08	YH	NA	NA		1E-08	7E-07	YH
Ingestion of Fruits (risk attributable to erosion and runoff)	1E-07	2E-06	YH	NA	NA		3E-08	1E-06	YH
SUM - Ingestion	5E-07	6E-06	YH	NA	NA		4E-06	2E-04	YH
Child of Farmer									
Groundwater Pathways									
Ingestion of Drinking Water	6E-07	3E-06	YQ	NA	NA		NA	NA	
Inhalation of Volatiles in the Rest of the House (non-bathroom)	NA	NA		NA	NA		NA	NA	
Inhalation of Volatiles While Showering	NA	NA		NA	NA		NA	NA	
Inhalation of Volatiles after Showering	NA	NA		NA	NA		NA	NA	
SUM - Inhalation	NA	NA		NA	NA		NA	NA	
Dermal Contact While Showering	NA	NA		NA	NA		NA	NA	

(continued)

Table 5-8. (continued)

	Arsenic			Chromium VI			TCDD TEQ		
	Central Tendency	High End	High end parameters	Central Tendency	High End	High end parameters	Central Tendency	High End	High end parameters
SUM - Dermal and Ingestion	6E-07	3E-06	YQ	NA	NA		NA	NA	
Nongroundwater Pathways									
Inhalation of Vapors	NA	NA		NA	NA		NA	NA	
Inhalation of Dust	NA	NA		2E-07	1E-06	HM	NA	NA	
SUM - Inhalation	NA	NA		2E-07	1E-06	HM	NA	NA	
Ingestion of Soil (risk attributable to air deposition)	<10-8	<10-8	YH	NA	NA		<10-8	<10-8	HZ
Ingestion of Soil (risk attributable to erosion and runoff)	1E-07	7E-07	YH	NA	NA		1E-07	2E-06	HZ
Ingestion of Beef (risk attributable to air deposition)	<10-8	1E-08	YH	NA	NA		9E-07	4E-05	HZ
Ingestion of Beef (risk attributable to erosion and runoff)	9E-08	5E-07	YH	NA	NA		7E-07	3E-05	HZ
Ingestion of Milk (risk attributable to air deposition)	<10-8	2E-09	YH	NA	NA		1E-06	1E-05	HZ
Ingestion of Milk (risk attributable to erosion and runoff)	1E-08	7E-08	YH	NA	NA		4E-07	4E-06	HZ
Ingestion of Root Vegetables (risk attributable to air deposition)	<10-8	<10-8	YH	NA	NA		<10-8	<10-8	HZ
Ingestion of Root Vegetables (risk attributable to erosion and runoff)	5E-09	3E-08	YH	NA	NA		<10-8	5E-08	HZ
Ingestion of Aboveground Vegetables (risk attributable to air deposition)	<10-8	<10-8	YH	NA	NA		<10-8	3E-08	HZ
Ingestion of Aboveground Vegetables (risk attributable to erosion and runoff)	6E-08	3E-07	YH	NA	NA		<10-8	5E-08	HZ
Ingestion of Fruits (risk attributable to air deposition)	<10-8	<10-8	YH	NA	NA		2E-08	3E-07	HZ

(continued)

Table 5-8. (continued)

	Arsenic			Chromium VI			TCDD TEQ		
	Central Tendency	High End	High end parameters	Central Tendency	High End	High end parameters	Central Tendency	High End	High end parameters
Ingestion of Fruits (risk attributable to erosion and runoff)	1E-07	6E-07	YH	NA	NA		<10-8	9E-08	HZ
SUM - Ingestion	4E-07	2E-06	YH	NA	NA		3E-06	8E-05	HZ

Double High End Combinations
DH = Fish intake, waste concentration
HK = Waste concentration, child soil intake
HM = Waste concentration, distance to receptor
HZ = Waste concentration, beef intake
LR = Site location, x-well
QR = Leachate concentration, X-well
YD = Exposure Duration, Fish intake
YH = Exposure duration, waste concentration
YQ = Exposure duration, leaching concentration
NA = Not applicable
(all receptor/pathway risk < 10^{-6}).

EDC/VCM Sludges Managed in a Municipal Landfill

Tables 5-9a and 5-9b summarize risk estimates greater than 1x10^{-6} for receptors exposed to contaminants in EDC/VCM sludges managed in an off-site municipal landfill. The risk estimates presented in Tables 5-9a and 5-9b are arsenic groundwater pathway risks. The route of exposure is ingestion of contaminated groundwater. The high end deterministic risk estimate for the farmer is 3x10^{-5}, which falls between the 97.5th percentile (1x10^{-5}) and the 100th percentile (3x10^{-4}) probabilistic risk estimates. We estimate that the arsenic risks attributable to the landfill (presented in Table 5-9a) would occur thousands of years in the future. Table 5-10 summarizes the deterministic risk results for arsenic by pathway.

Table 5-9a . Excess Lifetime Arsenic Cancer Risks, Landfill Scenario, Deterministic Results, Groundwater Pathways

Receptor	HE	CT	High End Parameters
Farmer	3E-05	9E-07	Distance To Receptor Well and Exposure Duration
Child of Farmer/Child Resident	9E-06	6E-07	
Adult Resident/Gardener/Fisher	2E-05	8E-07	

Time for Peak concentration to reach receptor is approximately 8800 years (HE), 9600 years (CT)

Table 5-9b. Excess Lifetime Arsenic Cancer Risks, Landfill Scenario, Probabilistic Results, Groundwater Pathways

Receptor	Percentile				
	50th	90th	95th	97.5th	100th
Farmer	2E-08	3E-06	8E-06	1E-05	3E-04
Adult Resident/Gardener/Fisher	2E-08	3E-06	6E-06	1E-05	2E-04
Child Resident					
Age 1-5	1E-08	2E-06	5E-06	9E-06	1E-04
Age 6-11	1E-08	2E-06	4E-06	8E-06	1E-04
Age 12-18	9E-09	1E-06	3E-06	6E-06	9E-05
Child of Farmer					
Age 1-5	3E-08	4E-06	9E-06	2E-05	2E-04
Age 6-11	2E-08	3E-06	6E-06	1E-05	2E-04
Age 12-18	1E-08	2E-06	5E-06	1E-05	1E-04

Table 5-10. Carcinogenic Risk for the Landfill Scenario

	Arsenic		
	Central Tendency	**High End**	**High End Parameters**
Adult Resident			
Groundwater Pathways			
Ingestion of Drinking Water	8E-07	**2E-05**	RY
Inhalation of Volatiles in the Rest of the House (non-bathroom)	NA	NA	
Inhalation of Volatiles While Showering	NA	NA	
Inhalation of Volatiles after Showering	NA	NA	
SUM - Inhalation	NA	NA	
Dermal Contact While Showering	9E-09	2E-07	RY
SUM - Dermal and Ingestion	8E-07	**2E-05**	RY
Nongroundwater Pathways			
Inhalation of Vapors	NA	NA	
Inhalation of Dust	NA	NA	
SUM - Inhalation	NA	NA	
Ingestion of Soil (risk attributable to air pathway)	NA	NA	
Ingestion of Soil (risk attributable to erosion and runoff)	NA	NA	
SUM - Ingestion	NA	NA	
Child Resident			
Groundwater Pathways			
Ingestion of Drinking Water	6E-07	**9E-06**	RY
Inhalation of Volatiles in the Rest of the House (non-bathroom)	NA	NA	
Inhalation of Volatiles While Showering	NA	NA	
Inhalation of Volatiles after Showering	NA	NA	
SUM - Inhalation	NA	NA	
Dermal Contact While Showering	NA	NA	
SUM - Dermal and Ingestion	6E-07	**9E-06**	RY
Nongroundwater Pathways			
Inhalation of Vapors	NA	NA	
Inhalation of Dust	NA	NA	
SUM - Inhalation	NA	NA	
Ingestion of Soil (risk attributable to air pathway)	NA	NA	
Ingestion of Soil (risk attributable to erosion and runoff)	NA	NA	
Dermal Contact with Soil	NA	NA	
SUM - Ingestion	NA	NA	
Additional Pathways for Home Gardener (In addition to Adult Resident)			
Nongroundwater Pathways			
Ingestion of Soil (risk attributable to air pathway)	NA	NA	

(continued)

Table 5-10. (continued)

	Arsenic		
	Central Tendency	High End	High End Parameters
Ingestion of Soil (risk attributable to erosion and runoff)	NA	NA	
Ingestion of Root Vegetables (risk attributable to air pathway)	NA	NA	
Ingestion of Root Vegetables (risk attributable to erosion and runoff)	NA	NA	
Ingestion of Aboveground Vegetables (risk attributable to air pathway)	NA	NA	
Ingestion of Aboveground Vegetables (risk attributable to erosion and runoff)	NA	NA	
Ingestion of Fruits (risk attributable to air pathway)	NA	NA	
Ingestion of Fruits (risk attributable to erosion and runoff)	NA	NA	
SUM - Ingestion	NA	NA	
Additional Pathway for Fisher (In addition to Adult Resident)			
Nongroundwater Pathways			
Ingestion of Fish (risk attributable to SW contaminated via air pathway)	<10-8	<10-8	
Ingestion of Fish (risk attributable to SW contaminated via erosion and runoff)	NA	NA	
SUM - Ingestion	<10-8	<10-8	
Groundwater Pathways			
Ingestion of Fish	<10-8	<10-8	
SUM (for receptor, groundwater pathways)	8E-07	**2E-05**	RY
Adult Farmer			
Groundwater Pathways			
Ingestion of Drinking Water	9E-07	3E-05	RY
Inhalation of Volatiles in the Rest of the House (non-bathroom)	NA	NA	
Inhalation of Volatiles While Showering	NA	NA	
Inhalation of Volatiles after Showering	NA	NA	
SUM - Inhalation	NA	NA	
Dermal Contact While Showering	1E-08	3E-07	RY
SUM - Dermal and Ingestion	9E-07	3E-05	RY
Nongroundwater Pathways			
Inhalation of Vapors	NA	NA	
Inhalation of Dust	NA	NA	
SUM - Inhalation	NA	NA	
Ingestion of Soil (risk attributable to air pathway)	NA	NA	
Ingestion of Soil (risk attributable to erosion and runoff)	NA	NA	
Ingestion of Beef (risk attributable to air pathway)	NA	NA	
Ingestion of Beef (risk attributable to erosion and runoff)	NA	NA	
Ingestion of Milk (risk attributable to air pathway)	NA	NA	
Ingestion of Milk (risk attributable to erosion and runoff)	NA	NA	
Ingestion of Root Vegetables (risk attributable to air pathway)	NA	NA	

(continued)

Table 5-10. (continued)

	Arsenic		
	Central Tendency	**High End**	**High End Parameters**
Ingestion of Root Vegetables (risk attributable to erosion and runoff)	NA	NA	
Ingestion of Aboveground Vegetables (risk attributable to air pathway)	NA	NA	
Ingestion of Aboveground Vegetables (risk attributable to erosion and runoff)	NA	NA	
Ingestion of Fruits (risk attributable to air pathway)	NA	NA	
Ingestion of Fruits (risk attributable to erosion and runoff)	NA	NA	
SUM - Ingestion	NA	NA	
Child of Farmer			
Groundwater Pathways			
Ingestion of Drinking Water	6E-07	**9E-06**	RY
Inhalation of Volatiles in the Rest of the House (non-bathroom)	NA	NA	
Inhalation of Volatiles While Showering	NA	NA	
Inhalation of Volatiles after Showering	NA	NA	
SUM - Inhalation	NA	NA	
Dermal Contact While Showering	NA	NA	
SUM - Dermal and Ingestion	6E-07	**9E-06**	RY
Nongroundwater Pathways			
Inhalation of Vapors	NA	NA	
Inhalation of Dust	NA	NA	
SUM - Inhalation	NA	NA	
Ingestion of Soil (risk attributable to air pathway)	NA	NA	
Ingestion of Soil (risk attributable to erosion and runoff)	NA	NA	
Ingestion of Beef (risk attributable to air pathway)	NA	NA	
Ingestion of Beef (risk attributable to erosion and runoff)	NA	NA	
Ingestion of Milk (risk attributable to air pathway)	NA	NA	
Ingestion of Milk (risk attributable to erosion and runoff)	NA	NA	
Ingestion of Root Vegetables (risk attributable to air pathway)	NA	NA	
Ingestion of Root Vegetables (risk attributable to erosion and runoff)	NA	NA	
Ingestion of Aboveground Vegetables (risk attributable to air pathway)	NA	NA	
Ingestion of Aboveground Vegetables (risk attributable to erosion and runoff)	NA	NA	
Ingestion of Fruits (risk attributable to air pathway)	NA	NA	
Ingestion of Fruits (risk attributable to erosion and runoff)	NA	NA	
SUM - Ingestion	NA	NA	

Double High End Combinations.
RY = X-well, exposure duration.
NA = not applicable (all receptor/pathway risk <10^{-6}).

5.1.3.3 Methyl Chloride Sludges

EPA conducted a deterministic analysis to estimate nongroundwater (air) pathway risks associated with management of methyl chloride sludges in an on-site landfill. All nongroundwater pathway carcinogenic risks were less than 1×10^{-8}, and all noncancer HQs were less than 0.0001. Appendix H.4 presents the complete methyl chloride sludge risk results.

5.2 Population Risk

EPA expects that the population risks resulting from management of chlorinated aliphatics wastewaters in tanks and EDC/VCM sludges in on-site land treatment units and landfills is not significant. With regard to groundwater pathway risks, EPA believes that the number of domestic drinking water wells (thus the population) potentially affected by groundwater contaminated with arsenic originating from the landfill and the land treatment unit would be very small. Furthermore, the arsenic concentrations predicted in receptor (drinking water) wells would result in risks only slightly above 1×10^{-5} for that very small number of people.

For nongroundwater pathways, EPA performed a screening evaluation of population risk for the waste management scenario and pathway that resulted in the greatest risk of any pathway evaluated in the chlorinated aliphatics risk assessment. Specifically, EPA evaluated risk associated with ingestion of beef and dairy products contaminated with dioxins derived from the on-site EDC/VCM land treatment unit. The farmer's total individual excess lifetime cancer risk from ingestion of beef and dairy was 2×10^{-4} for high end exposures and 4×10^{-6} for central tendency exposures.

The results of the population risk screening analysis are described below. The steps required to calculate population risk due to ingestion of contaminated beef and dairy products are as follows:

1. **Generate average dioxin TEQ concentrations in beef and dairy products.** We combined emission rates from the land treatment unit (calculated for the deterministic risk analysis) with results from air dispersion modeling for the area within a 2-km radius of the site. We used average air concentrations and deposition rates within the 2-km radius to estimate average dioxin concentrations in beef and dairy products (according to the equations used in the deterministic analysis). We did not consider dioxin

contribution from the erosion and runoff pathways because the relative contribution of dioxins from these pathways to total average dioxin concentrations within a 2-km radius of the site would be small compared to the contribution from air pathways. We calculated average dioxin concentrations of 7.0E-08 mg/kg and 1.9E-08 mg/kg in beef and dairy, respectively.

2. **Determine the average number of beef and dairy cattle raised within a 2-km radius of the site.** Agricultural census data were used to determine the number of farms and number of beef and dairy cattle within the Iberville Parish, the location of the land treatment unit (http://govinfo.library.orst.edu/cgi-bin/ag-list?14-047.lac). There were 5,415 beef cattle reported in the Iberville Parish for the latest census; however, no dairy cattle were reported. The area of cropland and pasture within the 2-km radius (minus the area of the Mississippi River) was estimated to be about 11 km^2, or about 2.8 percent of the total cropland and pasture (397 km^2) within the parish. Assuming an even distribution of cattle within the parish's cropland and pastureland, 150 beef cattle and no dairy cattle are raised within 2 km of the site.

3. **Determine the average amount of beef and dairy products produced per animal per year.** According to the 1998 agricultural statistics (USDA 1998), the average weight of a steer (1992 to 1997 data) is 1,241 pounds (564 kg) and the average dressed weight (1987 to 1996 data) is 750 pounds (338 kg), or about 60 percent of the live weight. Although dressed weight includes bone, we used 60 percent of the live weight as a conservative estimate of the amount of beef produced per animal. Since no dairy cattle are reported to be raised in the parish, a population risk estimate for dairy was unnecessary.

4. **Determine the average number of people ingesting beef produced from animals raised near the site**. We used the average beef ingestion rate of 0.0984 kg/d, or 36 kg/yr/person, used in the deterministic analysis to determine the potential number of people ingesting beef on an annual basis. We estimated that the annual amount of beef ingested is about 50,700 kg (150 beef cattle × 338 kg/animal), resulting in a potential population of about 1,410 beef consumers per year.

5. **Calculate the average excess lifetime cancer risk for an individual consuming contaminated beef or dairy products for 1 year.** The individual risk estimate for beef consumers was $2x10^{-7}$. This is based on the average TCDD TEQ concentration in beef (from Step 1) and the average beef ingestion rate (from Step 4), an adult body weight of 70 kg, and an exposure duration of 1 year.

6. **Calculate the annualized population risk estimate.** The annualized population risk is the product of the individual lifetime cancer risk estimate, the annual population of beef consumers, and the expected active life of the land treatment unit (40 years) divided by 70 years. In this case, the annualized population risk is $2x10^{-4}$ excess cancer cases per year.

Results of the population risk analysis indicate that 0.0002 excess cancer cases would be expected annually in a population of 1,411 individuals ingesting beef produced from cattle raised within 2 km of the land treatment unit over a 40-year operational period for the land treatment unit. Although the population risks attributable to the management of chlorinated aliphatics wastes are expected to be very small, “population risk” is not identified explicitly in the RCRA statute or the hazardous waste listing regulations at 40 CFR 261.11 as one of the factors that EPA must consider in making listing decisions. EPA does not believe it is appropriate to allow contamination from waste management activities to cause substantial risk to nearby residents simply because there are few individual in the immediate vicinity of the waste management units. 40 CRF 261.11 clearly states that wastes are to be listed if they are “capable of posing a substantial present or potential hazard,” it does not imply that a large number of people must be affected. Moreover, EPA’s Guidance for Risk Characterization (U.S. EPA 1995) states that when small populations are exposed, population risk estimates may be very small, however, “in such situations, individual risk estimates will usually be a more meaningful parameter for decision-makers.” Consequently, EPA’s decision to list wastes is based primarily on the concern over risks to those individuals who are significantly exposed, even if there are relatively few such individuals.

5.3 Overview of the Toxicity of the COCs

This section provides a summary of information that describes our understanding of the toxicity of arsenic and dioxin, the COCs identified in this risk assessment.

5.3.1 Arsenic

Arsenic is a naturally occurring element in the earth's crust that usually is found combined with other elements. Arsenic combined with elements such as oxygen, chlorine, and sulfur is referred to as inorganic arsenic; arsenic combined with carbon and hydrogen is referred to as organic arsenic. In this health effects summary, arsenic refers to inorganic arsenic and its associated compounds.

Cancer Effects. There is clear evidence that chronic exposure to inorganic arsenic in humans increases the risk of cancer. Studies have reported that inhalation of arsenic results in an increased risk of lung cancer. In addition, ingestion of arsenic has been associated with an increased risk of nonmelanoma skin cancer and bladder, liver, kidney, and lung cancer. No information is available on the risk of cancer in humans from dermal exposure to arsenic (U.S. EPA 1999).

Animal studies have not clearly associated arsenic exposure, via ingestion exposure, with cancer. No studies have investigated the risk of cancer in animals as a result of inhalation or dermal exposure (U.S. EPA 1999).

EPA has classified inorganic arsenic in Group A - Known Human Carcinogen. For arsenic, the Group A classification was based on the increased incidence in humans of lung cancer through inhalation exposure and the increased risk of skin, bladder, liver, kidney, and lung cancer through drinking water exposure (U.S. EPA 1999).

Inhalation Cancer Risk. EPA used the absolute-risk linear extrapolation model to estimate the inhalation unit risk for inorganic arsenic. Five studies on arsenic-exposed copper smelter workers were modeled for excess cancer risk (Brown and Chu 1983a, b, and c; Enterline and Marsh 1982; Higgins et al. 1982; Lee and Feldstein 1983; Welch et al. 1982). All five studies showed excess risks of lung cancer that were related to the intensity and duration of exposure and the duration of the latency period. The estimates of unit risk obtained from the five studies were in reasonably good agreement, ranging from 1.25×10^{-3} to 7.6×10^{-3} $(\mu g/m^3)^{-1}$. Using the geometric mean of these data, EPA calculated an inhalation unit risk estimate of 4.3×10^{-3} $(\mu g/m^3)^{-1}$ (U.S. EPA 1999).

Oral Cancer Risk. To estimate the risks posed by ingestion of arsenic, EPA used the data that Tseng (1977) obtained in Taiwan concerning skin cancer incidence, age, and level of exposure via drinking water. In 37 villages that had obtained drinking water for 45 years from artesian wells with various elevated levels of arsenic, 40,421 individuals were examined for hyperpigmentation, keratosis, skin cancer, and blackfoot disease (gangrene of the extremities caused by injury to the peripheral vasculature). The local well waters were analyzed for arsenic, and the age-specific cancer prevalence rates were found to be correlated with both local arsenic concentrations and age (duration of exposure). EPA used these data to calculate a unit risk estimate for arsenic. It was assumed that Taiwanese persons had a constant exposure from birth and that males consumed 3.5 liters of drinking water per day and females consumed 2.0 liters per day. Doses were converted to equivalent doses for U.S. males and females based on differences in body weights and differences in water consumption, and it was assumed that skin cancer risk in the U.S. population would be similar to that in the Taiwanese population. The multistage model with time was used to predict dose-specific and age-specific skin cancer prevalence rates associated with ingestion of inorganic arsenic. EPA calculated an oral cancer slope factor of 1.5 $(mg/kg/d)^{-1}$ with a corresponding unit risk estimate of 5.0×10^{-5} $(\mu g/L)^{-1}$ from oral exposure to arsenic in drinking water (see U.S. EPA 1999 for a further discussion of this study).

Noncancer Effects. The most common noncancer effects noted in humans from chronic exposure to arsenic, through both inhalation and oral exposure, are effects on the skin. The inhalation route has resulted primarily in irritation of the skin and mucous membranes (dermatitis, conjunctivitis, pharyngitis, and rhinitis), while chronic oral exposure has resulted in a pattern of skin changes that includes the formation of warts or corns on the palms and soles, along with areas of darkened skin on the face, neck, and back. Other effects noted from chronic oral exposure include peripheral neuropathy, cardiovascular disorders, liver and kidney disorders, and blackfoot disease. No information is available on effects in humans from chronic low-level dermal exposure to arsenic (ATSDR 1993). No studies are available on the chronic noncancer effects of arsenic in animals from inhalation or dermal exposure. Oral animal studies have noted effects on the kidney and liver (ATSDR 1993).

Reference Dose. EPA has established an RfD for inorganic arsenic of 3.0×10^{-4} mg/kg/d, based on a NOAEL (adjusted to include arsenic exposure from food) of 0.0008 mg/kg/d, an uncertainty factor of 3, and a modifying factor of 1 (U.S. EPA 1999). This was based on two studies (Tseng et al. 1968, and Tseng 1977, as cited in U.S. EPA 1999) that showed that the prevalence of blackfoot disease increased with both age and dose for individuals exposed to high levels of arsenic in drinking water. This same population also displayed a greater incidence of hyperpigmentation and skin lesions. Other human studies support these findings, with several studies noting an increase in skin lesions from chronic exposure to arsenic through the drinking water (Cebrian et al. 1983; Hindmarsh et al. 1977; Southwick et al. 1983, as cited in U.S. EPA 1999). An uncertainty factor of 3 was applied to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals (U.S. EPA 1999).

Reference Concentration. EPA has not established an RfC for inorganic arsenic (U.S. EPA 1999).

5.3.2 2,3,7,8-Tetrachlorodibenzo-p-Dioxin

2,3,7,8-TCDD belongs to the class of compounds, chlorinated dibenzo-p-dioxins, that are referred to as dioxins. 2,3,7,8-TCDD is a colorless solid with no known odor. It does not occur naturally nor is it intentionally manufactured by any industry, although it can be produced inadvertently in small amounts as an impurity during the manufacture of certain herbicides and germicides and has been detected in products of incineration of municipal and industrial wastes. The only current use for 2,3,7,8-TCDD is in chemical research (ATSDR 1997).

EPA issued a draft Health Assessment Document for 2,3,7,8-TCDD and Related Compounds in 1994. This document is a three-volume series consisting of a complete reassessment of the toxic effects of 2,3,7,8-TCDD (U.S.EPA 1994a, b). The document was reviewed by EPA's Science Advisory Board (SAB) but has not yet been issued in final form. Most of the information in this summary is from this draft document and is subject to change, pending the release of the final document.

Cancer Effects. A number of studies have found associations between exposure to 2,3,7,8-TCDD and several types of cancer. An increased incidence of soft tissue sarcoma was found to be elevated in several recent studies. EPA stated that (U.S.EPA 1994b):

> . . . the fact that similar results were obtained in independent studies of differing design and evaluating populations exposed to dioxin-like compounds under

> varying conditions, along with the rarity of this tumor type, weighs in favor of a consistent and real association. On the other hand, arguments regarding selection bias, differential exposure misclassification, confounding, and chance in each individual study have been presented in the scientific literature which increase uncertainty around this association. In addition excess respiratory cancer was noted in other studies. These results are also supported by significantly increased mortality from lung and liver cancers subsequent to the Japanese rice oil poisoning accident where exposure to PCDFs and PCBs occurred. Again, while smoking as a confounder cannot be totally eliminated as a potential explanation of these results, analyses conducted to date suggest that smoking is not likely to explain the entire increase in lung cancer. The question of confounding exposures, such as asbestos and other chemicals, in addition to smoking, has not been entirely ruled out and must be considered as potentially adding to the observed increases. Although increases of cancer at other sites (e.g., non-Hodgkin's lymphoma, stomach cancer) have been reported, the data for an association with exposure to dioxin-like compounds are less compelling.

Information on the carcinogenicity of 2,3,7,8-TCDD following inhalation exposure of animals is not available. In animal studies of oral exposure to 2,3,7,8-TCDD, multisite tumors in rats and mice, including the tongue, lung, nasal turbinates, liver, and thyroid, have been reported from long-term bioassays. It has also been shown to be carcinogenic in hamsters (U.S.EPA 1994b). EPA has classified 2,3,7,8-TCDD as a Group B2 - Probable Human Carcinogen (U.S.EPA 1997b).

Cancer Risk. EPA examined the available carcinogenicity data for 2,3,7,8-TCDD and concluded (U.S.EPA 1994b):

> With regard to carcinogenicity, a weight of evidence evaluation suggests that dioxin and related compounds (CDDs, CDFs, and dioxin-like PCBs) are likely to present a cancer hazard to humans. While major uncertainties remain, efforts of this reassessment to bring more data into the evaluation of cancer potency have resulted in a risk specific dose estimate (1×10^{-6} or one additional cancer in one million exposed) of approximately 0.01 pg TEQ/kg body weight/day. This risk specific dose estimate represents a plausible upper bound on risk based on the

evaluation of animal and human data. “True” risks are not likely to exceed this value, may be less, and may even be zero for some members of the population.

Dose-Response Modeling. In 1997, EPA concluded the following (U.S.EPA 1997a):

> It is clear from this analysis that dioxin causes a variety of toxicities in test animals following chronic and bolus exposures. The human data is less clear, but qualitatively and quantitatively consistent with the animal findings when expressed on the basis of steady-state body burden rather than a daily dose or area-under-the-curve basis. There are sufficient data suggesting response proportionate to dose to warrant concern that this compound will induce toxic effects in humans in the range of the experimental animal data. Also, based on a lack of data to argue for an immediate and steep change in slope for many of the responses analyzed there is the possibility of response 1 to 2 orders of magnitude below this range.

Oral Cancer Risk. EPA has derived an oral cancer slope factor of $1.5\text{x}10^5$ $(\text{mg/kg/d})^{-1}$ for 2,3,7,8,-TCDD based on a study in which rats were exposed to 2,3,4,8-TCDD in the diet. This exposure resulted in tumors of the respiratory system and liver (Kociba et al. 1978, as cited in U.S. EPA 1997b).

Inhalation Cancer Risk. EPA has calculated an inhalation cancer slope factor for 2,3,7,8-TCDD of $1.5\text{x}10^5$ $(\text{mg/kg/d})^{-1}$ and an inhalation unit risk estimate of $3.3\text{x}10^{-5}$ $(\text{pg/m}^3)^{-1}$. These values are under review and are subject to change; they are based on route-to-route extrapolation of the oral cancer slope factor described above.

Noncancer Effects. The first observed noncarcinogenic effect from exposure to 2,3,7,8-TCDD was chloracne, a severe acne-like condition that develops within months of first exposure to high levels of 2,3,7,8-TCDD. For many individuals, the condition disappears after discontinuation of exposure, for others it may remain for years. There are limited human data to suggest the doses at which chloracne is likely to occur. Occupational studies suggest that persistent chloracne is more often associated with high-intensity exposures, for long periods of time, and starting at an early age (U.S. EPA 1994a, b). Acute or chronic exposures to 2,3,7,8-TCDD at low levels have usually resulted in chloracne lasting for no longer than a few months to a few years (U.S.EPA 1994a, b).

Epidemiological studies have reported conflicting evidence on the immunotoxicity of 2,3,7,8-TCDD in humans. Some studies have suggested evidence of immunotoxicity, such as alterations in lymphocyte populations, cell surface markers, or lymphocyte proliferative response (ATSDR 1997). However, studies have not reported changes in the immune system directly related to 2,3,7,8-TCDD exposure (U.S.EPA 1994a, b).

An association has been reported between levels of male reproductive hormones and 2,3,7,8-TCDD exposure. Decreased testosterone levels were detected in several human studies, and animal data are available to support these findings. Other effects noted in human studies include an association between 2,3,7,8-TCDD exposure and

- An increased risk of diabetes and an elevated prevalence of abnormal fasting serum glucose levels

- The induction of cytochrome P-450 1A1, an enzyme involved in biotransformation reactions

- Elevation of gamma glutamyl transferase, a liver enzyme

- A possible increased risk of endometriosis, a disease of the female reproductive system (U.S.EPA 1994a, b).

Animal studies have reported reproductive and developmental effects from exposure to 2,3,7,8-TCDD. These studies have suggested that altered development may be among the most sensitive endpoints of 2,3,7,8-TCDD exposure since developmental toxicity occurred at lower levels than male and female reproductive toxicity. 2,3,7,8-TCDD appears to cause a large number of critical developmental effects at specific developmental stages leading to increases in fetal mortality, disruption of organ system structure, and irreversible impairment of organ function. Developmental toxicity from 2,3,7,8-TCDD has been seen in fish, birds, and mammals. Thus, it is likely to occur at some level in humans. However, it is not possible to state what sort of effects will occur or at what levels (U.S.EPA 1994a, b).

Animal studies have reported changes in the skin resembling chloracne from 2,3,7,8-TCDD exposure. Distinctive changes in animals include swelling and inflamed eyelids, nail loss, and facial hair loss (ATSDR 1997).

The immune system also appears to be affected by 2,3,7,8-TCDD exposure in animal studies. Alterations in specific immune effector functions and increased susceptibility to infectious diseases have been observed in animals exposed to 2,3,7,8-TCDD. Both cell-mediated and humoral immune responses were suppressed following 2,3,7,8-TCDD exposure (U.S.EPA 1994a, b).

EPA has not calculated an RfD or an RfC for 2,3,7,8-TCDD.

5.4 Uncertainty Analysis

EPA typically classifies the major areas of uncertainty in risk assessments as parameter uncertainty, scenario uncertainty, and model uncertainty. Parameter uncertainty is the "uncertainty regarding some parameter" of the analysis. Scenario uncertainty is "uncertainty regarding missing or incomplete information needed to fully define exposure and dose." Model uncertainty is "uncertainty regarding gaps in scientific theory required to make predictions on the basis of causal inferences" (U.S.EPA 1992). This section identifies the primary sources of each of these types of uncertainty in the chlorinated aliphatics risk assessment, and qualitatively describes how each may influence the results of the risk assessment.

Parameter Uncertainty

The sources of parameter uncertainty are measurement errors, sampling errors, variability, and use of generic or surrogate data (U.S.EPA 1992). Many of the parameters that we used to quantify contaminant fate and transport and contaminant exposure and dose either were not measured or could not be measured precisely and/or accurately. Some of the most important and sensitive parameters in our analyses include those that describe waste composition; waste management practices; site characteristics (for example, hydrogeological, topographical, meteorological, and soils data); the physiologic and behavioral exposure characteristics of the receptors; the physical, chemical, and biochemical properties of the contaminants; and toxicological effects.

We believe that the primary sources of parameter uncertainty in the chlorinated aliphatics risk assessment include the following:

- The risk analyses were based on a limited set of waste sample (concentration) data. It is possible that these data do not represent the true distribution of contaminant concentrations in the wastes evaluated, resulting in either an overestimation or underestimation of the actual risk to receptors. The sensitivity of the models to waste concentrations, the small sample size, the high demonstrated variability of contaminant concentrations across facilities, and difficulty in obtaining samples that represent only chlorinated aliphatics waste streams presented significant uncertainty issues for EPA. The objective of sampling is to characterize the waste produced by a particular industrial process. If the available samples are not truly representative of the waste, risk could be underestimated or overestimated.

- EPA obtained very little site-specific information regarding the design and operation of waste management units used by the chlorinated aliphatics industry, necessitating that we make a number of assumptions regarding waste management in off-site landfills, the land treatment unit, and wastewater tanks. Many of the facilities reported using off-site nonhazardous landfills to dispose of EDC/VCM sludges. We assumed that these landfills are municipal landfills, and modeled typical municipal landfills based on available data (such as data that describe the size of municipal landfills). Our major assumptions about the municipal landfills that have the effect of decreasing our risk estimates are that the landfills have daily covers and run-on/run-off controls. Our major assumptions about the municipal landfills that have the effect of increasing our risk estimates are that the landfills are not lined and have no leachate collection systems. For the land treatment unit, we assumed that no run-on/run-off controls were present to mitigate risk. We assumed that the industry’s wastewater treatment tanks are uncovered (which increases our risk estimates), are aerated (which increases our risk estimates), employ biological treatment techniques (which decreases our risk estimates), have structural integrity (which decreases our risk estimates), and have spill and overflow controls (which decreases our risk estimates).

- In our analysis of chromium and mercury fate and transport in the environment we evaluated the chromium and mercury species that would tend to maximize risk to the receptors. Although we evaluated chromium and mercury in this highly protective way, we ultimately did not identify either chromium or mercury as a COC, and as a result, our uncertainty regarding chromium and mercury fate and transport does not influence the conclusions of our analysis.

- We typically used regional databases to obtain the parameter values necessary to model contaminant fate and transport. Because the data that we used are not specific to the facilities at which the actual wastes are managed, the data represent our best estimates of actual site conditions. Use of these databases in lieu of site-specific data may result in either overestimates or underestimates of risk.

- Sources of uncertainty in toxicological benchmarks include one or more of the following: extrapolation from laboratory animal data to humans, variability of response within the human population, extrapolation of responses at high experimental doses under controlled conditions to low doses under highly variable environmental conditions, and adequacy of the database (number of studies available, toxic endpoints evaluated, exposure routes evaluated, sample sizes, length of study, etc.). Toxicological benchmarks are designed to be conservative (that is, overestimate risk) because of the uncertainties and challenges associated with condensing toxicity data into a single quantitative expression. Therefore, use of the current toxicological benchmarks most likely overestimated risk for the pathways evaluated. In addition, we did not develop and use PDFs that describe the uncertainty in our toxicological benchmarks in our probabilistic risk analyses.

- CSFs can vary by several orders of magnitude depending on the extrapolation model used. A limited understanding of cancer biology in laboratory animals and humans adds to the uncertainty of identifying true human carcinogens. The COCs identified in this report include arsenic and dioxins. Arsenic is a known human carcinogen. Dioxins are considered probable human carcinogens. The dioxin risk assessment is particularly complicated because there are numerous congeners that make up a dioxin sample and the distribution of congeners is variable across waste samples.

- EPA estimated the risk of developing cancer from the estimated lifetime average daily dose and the slope of the dose-response curve. A cancer slope factor is derived from either human or animal data and is taken as the upper bound on the slope of the dose-response curve in the low-dose region, generally assumed to be linear, expressed as a lifetime excess cancer risk per unit exposure. However, individuals exposed to carcinogens in the first few years of life may be at increased risk of developing cancer. For this reason, EPA recognizes that significant uncertainties and unknowns exist regarding the estimation of lifetime cancer risks in children. We also note that the analysis of cancer risks in children has not been externally peer reviewed.

Scenario Uncertainty

The sources of scenario uncertainty are descriptive errors, aggregation errors, errors in professional judgment, and incomplete analysis (U.S.EPA 1992). Scenario uncertainty results from the assumptions we make regarding how receptors become exposed to contaminants, and occurs because of the difficulty and general impracticality of making actual measurements of a receptor's exposure.

In certain cases our analysis may have been incomplete, for example, some scenarios that we omitted from this analysis include:

- Evaluation of risks to infants (age 0 to 1) and
- Evaluation of the wet deposition of particles onto plant surfaces

As discussed previously in this document, we expect that evaluation of these additional scenarios or pathways would increase our estimates of risk, but that the increases would likely be small and not impact the overall results of the risk assessment.

Our exposure modeling relied heavily on default assumptions regarding population activity patterns, mobility, dietary habits, body weights, and other factors. These default assumptions may be a source of aggregation error because we assume the populations that reside near the chlorinated aliphatics waste management units are homogeneous and are representative of the national population. Because our risk estimates are for hypothetical chronic exposures and are designed to provide a realistic range of potential receptor exposure scenarios, we develop predictions of long-term average exposures for each receptor. Although it is possible to study various populations to determine their exposure parameters (for example, age-specific soil ingestion rates or intake rates for food) or to assess past exposures (epidemiological studies) or current exposures, risk assessment is about prediction. Therefore, long-term exposure monitoring in this context is infeasible. The double-high end deterministic approach coupled with the probabilistic approach is designed to provide reasonable estimates of potential long-term exposures for various receptors. The *Exposure Factors Handbook* (U.S.EPA 1997c) provides the current state-of-the-science regarding exposure modeling and assumptions and was used in this risk assessment. To the extent that actual exposure scenarios vary from the assumptions we used, risks could be underestimated or overestimated. However, although there could be individuals living near a chlorinated aliphatics waste management unit who have higher exposures than those presented, it is more likely that actual exposures for most of these individuals would fall within the predicted range, and, moreover, would be similar to those predicted for the central tendency or 50th percentile.

Model Uncertainty

The sources of model uncertainty are relationship errors and modeling errors (U.S.EPA 1992). Models and their mathematical expressions are simplifications of reality that are used to approximate real-world conditions and processes, and their relationships. Models do not include all parameters or equations necessary to express reality because of the inherent complexity of the natural environment, and the lack of sufficient data to describe the natural environment. Consequently, models are based on numerous assumptions and simplifications, and reflect an incomplete understanding of natural processes. We selected the models used in this risk assessment, described in Section 3, based on science, policy, and professional judgment. We selected the wastewater emissions model, the air dispersion and deposition models, the indirect exposure equations, and the groundwater model because they provide the information needed for this analysis and because we generally consider them to be state-of-the-science.

Even though the models used in the risk analyses are used widely and have been accepted for numerous applications, they each retain significant sources of uncertainty. The following are some examples of these uncertainties:

- All of our models include assumptions that environmental processes are at equilibrium or have reached steady-state. These assumptions may result in overestimations or underestimations of risk in some cases.

- CHEMDAT8 (used to model air emissions from tanks) is considered to provide reasonable to slightly high estimates of air emissions, thus potentially overestimating risk in some cases.

- EPACMTP (used to model groundwater fate and transport) does not model colloidal transport, nor does it model possible geochemical interactions between different contaminants in the leachate. Either of these factors could result in underpredicting contaminant concentrations at the receptor well. Conversely, our EPACMTP modeling incorporates the following assumptions: (1) transverse dispersion is negligible in the unsaturated zone, potentially resulting in an overestimation of risks; (2) receptors use the uppermost aquifer, rather than a deeper aquifer, as a domestic source of drinking water, which, overestimates risks where the uppermost aquifer is not used; and (3) hydrogeologic conditions that influence contaminant fate and transport are uniform spatially, as well as uniform temporally (that is, in the period of time over which we model groundwater fate and transport, 10,000 years), potentially resulting in our underestimating or overestimating receptor well concentrations.

- ISCST3 (used to model air dispersion and wet deposition of vapors) does not include photochemical reactions or degradation of a chemical in the air, which results in additional model uncertainty for some chemicals. Dispersion modeling is highly sensitive to meteorological data and the surface area and dimensions of the waste management unit. Meteorological data used in the dispersion modeling include wind-speed and direction, temperature, precipitation type and amount, and stability class, among others. ISCST3 currently does not calculate dry deposition of vapors; however, the next version of ISCST3 will include this option. We used a conservative approach for modeling dry deposition as discussed in Section 3.3. The ISCST3 model uses hourly data as inputs, and this analysis used 5 years of hourly data to develop long-term unitized air concentrations (UACs).

- USLE (used to model erosion) was designed as a planning tool to predict longtime average soil losses from sheet and rill erosion. It is based on an empirical soil loss equation that is most accurate for medium textured soils, slope lengths of less than 400 feet, gradients of 3 to 18 percent, and conditions that were represented in the erosion plot studies used to derive the equation (USDA, 1978). However, it does not predict deposition. Deposition onto the intervening fields or residential plot is based on mass balance calculations.

Evaluated as a whole, the sources of model uncertainty in our analysis could result in either an overestimation or underestimation of risk.

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Addendum
to the
Risk Assessment Technical Background Document for the Chlorinated Aliphatics Listing Determination (July 30, 1999)

United States Environmental Protection Agency
Office of Solid Waste
July 30, 1999

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Introduction

This document is an addendum to the Environmental Protection Agency's (EPA's) July 30, 1999 draft of the "Risk Assessment Technical Background Document for the Chlorinated Aliphatics Listing Determination" (Risk Assessment Technical Background Document). The purpose of this addendum is 1) to address several technical issues that came to EPA's attention since EPA initially drafted the July 30,1999 Risk Assessment Technical Background Document and 2) to present some limited analyses that have been completed since the analyses supporting the Risk Assessment Technical Background Document initially were conducted. We classify the issues by the following topic areas:

1. Soil-to-Plant Uptake of Dioxins
2. Empirical Correction Factor (VG_{bg}) for the Root Concentration Factor (RCF)
3. Wet Deposition of Particulates onto Plants
4. Uncertainty Associated with Ambient Vapor/Particle Partitioning
5. Probabilistic Risk Analysis for Direct Inhalation of Chloroform Emitted from Chlorinated Aliphatic Wastewater Treatment Tanks
6. Screening Ecological Risk Assessment for the Wastewater Treatment Tank Scenario
7. Analyses to Support the Development of a 2,3,7,8-TCDD Toxicity Equivalent (TEQ) Limit for Chlorinated Aliphatics Wastewaters
8. Clarification of the Location of Modeled Pastureland Relative to the Location of Waste Management Units.

Sections 1 through 8 of this Addendum address each of these topics. Section 9 provides technical errata for the Risk Assessment Technical Background Document. Section 10 provides references for this addendum.

It is important to note, as discussed below, that after evaluating these issues and conducting additional analyses, we have determined that they do not change any of our regulatory conclusions and decisions. We are soliciting comment on these issues and analyses and intend to incorporate them, as appropriate, into the analyses to support the final rule.

1. Soil-to-Plant Uptake of Dioxins

EPA evaluated soil to above-ground plant uptake of dioxins using a plant-soil bioconcentration factor for above-ground produce (“Br”) calculated using an equation in Travis and Arms (1988). Given more recent information and analyses conducted by EPA that indicate that soil to above-ground plant uptake of dioxins is negligible, our evaluation of soil-to-plant uptake of dioxins by above-ground vegetation appears needlessly conservative (U.S.EPA 1994; Trapp and Matthies 1995).

To evaluate the impact of considering uptake of dioxins from soil, we recalculated high end deterministic above-ground vegetable concentrations, beef concentrations, and dairy concentrations for the EDC/VCM land treatment unit and wastewater tank scenarios (for the farmer receptor), removing the contribution of dioxins that would occur through soil-to-plant uptake. The results of this analysis are presented in Table 1a for the wastewater treatment tank scenario and Table 1b for the land treatment unit scenario. The conclusions of this analysis follows.

Ingestion of Above-Ground Vegetables.

- For the land treatment unit (LTU) scenario, considering soil-to-plant transfer of dioxins increases the concentrations of dioxins in above-ground vegetables by approximately a factor of 3. This result is relatively insignificant in terms of the risk results because only 0.4 percent of the farmer’s risk under the LTU high end scenario is attributable to ingestion of above-ground vegetables (Table 5-8 of the July 30th document).
- For the tank scenario, considering soil-to-plant transfer of dioxins does not appreciably affect the concentrations of dioxins in above ground vegetables (because the plants’ uptake primarily is through air-to-plant transfer of dioxin vapor). Moreover, only 0.1 percent of the farmer’s risk the wastewater tank high end scenario is attributable to ingestion of above-ground vegetables (Table 5-3 of the July 30th document).

Table 1a.
Concentrations of Dioxins in Exposed Vegetables, Beef, and Dairy Products When Plant Uptake From Soil is Eliminated Wastewater Treatment Tank Scenario, High End Deterministic Analysis

Chemical	CAS No.	TEF	Exposed Vegetable Concentration (Including plant uptake from soil) (mg/kg)	Exposed Vegetable Concentration (No plant uptake from soil) (mg/kg)	Beef Concentration (Including plant uptake from soil) (mg/kg)	Beef Concentration (No plant uptake from soil) (mg/kg)	Dairy Concentration (Including plant uptake from soil) (mg/kg)	Dairy Concentration (No plant uptake from soil) (mg/kg)
TCDD, 2,3,7,8-	1746-01-6	1	2.57E-11	2.48E-11	1.54E-09	1.54E-09	3.29E-10	3.28E-10
OCDD, 1,2,3,4,5,7,8,9-	3268-87-9	0.001	4.33E-12	4.33E-12	3.05E-11	3.05E-11	5.71E-12	5.71E-12
HxCDD, 1,2,3,7,8,9-	19408-74-3	0.1	9.34E-10	9.32E-10	2.46E-08	2.46E-08	7.38E-09	7.38E-09
HpCDD, 1,2,3,4,6,7,8,-	35822-46-9	0.01	9.13E-10	9.12E-10	4.82E-09	4.82E-09	1.20E-09	1.20E-09
OCDF, 1,2,3,4,6,7,8,9-	39001-02-0	0.001	4.64E-10	4.64E-10	2.04E-09	2.04E-09	6.12E-10	6.12E-10
HxCDD, 1,2,3,4,7,8-	39227-28-6	0.1	2.11E-10	2.11E-10	5.57E-09	5.57E-09	1.67E-09	1.67E-09
TCDF, 2,3,7,8-	51207-31-9	0.1	3.12E-10	3.02E-10	2.67E-09	2.66E-09	1.20E-09	1.20E-09
HpCDF,1,2,3,4,7,8,9-	55673-89-7	0.01	1.97E-08	1.97E-08	1.73E-07	1.73E-07	7.79E-08	7.79E-08
PeCDF, 2,3,4,7,8-	57117-31-4	0.5	1.78E-09	1.77E-09	7.79E-08	7.79E-08	2.10E-08	2.10E-08
HxCDF, 1,2,3,6,7,8-	57117-44-9	0.1	1.75E-09	1.74E-09	4.60E-08	4.60E-08	1.38E-08	1.38E-08
HxCDD, 1,2,3,6,7,8-	57653-85-7	0.1	7.72E-10	7.71E-10	2.04E-08	2.04E-08	5.09E-09	5.09E-09
HxCDF, 2,3,4,6,7,8-	60851-34-5	0.1	1.02E-09	1.02E-09	2.68E-08	2.68E-08	6.71E-09	6.71E-09
HpCDF,1,2,3,4,6,7,8-	67562-39-4	0.01	1.97E-08	1.97E-08	1.04E-07	1.04E-07	2.60E-08	2.60E-08
HxCDF, 1,2,3,4,7,8-	70648-26-9	0.1	1.75E-08	1.74E-08	6.14E-07	6.13E-07	1.61E-07	1.61E-07
HxCDF, 1,2,3,7,8,9-	72918-21-9	0.1	NA	NA	NA	NA	NA	NA
TEQ			3.57E-09	3.55E-09	1.17E-07	1.17E-07	3.16E-08	3.16E-08

High end parameters: Exposure duration and waste concentration

Table 1b.
Concentrations of Dioxins in Exposed Vegetables, Beef, and Dairy Products When Plant Uptake From Soil is Eliminated Land Treatment Unit Scenario, High End Deterministic Analysis

Chemical	CAS No.	TEF	Exposed Vegetable Concentration (Including plant uptake from soil) (mg/kg)	Exposed Vegetable Concentration (No plant uptake from soil) (mg/kg)	Beef Concentration (Including plant uptake from soil) (mg/kg)	Beef Concentration (No plant uptake from soil) (mg/kg)	Dairy Concentration (Including plant uptake from soil) (mg/kg)	Dairy Concentration (No plant uptake from soil) (mg/kg)
TCDD, 2,3,7,8-	1746-01-6	1	1.18E-08	9.47E-10	1.28E-07	1.19E-07	2.10E-08	1.87E-08
OCDD, 1,2,3,4,5,7,8,9-	3268-87-9	0.001	1.37E-06	5.71E-07	5.90E-06	5.83E-06	9.33E-07	9.16E-07
HxCDD, 1,2,3,7,8,9-	19408-74-3	0.1	1.51E-08	5.55E-09	2.05E-07	2.01E-07	5.30E-08	5.19E-08
HpCDD, 1,2,3,4,6,7,8-	35822-46-9	0.01	6.23E-08	2.14E-08	2.72E-07	2.69E-07	4.78E-08	4.69E-08
OCDF, 1,2,3,4,6,7,8,9-	39001-02-0	0.001	1.10E-05	5.82E-06	6.27E-05	6.24E-05	1.31E-05	1.30E-05
HxCDD, 1,2,3,4,7,8-	39227-28-6	0.1	NA	NA	NA	NA	NA	NA
PeCDD, 1,2,3,7,8-	40321-76-4	0.5	NA	NA	NA	NA	NA	NA
TCDF, 2,3,7,8-	51207-31-9	0.1	5.43E-08	4.04E-09	7.60E-08	7.01E-08	2.65E-08	2.34E-08
HpCDF,1,2,3,4,7,8,9-	55673-89-7	0.01	2.13E-06	1.20E-06	1.47E-05	1.46E-05	5.68E-06	5.62E-06
PeCDF, 2,3,4,7,8-	57117-31-4	0.5	4.41E-08	1.27E-08	7.58E-07	7.39E-07	1.80E-07	1.74E-07
PeCDF, 1,2,3,7,8-	57117-41-6	0.05	NA	NA	NA	NA	NA	NA
HxCDF, 1,2,3,6,7,8-	57117-44-9	0.1	NA	NA	NA	NA	NA	NA
HxCDD, 1,2,3,6,7,8-	57653-85-7	0.1	2.02E-08	7.43E-09	2.74E-07	2.69E-07	5.91E-08	5.78E-08
HxCDF, 2,3,4,6,7,8-	60851-34-5	0.1	1.21E-07	2.04E-08	1.15E-06	1.12E-06	2.14E-07	2.04E-07
HpCDF,1,2,3,4,6,7,8-	67562-39-4	0.01	3.27E-06	1.85E-06	1.35E-05	1.34E-05	2.90E-06	2.87E-06
HxCDF, 1,2,3,4,7,8-	70648-26-9	0.1	2.64E-07	4.95E-08	3.49E-06	3.39E-06	6.96E-07	6.65E-07
HxCDF, 1,2,3,7,8,9-	72918-21-9	0.1	NA	NA	NA	NA	NA	NA
TEQ			1.48E-07	5.31E-08	1.38E-06	1.34E-06	3.16E-07	3.05E-07

Ingestion of Beef and Dairy Products. For both the land treatment unit and tank scenarios, considering soil-to-plant transfer of dioxins produces no significant effect on the concentrations of dioxins in beef and dairy products, since most of the dioxins in forage consumed by cattle comes from air to plant transfer (vapor phase).

2. Empirical Correction Factor (VG_{bg}) for the Root Concentration Factor (RCF)

EPA applies an empirical correction factor, $VG_{bg,}$ to the root concentration factor (RCF) that is used to calculate the concentrations of contaminants in root vegetables. This correction factor accounts for the difference in size between the roots evaluated in experiments used to derive the RCF and the root crops that we evaluate in our risk assessment. In the risk assessment conducted to support the chlorinated aliphatics listing determination, we used a value of VG_{bg} of 0.01, which we obtained from the *Parameter Guidance Document* (U.S.EPA 1997) and from *Estimating Exposure to Dioxin-Like Compounds* (U.S.EPA 1994). More recent studies in progress at EPA indicate that this correction factor should be 0.25.

Use of a VG_{bg} factor of 0.25, rather than 0.01, would increase the concentration of dioxins in root vegetables by a factor of 25. However, the contribution of ingestion of root vegetables to the overall risk for the farmer under the high end EDC/VCM LTU scenario is only 0.2 percent (Table 5-8 of the July 30th document) and the contribution of ingestion of root vegetables to the overall risk for the farmer under the high end wastewater treatment tank scenario is 0.0004 percent (Table 5-3 of the July 30th document). Therefore, changing the value of VG_{bg} to 0.25 would not significantly influence the overall risks for these scenarios. Moreover, changing our value for VG_{bg} would increase risks that already are in the range that is significant to the listing program, so our listing decisions for these scenarios would not change.

3. Wet Deposition of Particulates onto Plants

Particle deposition onto plants occurs under both wet and dry meteorological conditions. Although EPA typically incorporates both wet deposition of particles and dry deposition of particles into our analyses, we neglected to account for wet deposition of particles onto plants in the chlorinated aliphatics risk assessment. Particle deposition onto plants typically is driven by dry deposition of particles, since, in most locations, dry meteorological conditions predominate. Nevertheless, we conducted an analysis to determine how considering wet deposition of particulates onto plants would influence our risk results. Our analysis indicated that evaluating wet deposition of particulates onto plants would account for less than 1 percent of the total contaminant contribution from particulate deposition, that is, both wet and dry particulate deposition. Consequently, including the evaluation of wet deposition of particulates onto plants in our models would not have influenced our risk results. Table 2 presents the proportion of contaminant concentrations in exposed vegetables attributable to both wet and dry particle deposition versus dry deposition for the central tendency farmer (land treatment unit scenario).

4. Uncertainty Associated with Ambient Vapor/Particle Partitioning

Following an internal review of a draft of the July 30, 1999 Risk Assessment Technical Background Document, we became aware of a source of uncertainty in our analysis that was not presented in the document. That uncertainty is incorporated into our assumption that vapor emissions of dioxins from chlorinated aliphatics wastewaters and wastewater treatment sludges do not appreciably sorb to particulate matter in the ambient air in approximately 1.2 minutes, the average the time required for emissions from the waste management units to reach a receptor located 300 meters away (our central tendency distance to receptor). Sorption of dioxins onto particles in air would remove dioxins from the vapor phase, thereby reducing the vapor-phase diffusion of dioxins into plants. As a result, our calculated dioxin concentrations in plants, and in animals consuming plants (particularly grasses), are higher than they would be if we assumed that some fraction of the vapor phase dioxin irreversibly partitions onto particles in the ambient air. Given the uncertainties regarding rates of dioxin partitioning, magnitude of partitioning, and other factors potentially influencing dioxin sorption onto particles (such as temperature, humidity, and particle size, type and density), we believe our assumption that dioxins remain as vapors during their transport from the waste management unit source to the receptor location is appropriate. Because we understand that our assumption results in increased risk estimates, we are soliciting public comment on this issue. We also charged peer reviewers with providing comment on the issue during the peer review process.

Table 2.
Proportion of Total (Wet and Dry) Particle Deposition Attributable to Dry Deposition

Chemical	CAS	Exposed Vegetable Concentration (ug/g Dry Weight)		
		Dry Deposition of Particles	Wet and Dry Deposition of Particles	Ratio of Dry to Wet and Dry
Benzoic acid	65-85-0	4.50E-06	4.50E-06	0.9998
Acetone	67-64-1	9.26E-08	9.52E-08	0.9734
Chloroform	67-66-3	1.23E-08	1.29E-08	0.9531
Vinyl chloride	75-01-4	6.47E-10	6.82E-10	0.9489
Methylene chloride	75-09-2	1.64E-09	1.72E-09	0.9525
Carbon disulfide	75-15-0	1.01E-09	1.07E-09	0.9491
Methyl ethyl ketone	78-93-3	7.06E-09	7.22E-09	0.9786
Trichloroethylene	79-01-6	2.24E-10	2.35E-10	0.9528
Allyl chloride	107-05-1	4.50E-10	4.75E-10	0.9493
Dichloroethane, 1,2-	107-06-2	1.34E-08	1.40E-08	0.9585
Vinyl acetate	108-05-4	5.77E-10	6.01E-10	0.9590
Bis(2-chlorethyl)ether	111-44-4	5.23E-07	5.25E-07	0.9961
Bis(2-ethylhexyl)phthalate	117-81-7	1.02E-05	1.02E-05	0.9991
Hexachlorobenzene	118-74-1	4.04E-07	4.06E-07	0.9959
Tetrachloroethylene	127-18-4	7.31E-10	7.69E-10	0.9510
TCDD, 2,3,7,8-	1746-01-6	NA	NA	NA
OCDD, 1,2,3,4,5,7,8,9-	3268-87-9	4.78E-07	4.79E-07	0.9986
Aluminum	7429-90-5	4.13E-02	4.35E-02	0.9484
Lead	7439-92-1	3.03E-05	3.17E-05	0.9550
Manganese	7439-96-5	7.38E-01	7.38E-01	0.9999
Molybdenum	7439-98-7	5.20E-04	5.20E-04	0.9999
Nickel	7440-02-0	5.44E-02	5.44E-02	0.9998
Arsenic	7440-38-2	1.16E-02	1.16E-02	0.9998
Barium	7440-39-3	7.27E-02	7.27E-02	0.9999
Cadmium	7440-43-9	2.11E-05	2.11E-05	0.9998
Chromium VI	7440-47-3	4.61E-02	4.61E-02	0.9994
Cobalt	7440-48-4	5.10E-04	5.10E-04	0.9992
Copper	7440-50-8	1.69E+00	1.69E+00	1.0000
Vanadium	7440-62-2	2.51E-03	2.51E-03	0.9992
Zinc	7440-66-6	2.93E-01	2.93E-01	0.9999
HxCDD, 1,2,3,7,8,9-	19408-74-3	1.42E-09	1.43E-09	0.9990
HpCDD, 1,2,3,4,6,7,8,-	35822-46-9	1.91E-08	1.92E-08	0.9964
OCDF, 1,2,3,4,6,7,8,9-	39001-02-0	5.73E-07	5.77E-07	0.9943
HxCDD, 1,2,3,4,7,8-	39227-28-6	1.27E-09	1.27E-09	0.9981
PeCDD, 1,2,3,7,8-	40321-76-4	NA	NA	NA
TCDF, 2,3,7,8-	51207-31-9	4.20E-10	4.20E-10	0.9994
HpCDF,1,2,3,4,7,8,9-	55673-89-7	7.00E-08	7.01E-08	0.9985
PeCDF, 2,3,4,7,8-	57117-31-4	3.91E-09	3.91E-09	0.9993
PeCDF, 1,2,3,7,8-	57117-41-6	3.46E-09	3.46E-09	0.9994
HxCDF, 1,2,3,6,7,8-	57117-44-9	1.60E-08	1.60E-08	0.9986
HxCDD, 1,2,3,6,7,8-	57653-85-7	1.98E-09	1.99E-09	0.9990
HxCDF, 2,3,4,6,7,8-	60851-34-5	1.37E-08	1.37E-08	0.9986
HpCDF,1,2,3,4,6,7,8-	67562-39-4	3.56E-07	3.57E-07	0.9985
HxCDF, 1,2,3,4,7,8-	70648-26-9	2.05E-08	2.05E-08	0.9987
HxCDF, 1,2,3,7,8,9-	72918-21-9	7.40E-09	7.41E-09	0.9986

5. Probabilistic Risk Analysis for Direct Inhalation of Chloroform Emitted from Chlorinated Aliphatic Wastewater Treatment Tanks

The July 30, 1999 draft of the Risk Assessment Technical Background Document does not provide probabilistic risk assessment results for the direct inhalation of chloroform from aerated biological wastewater treatment tanks. Although the deterministic results for chloroform initially were not deemed high enough to warrant conducting a probabilistic risk analysis, we reconsidered our decision to evaluate the chloroform risks probabilistically, and provide the results in this Addendum for the sake of completeness. Attachment 1 provides detailed results of the probabilistic risk analysis for chloroform, and Table 3 (below) provides a summary of the results.

Table 3. Excess Lifetime Chloroform Cancer Risk, Tank Scenario, Probabilistic Results, Non-Groundwater Pathways (Direct Inhalation of Vapor)

	Percentile				
Receptor	**50th**	**90th**	**95th**	**97.5th**	**100th**
Farmer	3E-08	6E-07	1E-06	2E-06	6E-05
Home Gardener	2E-08	5E-07	1E-06	2E-06	5E-05
Resident	2E-08	5E-07	1E-06	2E-06	5E-05
Fisher	2E-08	5E-07	1E-06	2E-06	5E-05
Child Resident					
Age 1-5	3E-08	6E-07	1E-06	2E-06	6E-05
Age 6-11	3E-08	6E-07	1E-06	2E-06	5E-05
Age 12-18	2E-08	4E-07	8E-07	1E-06	4E-05
Child of Farmer					
Age 1-5	6E-08	1E-06	2E-06	4E-06	6E-05
Age 6-11	4E-08	8E-07	2E-06	3E-06	5E-05
Age 12-18	3E-08	6E-07	1E-06	2E-06	4E-05

6. Screening Ecological Risk Assessment for the Wastewater Treatment Tank Scenario

The Screening Ecological Risk Assessment presented in Appendix J of the July 30, 1999 draft of the Risk Assessment Technical Background Document does not address ecological risks attributable to management of chlorinated aliphatics wastewaters in aerated, biological treatment tanks. Attachment 2 provides the Screening Ecological Risk Assessment for the Wastewater Treatment Tank Scenario. Based on the risk results, we do not anticipate significant risk for the ecological receptors evaluated under either the high end or central tendency chlorinated aliphatics wastewater tank scenarios.

7. Analyses to Support the Development of a 2,3,7,8-TCDD TEQ Limit for Chlorinated Aliphatics Wastewaters

The July 30, 1999 draft of the Risk Assessment Technical Background Document does not provide the analyses the EPA used to support the development of the TCDD TEQ limit for wastewaters that is described in the preamble to the proposed rule. As stated in the preamble, after considering options for setting the TCDD TEQ limit, we chose to base the TCDD TEQ limit on the lowest TCDD TEQ concentration measured in a dedicated wastewater sample for which a high end deterministic risk estimate is 1 x 10^{-5}. This concentration is 0.64 ng/L, calculated using the TEFs developed by the World Health Organization, and corresponding to the TCDD TEQ concentration for EPA's sample no. PL-02. (The TCDD TEQ concentration based on the International toxicity equivalency factors [I-TEFs] is 0.66 ng/L. See the Risk Assessment Technical Background Document for a explanation of the TEFs). These results are presented in Table 1 of Attachment 3 to this Addendum. The high end deterministic risk estimate was based on the evaluation of a farmer scenario in which the exposure duration of the farmer was set at its high end value, 48.3 years. For the purpose of establishing the TCDD TEQ limit, we did not set any additional values at high end. We used the "single high end" approach to account for sources of uncertainty in the risk analysis and our understanding that not all of the underlying assumptions of the analysis may be relevant to any one chlorinated aliphatics facility. For example, not all facilities may operate the type of aerated biological treatment tank that was modeled, grazing of cattle may not occur in the vicinity of all facilities on the centerline of the contaminant plume (the farmer's risk primarily is due to the ingestion of contaminated beef and dairy products).

For reference, the adult resident's "single high end" risk is 1 x 10^{-9} when the wastewater TCDD I-TEQ concentration is 0.66 ng/L (based on meteorological location as a high end parameter). These results are presented in Table 2 of Attachment 3. The wastewater sample with the next lowest TCDD I-TEQ concentration (0.31 ng/L, sample PL-01) produces a single high end risk estimate for the farmer of 2 x 10^{-6}. These results are presented in Table 3 of Attachment 3.

8. Clarification of the Location of Modeled Pastureland Relative to the Location of Waste Management Units

This section provides clarification regarding EPA's assumptions about the location of pastureland relative to the location of waste management units. It also explains in greater detail how EPA calculates the concentrations of contaminants in pastureland. The primary sources of contaminants ingested by cattle raised on pastureland are the pasture grasses (forage) and pastureland soils. Pastureland grass and soil may be contaminated by transport of contaminants in the air and transport of contaminants in runoff/erosion.

In our deterministic analyses for these pathways EPA assumes that receptors are located either 75 meters (m) or 300m from the waste management unit. In our deterministic analyses, the distance to the pastureland and cattle (the distance to the farmer receptor) was set at 300 m for both the EDC/VCM land treatment unit and the chlorinated aliphatics wastewater tank. For the probabilistic analysis, we assume the receptors are located 50, 75, 100, 200, 300, 500, or 1000m from the waste management unit.

When we evaluate runoff/erosion, we must specify the size of the farm onto which the erosion occurs because the size of the farm establishes the area over which eroded soil is distributed. We assume that the eroded waste is distributed evenly over the farm, such that the concentration of a contaminant at one point on the farm is no different than the concentration on another part of the farm. The size of agricultural farm that we evaluated was 2,000,000m^2, or approximately 1400 by 1400m.

When we conduct air dispersion modeling, we calculate both contaminant concentrations in air and deposition of contaminants at specified points around the waste management unit.

For both the deterministic and probabilistic analyses, the receptor points were modeled at 64 and 32 directions, respectively, for tanks and land-based units (the land treatment unit and the landfills). We always assume that the receptors live in the direction associated with the highest air releases from the waste management. That is, we assume that the air concentration at the receptor location (the pasture) is the maximum of the 64 (or 32) air concentrations for the receptor distance evaluated (for our deterministic analyses, 300 meters). While using the maximum point estimate as the concentration over the grazing range of the cattle appears to be very conservative, in fact the concentrations do not change that much as one moves away from that particular point. Table 4 presents air concentrations for 64 points located 300m around the central tendency wastewater treatment tank. These points are located approximately 30 meters apart. The difference between the maximum concentration and the average concentration over an arc of about 200 meters around that point is only about 20%.

9. Technical Errata

Certain equations in Appendix E of the Risk Assessment Technical Background Document include a variable Fv (or [1-Fv]), which is the fraction of the constituent concentration that is in the vapor phase. This factor partitions a total air concentration into the fraction that is in the vapor phase and the fraction that is in the particle phase. However, because we calculated vapor and particulate emissions separately, and thus calculated dispersion and deposition of particle and vapor emissions separately, we actually did not need to implement the "Fv" portion of these equations as we would have if we had started with "total" (vapor plus particulate) air emissions. Similarly, the values for Fv that are included in Appendix C of the Risk Assessment Technical Background Document are not necessary.

Table 4. Concentrations of Dioxins in Air at 64 Points Located 300 Meters from the Wastewater Treatment Tank Central Tendency Location, Memphis, TN

Receptor Direction	Concentration (ug/m^3 / $mg/s\text{-}m^2$)
SW	1.2568
	1.36036
	1.5424
	1.84392
SSW	2.32404
	2.70777
	2.58606
	2.60314
S	2.63624
	2.00194
	1.73581
	1.50264
SSE	1.25577
	1.01697
	0.92195
	0.82953
SE	0.72703
	0.86244
	1.02141
	1.07255
ESE	1.18654
	1.32842
	1.10624
	1.2783
E	1.28475
	1.23271
	1.17376
	1.52736
ENE	1.86402
	1.90276
	1.66429
	1.68193
NE	1.57969
	1.79242
	2.13602
	2.37193

Receptor Direction	Concentration (ug/m^3 / $mg/s\text{-}m^2$)
NNE	2.62736
	2.40257
	2.46014
	3.26319
N	4.70998
	5.13137
	5.06794
	4.3813
NNW	3.18078
	2.85208
	2.20599
	1.94446
NW	1.60932
	1.60011
	1.97633
	2.05037
WNW	2.00156
	1.62487
	2.03714
	2.63879
W	3.65328
	2.48129
	2.20659
	1.76247
WSW	1.63511
	1.66917
	1.48373
	1.43385

max	5.13
50th %ile	1.78

10. References

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Attachment 1

Chlorinated Aliphatics Wastewater Tank Scenario
Probabilistic Risk Results for
Direct Inhalation of Chloroform (Non-Groundwater Pathways)

Chlorinated Aliphatics Wastewater Tank Scenario
Probabilistic Risk Results for
Direct Inhalation of Chloroform (Non-Groundwater Pathways)
Adult Receptors

Percentiles	Adult Resident, Home Gardener, Fisher	Farmer
10%	1.04E-09	1.14E-09
20%	3.11E-09	3.48E-09
30%	6.87E-09	7.82E-09
40%	1.35E-08	1.55E-08
50%	2.51E-08	2.93E-08
60%	4.67E-08	5.49E-08
70%	9.00E-08	1.07E-07
80%	1.91E-07	2.29E-07
90%	5.16E-07	6.25E-07
95%	1.08E-06	1.32E-06
97.5%	1.92E-06	2.36E-06
99%	3.50E-06	4.32E-06
100%	4.96E-05	6.05E-05

Chlorinated Aliphatics Wastewater Tank Scenario
Probabilistic Risk Results for
Direct Inhalation of Chloroform (Non-Groundwater Pathways)
Child Receptors

Percentiles	Resident Child			Farm Child		
	Age 1-5	Age 6-11	Age 12-18	Age 1-5	Age 6-11	Age 12-18
10%	1.22E-09	1.37E-09	8.89E-10	2.40E-09	1.82E-09	1.21E-09
20%	3.59E-09	4.00E-09	2.63E-09	7.08E-09	5.39E-09	3.62E-09
30%	7.83E-09	8.66E-09	5.75E-09	1.55E-08	1.18E-08	8.00E-09
40%	1.51E-08	1.66E-08	1.11E-08	2.99E-08	2.28E-08	1.56E-08
50%	2.80E-08	3.04E-08	2.06E-08	5.51E-08	4.21E-08	2.90E-08
60%	5.17E-08	5.56E-08	3.80E-08	1.02E-07	7.75E-08	5.38E-08
70%	9.89E-08	1.05E-07	7.24E-08	1.93E-07	1.47E-07	1.03E-07
80%	2.09E-07	2.20E-07	1.53E-07	4.05E-07	3.09E-07	2.18E-07
90%	5.55E-07	5.78E-07	4.06E-07	1.08E-06	8.22E-07	5.84E-07
95%	1.15E-06	1.16E-06	8.32E-07	2.18E-06	1.66E-06	1.20E-06
97.5%	2.02E-06	1.99E-06	1.45E-06	3.75E-06	2.85E-06	2.09E-06
99%	3.67E-06	3.50E-06	2.59E-06	6.56E-06	4.98E-06	3.72E-06
100%	6.45E-05	4.67E-05	3.89E-05	6.40E-05	4.57E-05	4.03E-05

Attachment 2

Screening Ecological Risk Analysis for the Wastewater Treatment Tank Scenario

ADDENDUM TO APPENDIX J: ECOLOGICAL ASSESSMENT OF CHLORINATED ALIPHATIC WASTEWATERS MANAGED IN TANKS

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July 19, 1999

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ADDENDUM TO APPENDIX J: ECOLOGICAL ASSESSMENT OF CHLORINATED ALIPHATIC WASTEWATERS MANAGED IN TANKS

This addendum presents the results of the ecological screening analysis completed for chlorinated aliphatic wastewaters managed in tanks. The methods implemented to conduct the screening analysis are outlined in Sections 1 through 4 of Appendix J. This addendum does not incorporate discussions of the methods. Rather, the goals of the addendum are to present the results of (1) benchmark and chemical stressor concentration limits (CSCLs) development for the new list of constituents evaluated in wastewater tanks analysis and (2) the modeled media concentrations and hazard quotients (HQs) calculated for soil, surface water, sediment, and plant tissue.

1.0 Benchmark and CSCL Development

Since some of the constituents of concern for wastewaters managed in tanks were different than those identified for other chlorinated aliphatics waste management scenarios, another iteration of data collection and review was conducted to identify appropriate benchmarks and CSCLs for the additional constituents. For constituents that were already evaluated under other waste management scenarios, the previously applied screening values were adopted. The same sources as those identified in Table 4 of Appendix J were consulted during literature reviews.

As outlined in Appendix J, benchmarks, in units of dose (mg/kg-d), were first identified for species of mammals and birds. Benchmarks were then converted to media concentrations or CSCLs by estimating conservative exposure scenarios for ingestion pathways. For other receptors exposed via direct contact, the media concentrations indicating no- or low-effects levels were adopted directly as CSCLs. Given the differences between mammalian and avian CSCL derivation compared to other ecological receptors evaluated, discussions of these values are considered separately.

1.1 Mammal and Bird Benchmark/CSCL Derivation

Very few additional constituent benchmark doses were identified for mammal and bird receptor taxa. Food chain impacts associated with the transfer of constituents to higher trophic levels are not expected to occur for volatile and semivolatile compounds. Typically, compounds with log K_{ow}s less than 3.5 do not bioaccumulate to a significant degree, and moreover, many of these compounds volatilize before they are taken up into biota. Therefore, impacts through food web exposures are not expected to be a significant for many of the volatile and semivolatile constituents evaluated in this analysis.

The detailed progression of steps used in deriving mammalian and avian CSCLs from benchmark studies was outlined in detail in Section 4.1 of Appendix J. The steps are provided here along with the corresponding results tables.

Step 1: Identify Appropriate Benchmark Study. Tables 1 and 2 contain the mammalian and avian benchmark study doses identified for constituents.

Step 2: Scale Benchmark. Table 3 contains the scaled benchmark doses derived for mammals. Since an allometric scaling factor of 1 was applied for avian receptors, the benchmark study doses are equal to the scaled doses.

Step 3: Identify Uptake Factors. The uptake factors (e.g., bioconcentration and bioaccumulation factors, BCFs/BAFs) identified for prey items of terrestrial and freshwater ecosystems are presented in Tables 4 and 5. The prey items in the terrestrial food web consisted of worms, terrestrial plants, and small vertebrates; the prey items in the freshwater food web consisted of trophic level 3 and 4 fish and aquatic invertebrates. Data collection efforts for uptake factors focused on constituents for which ecotoxicity data for mammals or birds had already been identified. When ecotoxicity data were not available, the exposure pathways could not be completely evaluated. Therefore, there was low utility in identifying additional uptake data.

Step 4: Derive CSCL from Benchmark Doses. Protective media concentrations in surface water, sediment, soil, and plant tissue were back-calculated from benchmark doses using the uptake factors, prey preferences of receptors, and other life history parameters as outlined in Section 4.1 of Appendix J. The results of these calculations are presented for representative species of mammals and birds in Tables 6 and 7.

1.2 CSCL Derivation for Other Receptors

For other receptors, CSCLs reflecting direct contact exposures were identified for freshwater (i.e., algae and aquatic plants, amphibians, freshwater community, benthic community) and terrestrial (i.e., plants, earthworms) receptors. Limited ecotoxicity data were identified to evaluate amphibians, earthworms, and algae and aquatic plants. More ecotoxicity data were identified for the freshwater community, benthic community, and terrestrial plants. Searches of the available compendia sources resulted in the CSCLs presented in Table 8. In some instances, additional ecotoxicity data were available to evaluate specific constituents because of their priority status under other OSW efforts. These constituents included pentachlorophenol, diethyl phthalate, dimethyl phthalate, and methyl mercury.

2.0 Presentation and Discussion of Results

Because this was a screening analysis, comparisons were made between the modeled media concentrations and the lowest CSCL identified within each media compartment (i.e., surface water, sediment, soil, and plant tissues). The lowest CSCL was determined for each media compartment by comparing all the surface water CSCLs across the freshwater receptor taxa: representative mammals and birds foraging in freshwater ecosystem, the freshwater community, amphibians, and algae and aquatic plants. The lowest media concentration was then selected to calculate risk estimates. An analogous comparison was completed among terrestrial receptor taxa: representative mammals and birds foraging in the terrestrial ecosystem, plants, and the soil community. For other media, the lowest plant tissue concentrations for representative mammalian and avian herbivores were selected, and since only one receptor taxa was considered for sediment (i.e., benthic community) this, by default, was the lowest value. The lowest CSCLs identified for each media are presented in Table 9 and were subsequently used to make final risk determinations.

Modeled media concentrations were generated for two high-end sites (Baltimore, MD and Baton Rouge, LA) and one central tendency site (Memphis, TN). Media concentrations are presented in Tables 10, 11 and 12 for the three sites. The corresponding HQs were generated where CSCL data were available and are presented in Tables 13, 14, and 15. Briefly, the HQs were calculated by taking the ratio of the modeled media concentrations to the CSCLs presented in Table 9. None of the HQs generated for these comparisons exceeded 1. Therefore, risks were not indicated for any of the receptor taxa evaluated under the high-end or central tendency management scenarios.

However, given the lack of ecotoxicity data for many of the constituents, these results should not be construed as suggesting no risk to ecological systems. They do suggest minimal risks for the receptor taxa evaluated given the conservative screening assumptions applied in estimating exposures and CSCLs. In cases where data were insufficient to evaluate risk, there is uncertainty with the level of protection provided for these receptors. The lack of toxicity data introduces the greatest uncertainty in the development of this screening analysis for wastewater tanks. In addition, the key uncertainties reviewed in Section 6.0 of Appendix J also apply for this portion of the analysis.

Table 1. Studies Used to Develop Dose Benchmarks for Mammals

(ID = insufficient data available)

CAS Number	Chemical Name	NOAEL (mg/kg-day)	Test Species Body Weight (kg)	Test Species	Sex	Reference
67-64-1	Acetone	ID	ID	ID	ID	ID
107-05-1	Allyl chloride	ID	ID	ID	ID	ID
65-85-0	Benzoic acid	ID	ID	ID	ID	ID
100-51-6	Benzyl alcohol	ID	ID	ID	ID	ID
111-44-4	Bis(2-chloroethyl) ether	ID	ID	ID	ID	ID
39638-32-9	Bis(2-chloroisopropyl) ether	ID	ID	ID	ID	ID
117-81-7	Bis(2-ethylhexyl) phthalate	7.0e+01	3.0e-02	Mice	Female	Shiota and Nishimura, 1982
75-27-4	Bromodichloromethane	ID	ID	ID	ID	ID
75-25-2	Bromoform	ID	ID	ID	ID	ID
75-15-0	Carbon disulfide	ID	ID	ID	ID	ID
126-99-8	Chloro-1,3-butadiene, 2-	ID	ID	ID	ID	ID
124-48-1	Chlorodibromomethane	ID	ID	ID	ID	ID
67-66-3	Chloroform	1.5e+01	3.5e-01	Rat	Both	Sample et al., 1996
108-90-7	Chlorobenzene	ID	ID	ID	ID	ID
95-48-7	Cresol, o-	2.2e+02	1.0e+00	Mink	Both	Sample et al., 1996
106-44-5	Cresol, p-	ID	ID	ID	ID	ID
107-06-2	Dichloroethane, 1,2-	5.0e+01	3.5e-02	Mouse	Female	Sample et al., 1996
156-59-2	Dichloroethylene, cis-1,2-	ID	ID	ID	ID	ID
156-60-5	Dichloroethylene, trans-1,2-	ID	ID	ID	ID	ID
84-66-2	Diethyl phthalate	4.6e+03	3.0e-02	Mouse	ID	Sample et al., 1996
131-11-3	Dimethyl phthalate	ID	ID	ID	ID	ID
117-84-0	Di-n-octyl phthalate	ID	ID	ID	ID	ID
100-41-4	Ethylbenzene	ID	ID	ID	ID	ID
75-00-3	Ethyl chloride	ID	ID	ID	ID	ID
118-74-1	Hexachlorobenzene	1.5e+00	4.6e-01	Rat	Female	Grant, 1977
78-93-3	Methyl ethyl ketone	1.8e+03	3.5e-01	Rat	Female	Sample et al., 1996
75-09-2	Methylene chloride	ID	ID	ID	ID	ID
7439-97-6	Methyl mercury	9.9e-02	8.0e-01	Mink	Female	U.S. EPA, 1997
87-86-5	Pentachlorophenol	4.0e+00	3.5e-01	Rat	Both	Welsh et al., 1987
108-95-2	Phenol	ID	ID	ID	ID	ID

Table 1. Studies Used to Develop Dose Benchmarks for Mammals

(ID = insufficient data available)

CAS Number	Chemical Name	NOAEL (mg/kg-day)	Test Species Body Weight (kg)	Test Species	Sex	Reference
100-42-5	Styrene	ID	ID	ID	ID	ID
1746-01-6	TCDD 2,3,7,8-	1.0e-06	3.5e-01	Sprague-Dawley Rat	Both	Murray et al., 1979
127-18-4	Tetrachloroethylene	ID	ID	ID	ID	ID
79-01-6	Trichloroethylene	7.0e-01	3.0e-02	Mouse	Both	Sample et al., 1996
95-95-4	Trichlorophenol, 2,4,5-	ID	ID	ID	ID	ID
88-06-2	Trichlorophenol, 2,4,6-	ID	ID	ID	ID	ID

Table 2. Studies Used to Develop Dose Benchmarks for Birds

(ID = insufficient data available)

CAS Number	Chemical Name	NOAEL (mg/kg-day)	Test Species Body Weight (kg)	Test Species	Sex	Reference
67-64-1	Acetone	ID	ID	ID	ID	ID
107-05-1	Allyl chloride	ID	ID	ID	ID	ID
65-85-0	Benzoic Acid	ID	ID	ID	ID	ID
100-51-6	Benzyl alcohol	ID	ID	ID	ID	ID
111-44-4	Bis (2-chloroethyl) ether	ID	ID	ID	ID	ID
39638-32-9	Bis (2-chloroisopropyl) ether	ID	ID	ID	ID	ID
117-81-7	Bis(2-ethylhexyl) phthalate	1.1e+00	1.6e-01	Ringed Dove	Both	Sample et al., 1996
75-27-4	Bromodichloromethane	ID	ID	ID	ID	ID
75-25-2	Bromoform	ID	ID	ID	ID	ID
75-15-0	Carbon disulfide	ID	ID	ID	ID	ID
126-99-8	Chloro-1,3-butadiene, 2-	ID	ID	ID	ID	ID
124-48-1	Chlorodibromomethane	ID	ID	ID	ID	ID
67-66-3	Chloroform	ID	ID	ID	ID	ID
108-90-7	Chlorobenzene	ID	ID	ID	ID	ID
95-48-7	Cresol, o-	ID	ID	ID	ID	ID
106-44-5	Cresol, p-	ID	ID	ID	ID	ID
107-06-2	Dichloroethane, 1,2-	1.7e+01	1.6e+00	Chicken	Female	Sample et al., 1996
156-59-2	Dichloroethylene, cis-1,2-	ID	ID	ID	ID	ID
156-60-5	Dichloroethylene, trans-1,2-	ID	ID	ID	ID	ID
84-66-2	Diethyl phthalate	ID	ID	ID	ID	ID
131-11-3	Dimethyl phthalate	ID	ID	ID	ID	ID
117-84-0	Di-n-octyl phthalate	ID	ID	ID	ID	ID
100-41-4	Ethylbenzene	ID	ID	ID	ID	ID
75-00-3	Ethyl chloride	ID	ID	ID	ID	ID
118-74-1	Hexachlorobenzene	6.0e-01	1.3e-01	Japanese Quail	Female	Vos et al., 1971
78-93-3	Methyl ethyl ketone	ID	ID	ID	ID	ID
75-09-2	Methylene chloride	ID	ID	ID	ID	ID
7439-97-6	Methyl mercury	2.5e-02	1.2e+00	Mallard	Both	Heinz, 1974; Heinz, 1976a,b; Heinz, 1979; U.S. EPA, 1997
87-86-5	Pentachlorophenol	4.4e+01	1.3e+00	Chicks	Both	Prescott et al., 1982

Table 2. Studies Used to Develop Dose Benchmarks for Birds

(ID = insufficient data available)

CAS Number	Chemical Name	NOAEL (mg/kg-day)	Test Species Body Weight (kg)	Test Species	Sex	Reference
108-95-2	Phenol	ID	ID	ID	ID	ID
100-42-5	Styrene	ID	ID	ID	ID	ID
1746-01-6	TCDD 2,3,7,8-	1.4e-05	9.0e-01	Pheasant	Female	Nosek et al., 1992
127-18-4	Tetrachloroethylene	ID	ID	ID	ID	ID
79-01-6	Trichloroethylene	ID	ID	ID	ID	ID
95-95-4	Trichlorophenol, 2,4,5-	ID	ID	ID	ID	ID
88-06-2	Trichlorophenol, 2,4,6-	ID	ID	ID	ID	ID

Table 3. Scaled Benchmarks for Representative Mammals in Freshwater and Terrestrial Ecosystems (mg/kg-d)

CAS Number	Chemical Name	Mink	River Otter	Short-tailed Shrew	Deer Mouse	Meadow Vole	Eastern Cottontail	Red Fox	Raccoon	White-tailed Deer
67-64-1	Acetone	ID	ID	ID	ID	ID	ID	ID	ID	ID
107-05-1	Allyl chloride	ID	ID	ID	ID	ID	ID	ID	ID	ID
65-85-0	Benzoic acid	ID	ID	ID	ID	ID	ID	ID	ID	ID
100-51-6	Benzyl alcohol	ID	ID	ID	ID	ID	ID	ID	ID	ID
111-44-4	Bis (2-chloroethyl) ether	ID	ID	ID	ID	ID	ID	ID	ID	ID
39638-32-9	Bis (2-chloroisopropyl) ether	ID	ID	ID	ID	ID	ID	ID	ID	ID
117-81-7	Bis(2-ethylhexyl) phthalate	3.2e+01	1.8e+01	8.1e+01	7.9e+01	6.6e+01	2.8e+01	2.1e+01	2.0e+01	9.9e+00
75-27-4	Bromodichloromethane	ID	ID	ID	ID	ID	ID	ID	ID	ID
75-25-2	Bromoform	ID	ID	ID	ID	ID	ID	ID	ID	ID
75-15-0	Carbon disulfide	ID	ID	ID	ID	ID	ID	ID	ID	ID
126-99-8	Chloro-1,3-butadiene, 2-	ID	ID	ID	ID	ID	ID	ID	ID	ID
124-48-1	Chlorodibromomethane	ID	ID	ID	ID	ID	ID	ID	ID	ID
67-66-3	Chloroform	1.1e+01	6.9e+00	3.2e+01	3.1e+01	2.7e+01	1.1e+01	7.9e+00	7.5e+00	3.8e+00
108-90-7	Chlorobenzene	ID	ID	ID	ID	ID	ID	ID	ID	ID
95-48-7	Cresol, o-	2.2e+02	1.3e+02	6.1e+02	5.9e+02	5.1e+02	2.1e+02	1.5e+02	1.4e+02	7.2e+01
106-44-5	Cresol, p-	ID	ID	ID	ID	ID	ID	ID	ID	ID
107-06-2	Dichloroethane, 1,2-	2.4e+01	1.3e+01	6.0e+01	5.9e+01	4.9e+01	2.1e+01	1.5e+01	1.5e+01	7.3e+00
156-59-2	Dichloroethylene, cis-1,2-	ID	ID	ID	ID	ID	ID	ID	ID	ID
156-60-5	Dichloroethylene, trans-1,2-	ID	ID	ID	ID	ID	ID	ID	ID	ID
84-66-2	Diethyl phthalate	1.9e+03	1.1e+03	5.3e+03	5.1e+03	4.5e+03	1.8e+03	1.3e+03	1.2e+03	6.3e+02
131-11-3	Dimethyl phthalate	ID	ID	ID	ID	ID	ID	ID	ID	ID

Table 3. Scaled Benchmarks for Representative Mammals in Freshwater and Terrestrial Ecosystems (mg/kg-d)

CAS Number	Chemical Name	Mink	River Otter	Short-tailed Shrew	Deer Mouse	Meadow Vole	Eastern Cottontail	Red Fox	Raccoon	White-tailed Deer
117-84-0	Di-n-octyl phthalate	ID	ID	ID	ID	ID	ID	ID	ID	ID
100-41-4	Ethylbenzene	ID	ID	ID	ID	ID	ID	ID	ID	ID
75-00-3	Ethyl chloride	ID	ID	ID	ID	ID	ID	ID	ID	ID
118-74-1	Hexachlorobenzene	1.3e+00	7.3e-01	3.3e+00	3.3e+00	2.7e+00	1.1e+00	8.5e-01	8.2e-01	4.1e-01
78-93-3	Methyl ethyl ketone	1.5e+03	8.3e+02	3.8e+03	3.7e+03	3.1e+03	1.3e+03	9.6e+02	9.2e+02	4.6e+02
75-09-2	Methylene chloride	ID	ID	ID	ID	ID	ID	ID	ID	ID
7439-97-6	Methyl mercury	1.0e-01	5.7e-02	2.6e-01	2.5e-01	2.1e-01	8.9e-02	6.6e-02	6.4e-02	3.2e-02
87-86-5	Pentachlorophenol	3.1e+00	1.8e+00	8.5e+00	8.3e+00	7.2e+00	2.9e+00	2.1e+00	2.0e+00	1.0e+00
108-95-2	Phenol	ID	ID	ID	ID	ID	ID	ID	ID	ID
100-42-5	Styrene	ID	ID	ID	ID	ID	ID	ID	ID	ID
1746-01-6	TCDD 2,3,7,8-	7.7e-07	4.6e-07	2.1e-06	2.1e-06	1.8e-06	7.3e-07	5.3e-07	5.0e-07	2.5e-07
127-18-4	Tetrachloroethylene	ID	ID	ID	ID	ID	ID	ID	ID	ID
79-01-6	Trichloroethylene	2.9e-01	1.7e-01	8.1e-01	7.8e-01	6.8e-01	2.8e-01	2.0e-01	1.9e-01	0.095945
95-95-4	Trichlorophenol, 2,4,5-	ID	ID	ID	ID	ID	ID	ID	ID	ID
88-06-2	Trichlorophenol, 2,4,6-	ID	ID	ID	ID	ID	ID	ID	ID	ID

Note: Avian benchmarks were scaled using a factor of 1, therefore, they are equivalent to the NOAEL for all representative receptors.

Table 4. Uptake Factors for Prey Items of Representative Receptors in Generalized Freshwater Ecosystems

CAS Number	Chemical Name	Trophic Level 3 Fish	Source	Trophic Level 4 Fish	Source	Aquatic Invertebrates	Source
67-64-1	Acetone	3.9e-01	Sample et al., 1996	3.9e-01	Sample et al., 1996	**1.0e+00**	Default
107-05-1	Allyl chloride	7.4e+00	Sample et al., 1996	7.4e+00	Sample et al., 1996	**1.0e+00**	Default
65-85-0	Benzoic acid	1.5e+01	Sample et al., 1996	1.5e+01	Sample et al., 1996	**1.0e+00**	Default
100-51-6	Benzyl alcohol	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
111-44-4	Bis(2-chloroethyl) ether	4.8e+00	Sample et al., 1996	4.8e+00	Sample et al., 1996	**1.0e+00**	Default
39638-32-9	Bis(2-chloroisopropyl) ether	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
117-81-7	Bis(2-ethylhexyl) phthalate	3.6e+00	Sample et al., 1996	1.5e+01	Sample et al., 1996	3.3e-01	Sample et al., 1996
75-27-4	Bromodichloromethane	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
75-25-2	Bromoform	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
75-15-0	Carbon disulfide	2.0e+01	Sample et al., 1996	2.0e+01	Sample et al., 1996	**1.0e+00**	Default
126-99-8	Chloro-1,3-butadiene, 2-	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
124-48-1	Chlorodibromomethane	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
67-66-3	Chloroform	1.1e+02	Sample et al., 1996	1.1e+02	Sample et al., 1996	**1.0e+00**	Default
108-90-7	Chlorobenzene	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
95-48-7	Cresol, o-	1.9e+01	Sample et al., 1996	1.9e+01	Sample et al., 1996	**1.0e+00**	Default
106-44-5	Cresol, p-	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
107-06-2	Dichloroethane, 1,2-	7.7e+00	Sample et al., 1996	7.7e+00	Sample et al., 1996	**1.0e+00**	Default
156-59-2	Dichloroethylene, cis-1,2-	1.5e+01	Sample et al., 1996	1.5e+01	Sample et al., 1996	**1.0e+00**	Default
156-60-5	Dichloroethylene, trans-1,2-	1.5e+01	Sample et al., 1996	1.5e+01	Sample et al., 1996	**1.0e+00**	Default
84-66-2	Diethyl phthalate	4.7e+01	Sample et al., 1996	4.7e+01	Sample et al., 1996	**1.0e+00**	Default
131-11-3	Dimethyl phthalate	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default

Table 4. Uptake Factors for Prey Items of Representative Receptors in Generalized Freshwater Ecosystems

CAS Number	Chemical Name	Trophic Level 3 Fish	Source	Trophic Level 4 Fish	Source	Aquatic Invertebrates	Source
117-84-0	Di-n-octyl phthalate	5.7e+06	Sample et al., 1996	4.7e+06	Sample et al., 1996	**1.0e+00**	Default
100-41-4	Ethylbenzene	1.5e+02	Sample et al., 1996	1.4e+02	Sample et al., 1996	**1.0e+00**	Default
75-00-3	Ethyl chloride	7.2e+00	Sample et al., 1996	7.2e+00	Sample et al., 1996	**1.0e+00**	Default
118-74-1	Hexachlorobenzene	1.7e+05	Sample et al., 1996	2.5e+05	Sample et al., 1996	**1.0e+00**	Default
78-93-3	Methyl ethyl ketone	9.6e-01	Sample et al., 1996	9.6e-01	Sample et al., 1996	**1.0e+00**	Default
75-09-2	Methylene chloride	5.3e+00	Sample et al., 1996	5.3e+00	Sample et al., 1996	**1.0e+00**	Default
7439-97-6	Methyl mercury	1.6e+06	U.S. EPA, 1997	6.8e+06	U.S. EPA, 1997	**1.0e+00**	Default
87-86-5	Pentachlorophenol	3.6e+03	Sample et al., 1996	3.2e+03	Sample et al., 1996	**1.0e+00**	Default
108-95-2	Phenol	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
100-42-5	Styrene	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
1746-01-6	TCDD 2,3,7,8-	1.7e+05	U.S. EPA, 1995	2.6e+05	U.S. EPA, 1995	**1.0e+00**	Default
127-18-4	Tetrachloroethylene	3.4e+00	Sample et al., 1996	3.4e+00	Sample et al., 1996	**1.0e+00**	Default
79-01-6	Trichloroethylene	6.8e+01	Sample et al., 1996	6.8e+01	Sample et al., 1996	7.1e+01	Sample et al., 1996
95-95-4	Trichlorophenol, 2,4,5-	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
88-06-2	Trichlorophenol, 2,4,6-	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default

Note: Bold numbers are default values. Values for trophic level 3 and trophic level 4 fish cited as Sample et al. (1996) were calculated using the following relationship from Lyman et al. (1982): log BCF = 0.76*log(Kow)-0.23, and the results were multiplied by food chain multipliers (U.S. EPA, 1993).

Table 5. Bioaccumulation Factors for Prey Items of Representative Receptors in Generalized Terrestrial Ecosystems

CAS Number	Chemical Name	Invertebrates (including earthworms)	Source	Plants	Source	Vertebrates	Source
67-64-1	Acetone	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
107-05-1	Allyl chloride	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
65-85-0	Benzoic Acid	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
100-51-6	Benzyl alcohol	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
111-44-4	Bis(2-chloroethyl) ether	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
39638-32-9	Bis (2-chloroisopropyl) ether	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
117-81-7	Bis(2-ethylhexyl) phthalate	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
75-27-4	Bromodichloromethane	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
75-25-2	Bromoform	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
75-15-0	Carbon disulfide	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
126-99-8	Chloro-1,3-butadiene, 2-	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
124-48-1	Chlorodibromomethane	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
67-66-3	Chloroform	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
108-90-7	Chlorobenzene	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
95-48-7	Cresol, o-	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
106-44-5	Cresol, p-	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
107-06-2	Dichloroethane, 1,2-	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
156-59-2	Dichloroethylene, cis-1,2-	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
156-60-5	Dichloroethylene, trans-1,2-	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
84-66-2	Diethyl phthalate	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
131-11-3	Dimethyl phthalate	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default

Table 5. Bioaccumulation Factors for Prey Items of Representative Receptors in Generalized Terrestrial Ecosystems

CAS Number	Chemical Name	Invertebrates (including earthworms)	Source	Plants	Source	Vertebrates	Source
117-84-0	Di-n-octyl phthalate	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
100-41-4	Ethylbenzene	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
75-00-3	Ethyl chloride	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
118-74-1	Hexachlorobenzene	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
78-93-3	Methyl ethyl ketone	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
75-09-2	Methylene chloride	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
7439-97-6	Methyl mercury	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
87-86-5	Pentachlorophenol	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
108-95-2	Phenol	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
100-42-5	Styrene	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
1746-01-6	TCDD 2,3,7,8-	3.4e+00	Sample et al., 1996	**1.0e+00**	Default	7.2e+00	Sample et al.,, 1996
127-18-4	Tetrachloroethylene	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
79-01-6	Trichloroethylene	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
95-95-4	Trichlorophenol, 2,4,5-	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default
88-06-2	Trichlorophenol, 2,4,6-	**1.0e+00**	Default	**1.0e+00**	Default	**1.0e+00**	Default

Note: Bold numbers are default values and the shaded cells are measured values.

Table 6. Calculated CSCLs for Representative Mammals in Freshwater (mg/L) and Terrestrial Ecosystems (mg/kg soil)

(ID = insufficient data available)

		Freshwater		Terrestrial						
CAS Number	**Chemical Name**	**Mink**	**River Otter**	**Short-tailed Shrew**	**Deer Mouse**	**Meadow Vole**	**Eastern Cottontail**	**Red Fox**	**Raccoon**	**White-tailed Deer**
67-64-1	Acetone	ID	ID	ID	ID	ID	ID	ID	ID	ID
107-05-1	Allyl chloride	ID	ID	ID	ID	ID	ID	ID	ID	ID
65-85-0	Benzoic Acid	ID	ID	ID	ID	ID	ID	ID	ID	ID
100-51-6	Benzyl alcohol	ID	ID	ID	ID	ID	ID	ID	ID	ID
111-44-4	Bis (2-chloroethyl) ether	ID	ID	ID	ID	ID	ID	ID	ID	ID
39638-32-9	Bis (2-chloroisopropyl) ether	ID	ID	ID	ID	ID	ID	ID	ID	ID
117-81-7	Bis(2-ethylhexyl) phthalate	5.0e+01	1.1e+01	1.8e+02	4.7e+02	2.0e+02	3.4e+02	2.2e+02	4.4e+02	3.8e+02
75-27-4	Bromodichloromethane	ID	ID	ID	ID	ID	ID	ID	ID	ID
75-25-2	Bromoform	ID	ID	ID	ID	ID	ID	ID	ID	ID
75-15-0	Carbon disulfide	ID	ID	ID	ID	ID	ID	ID	ID	ID
126-99-8	Chloro-1,3-butadiene, 2-	ID	ID	ID	ID	ID	ID	ID	ID	ID
124-48-1	Chlorodibromomethane	ID	ID	ID	ID	ID	ID	ID	ID	ID
67-66-3	Chloroform	6.6e-01	3.9e-01	7.1e+01	1.8e+02	8.1e+01	1.3e+02	8.3e+01	1.7e+02	1.5e+02
108-90-7	Chlorobenzene	ID	ID	ID	ID	ID	ID	ID	ID	ID
95-48-7	Cresol, o-	7.0e+01	4.2e+01	1.4e+03	3.5e+03	1.5e+03	2.5e+03	1.6e+03	3.1e+03	2.8e+03
106-44-5	Cresol, p-	ID	ID	ID	ID	ID	ID	ID	ID	ID
107-06-2	Dichloroethane, 1,2-	1.8e+01	1.0e+01	1.3e+02	3.5e+02	1.5e+02	2.5e+02	1.6e+02	3.2e+02	2.8e+02
156-59-6	Dichloroethylene, cis-1,2-	ID	ID	ID	ID	ID	ID	ID	ID	ID
156-60-5	Dichloroethylene, trans-1,2-	ID	ID	ID	ID	ID	ID	ID	ID	ID
84-66-2	Diethyl phthalate	2.5e+02	1.5e+02	1.2e+04	3.0e+04	1.3e+04	2.2e+04	1.4e+04	2.7e+04	2.4e+04

Table 6. Calculated CSCLs for Representative Mammals in Freshwater (mg/L) and Terrestrial Ecosystems (mg/kg soil)

(ID = insufficient data available)

		Freshwater		Terrestrial						
CAS Number	**Chemical Name**	**Mink**	**River Otter**	**Short-tailed Shrew**	**Deer Mouse**	**Meadow Vole**	**Eastern Cottontail**	**Red Fox**	**Raccoon**	**White-tailed Deer**
131-11-3	Dimethyl phthalate	ID	ID	ID	ID	ID	ID	ID	ID	ID
117-84-0	Di-n-octyl phthalate	ID	ID	ID	ID	ID	ID	ID	ID	ID
100-41-4	Ethylbenzene	ID	ID	ID	ID	ID	ID	ID	ID	ID
75-00-3	Ethyl chloride	ID	ID	ID	ID	ID	ID	ID	ID	ID
118-74-1	Hexachlorobenzene	4.9e-05	2.2e-05	7.4e+00	1.9e+01	8.1e+00	1.4e+01	8.9e+00	1.8e+01	1.6e+01
78-93-3	Methyl ethyl ketone	6.5e+03	3.6e+03	8.4e+03	2.2e+04	9.2e+03	1.6e+04	1.0e+04	2.0e+04	1.8e+04
75-09-2	Methylene chloride	ID	ID	ID	ID	ID	ID	ID	ID	ID
7439-97-6	Methyl mercury	4.1e-07	8.6e-08	5.8e-01	1.5e+00	6.3e-01	1.1e+00	7.0e-01	1.4e+00	1.2e+00
87-86-5	Pentachlorophenol	5.4e-03	3.4e-03	1.9e+01	4.9e+01	2.2e+01	3.6e+01	2.2e+01	4.4e+01	3.9e+01
108-95-2	Phenol	ID	ID	ID	ID	ID	ID	ID	ID	ID
100-42-5	Styrene	ID	ID	ID	ID	ID	ID	ID	ID	ID
1746-01-6	TCDD 2,3,7,8-	2.8e-11	1.3e-11	1.6e-06	7.2e-06	7.9e-05	8.9e-06	1.7e-06	4.0e-06	9.7e-06
127-18-4	Tetrachloroethylene	ID	ID	ID	ID	ID	ID	ID	ID	ID
79-01-6	Trichloroethylene	2.7e-02	1.6e-02	1.8e+00	4.6e+00	2.1e+00	3.4e+00	2.1e+00	4.2e+00	3.7e+00
95-95-4	Trichlorophenol, 2,4,5-	ID	ID	ID	ID	ID	ID	ID	ID	ID
88-06-2	Trichlorophenol, 2,4,6-	ID	ID	ID	ID	ID	ID	ID	ID	ID

Table 7. Calculated CSCLs for Representative Birds in Freshwater and Terrestrial Ecosystems (mg/L water and mg/kg soil)

(ID = insufficient data available)

CAS Number	Chemical Name	Bald Eagle	Osprey	Great Blue Heron	Mallard	Lesser Scaup	Kingfisher	Spotted Sandpiper	Herring Gull
67-64-1	Acetone	ID	ID	ID	ID	ID	ID	ID	ID
107-05-1	Allyl chloride	ID	ID	ID	ID	ID	ID	ID	ID
65-85-0	Benzoic acid	ID	ID	ID	ID	ID	ID	ID	ID
100-51-6	Benzyl alcohol	ID	ID	ID	ID	ID	ID	ID	ID
111-44-4	Bis (2-chloroethyl) ether	ID	ID	ID	ID	ID	ID	ID	ID
39638-32-9	Bis (2-chloroisopropyl) ether	ID	ID	ID	ID	ID	ID	ID	ID
117-81-7	Bis(2-ethylhexyl) phthalate	5.8e-01	1.4e+00	3.9e-01	7.3e+00	6.4e+00	6.0e-01	2.3e+00	1.5e+00
75-27-4	Bromodichloromethane	ID	ID	ID	ID	ID	ID	ID	ID
75-25-2	Bromoform	ID	ID	ID	ID	ID	ID	ID	ID
75-15-0	Carbon disulfide	ID	ID	ID	ID	ID	ID	ID	ID
126-99-8	Chloro-1,3-butadiene, 2-	ID	ID	ID	ID	ID	ID	ID	ID
124-48-1	Chlorodibromomethane	ID	ID	ID	ID	ID	ID	ID	ID
67-66-3	Chloroform	ID	ID	ID	ID	ID	ID	ID	ID
108-90-7	Chlorobenzene	ID	ID	ID	ID	ID	ID	ID	ID
95-48-7	Cresol, o-	ID	ID	ID	ID	ID	ID	ID	ID
106-44-5	Cresol, p-	ID	ID	ID	ID	ID	ID	ID	ID
107-06-2	Dichloroethane, 1,2-	1.8e+01	1.0e+01	1.2e+01	5.1e+01	4.4e+01	4.5e+00	1.8e+01	1.1e+01
156-59-2	Dichloroethylene, cis-1,2-	ID	ID	ID	ID	ID	ID	ID	ID
156-60-5	Dichloroethylene, trans-1,2-	ID	ID	ID	ID	ID	ID	ID	ID
84-66-2	Diethyl phthalate	ID	ID	ID	ID	ID	ID	ID	ID
131-11-3	Dimethyl phthalate	ID	ID	ID	ID	ID	ID	ID	ID

Table 7. Calculated CSCLs for Representative Birds in Freshwater and Terrestrial Ecosystems (mg/L water and mg/kg soil)

(ID = insufficient data available)

CAS Number	Chemical Name	Bald Eagle	Osprey	Great Blue Heron	Mallard	Lesser Scaup	Kingfisher	Spotted Sandpiper	Herring Gull
117-84-0	Di-n-octyl phthalate	ID	ID	ID	ID	ID	ID	ID	ID
100-41-4	Ethylbenzene	ID	ID	ID	ID	ID	ID	ID	ID
75-00-3	Ethyl chloride	ID	ID	ID	ID	ID	ID	ID	ID
118-74-1	Hexachlorobenzene	2.0e-05	1.7e-05	1.4e-05	1.8e+00	1.6e+00	7.3e-06	6.4e-01	1.8e-05
78-93-3	Methyl ethyl ketone	ID	ID	ID	ID	ID	ID	ID	ID
75-09-2	Methylene chloride	ID	ID	ID	ID	ID	ID	ID	ID
7439-97-6	Methyl mercury	3.1e-08	7.5e-08	2.0e-08	7.4e-02	6.5e-02	3.3e-08	2.7e-02	8.1e-08
87-86-5	Pentachlorophenol	1.1e-01	5.9e-02	7.6e-02	1.3e+02	1.1e+02	2.6e-02	4.7e+01	6.4e-02
108-95-2	Phenol	ID	ID	ID	ID	ID	ID	ID	ID
100-42-5	Styrene	ID	ID	ID	ID	ID	ID	ID	ID
1746-01-6	TCDD 2,3,7,8-	4.4e-10	3.9e-10	3.0e-10	2.3e-04	2.1e-04	1.7e-10	6.0e-05	4.2e-10
127-18-4	Tetrachloroethylene	ID	ID	ID	ID	ID	ID	ID	ID
79-01-6	Trichloroethylene	ID	ID	ID	ID	ID	ID	ID	ID
95-95-4	Trichlorophenol, 2,4,5-	ID	ID	ID	ID	ID	ID	ID	ID
88-06-2	Trichlorophenol, 2,4,6-	ID	ID	ID	ID	ID	ID	ID	ID

Table 7 (Continued). Calculated CSCLs for Representative Birds in Freshwater and Terrestrial Ecosystems (mg/L water and mg/kg soil)

(ID = insufficient data available)

CAS Number	Chemical Name	Red-tailed Hawk	American Kestrel	Northern Bobwhite	American Robin	American Woodcock
67-64-1	Acetone	ID	ID	ID	ID	ID
107-05-1	Allyl chloride	ID	ID	ID	ID	ID
100-51-6	Benzyl alcohol	ID	ID	ID	ID	ID
65-85-0	Benzoic acid	ID	ID	ID	ID	ID
111-44-4	Bis (2-chloroethyl) ether	ID	ID	ID	ID	ID
39638-32-9	Bis (2-chloroisopropyl)	ID	ID	ID	ID	ID
117-81-7	Bis(2-ethylhexyl) phthalate	1.1e+01	3.6e+00	1.4e+01	8.9e-01	1.4e+00
75-27-4	Bromodichloromethane	ID	ID	ID	ID	ID
75-25-2	Bromoform	ID	ID	ID	ID	ID
75-15-0	Carbon disulfide	ID	ID	ID	ID	ID
126-99-8	Chloro-1,3-butadiene, 2-	ID	ID	ID	ID	ID
124-48-1	Chlorodibromomethane	ID	ID	ID	ID	ID
67-66-3	Chloroform	ID	ID	ID	ID	ID
108-90-7	Chlorobenzene	ID	ID	ID	ID	ID
95-48-7	Cresol, o-	ID	ID	ID	ID	ID
106-44-5	Cresol, p-	ID	ID	ID	ID	ID
107-06-2	Dichloroethane, 1,2-	1.8e+02	5.7e+01	2.1e+02	1.4e+01	2.3e+01
156-59-2	Dichloroethylene, cis-1,2-	ID	ID	ID	ID	ID
156-60-5	Dichloroethylene, trans-1,2-	ID	ID	ID	ID	ID
84-66-2	Diethyl phthalate	ID	ID	ID	ID	ID
131-11-3	Dimethyl phthalate	ID	ID	ID	ID	ID
117-84-0	Di-n-octyl phthalate	ID	ID	ID	ID	ID
100-41-4	Ethylbenzene	ID	ID	ID	ID	ID
75-00-3	Ethyl chloride	ID	ID	ID	ID	ID
118-74-1	Hexachlorobenzene	6.2e+00	2.0e+00	7.4e+00	4.9e-01	7.9e-01
78-93-3	Methyl ethyl ketone	ID	ID	ID	ID	ID
75-09-2	Methylene chloride	ID	ID	ID	ID	ID
7439-97-6	Methyl mercury	2.6e-01	8.3e-02	3.1e-01	2.0e-02	3.3e-02
87-86-5	Pentachlorophenol	4.5e+02	1.5e+02	5.5e+02	3.6e+01	5.8e+01
108-95-2	Phenol	ID	ID	ID	ID	ID
100-42-5	Styrene	ID	ID	ID	ID	ID
1746-01-6	TCDD 2,3,7,8- (TEQ)	2.0e-05	8.7e-06	3.9e-04	3.7e-06	6.0e-06
127-18-4	Tetrachloroethylene	ID	ID	ID	ID	ID
79-01-6	Trichloroethylene	ID	ID	ID	ID	ID
95-95-4	Trichlorophenol, 2,4,5-	ID	ID	ID	ID	ID
88-06-2	Trichlorophenol, 2,4,6-	ID	ID	ID	ID	ID

Table 8. Community CSCLs for Freshwater and Terrestrial Receptors

(ID = insufficient data available)

		Sediment (mg/kg)	Surface Water (mg/L)				Soil (mg/kg soil)	
CAS Number	**Chemical Name**	**Benthic Community**	**Freshwater Community (total)**	**Freshwater Community (dissolved)**	**Algae**	**Amphibians**	**Plants**	**Soil Community**
67-64-1	Acetone	8.7e-03	1.5e+00	ID	ID	ID	ID	ID
107-05-1	Allyl Chloride	ID	ID	ID	ID	ID	ID	ID
65-85-0	Benzoic acid	ID	4.2e-02	ID	ID	ID	ID	ID
100-51-6	Benzyl alcohol	1.1e-03	8.6e-03	ID	ID	ID	ID	ID
111-44-4	Bis(2-chloroethyl) ether	ID	1.0e+03	ID	ID	ID	ID	ID
39638-32-9	Bis(2-chloroisopropyl) ether	ID	ID	ID	ID	ID	ID	ID
117-81-7	Bis(2-ethylhexyl) phthalate	1.8e-01	3.0e-03	ID	ID	ID	ID	ID
75-27-4	Bromodichloromethane	ID	ID	ID	ID	ID	ID	ID
75-25-2	Bromoform	ID	ID	ID	ID	ID	ID	ID
75-15-0	Carbon disulfide	8.5e-04	9.2e-04	ID	ID	ID	ID	ID
126-99-8	Chloro-1,3-butadiene, 2-	ID	ID	ID	ID	ID	ID	ID
124-48-1	Chlorodibromomethane	ID	ID	ID	ID	ID	ID	ID
67-66-3	Chloroform	2.2e-02	2.8e-02	ID	ID	1.5e+01	ID	ID
108-90-7	Chlorobenzene	4.1e-01	6.4e-02	ID	2.4e+02	ID	ID	4.0e+01
95-48-7	Cresol, o-	ID	ID	ID	ID	ID	ID	ID
106-44-5	Cresol, p-	ID	ID	ID	ID	ID	ID	ID
107-06-2	Dichloroethane, 1,2-	2.5e-01	9.1e-01	ID	ID	3.3e+00	ID	ID
156-59-2	Dichloroethylene, cis-1,2-	ID	5.9e-01	ID	ID	ID	ID	ID
156-60-5	Dichloroethylene, trans-1,2-	ID	5.9e-01	ID	ID	ID	ID	ID
84-66-2	Diethyl phthalate	ID	2.1e-01	ID	8.6e+01	ID	1.0e+02	ID
131-11-3	Dimethyl phthalate	6.0e-01	1.4e-01	ID	ID	ID	ID	2.0e+02
117-84-0	Di-n-octyl phthalate	ID	ID	ID	ID	ID	ID	ID
100-41-4	Ethylbenzene	8.9e-02	7.3e-03	ID	ID	ID	ID	1.0e-01
75-00-3	Ethyl chloride	ID	ID	ID	ID	ID	ID	ID
118-74-1	Hexachlorobenzene	7.7e+01	3.7e-03	ID	ID	ID	ID	ID

Table 8. Community CSCLs for Freshwater and Terrestrial Receptors

(ID = insufficient data available)

		Sediment (mg/kg)	Surface Water (mg/L)				Soil (mg/kg soil)	
CAS Number	**Chemical Name**	**Benthic Community**	**Freshwater Community (total)**	**Freshwater Community (dissolved)**	**Algae**	**Amphibians**	**Plants**	**Soil Community**
78-93-3	Methyl ethyl ketone	ID	ID	ID	ID	ID	ID	ID
75-09-2	Methylene chloride	3.7e-01	2.2e+00	ID	ID	ID	ID	ID
7439-97-6	Methyl mercury	ID	2.8e-06	ID	ID	ID	ID	ID
87-86-5	Pentachlorophenol	ID	5.5e-03	ID	ID	1.3e-02	3.0e+00	6.0e+00
108-95-2	Phenol	3.1e-02	1.1e-01	ID	2.0e+01	1.3e+00	7.0e+01	3.0e+01
100-42-5	Styrene	ID	ID	ID	ID	ID	3.0e+02	ID
1746-01-6	TCDD 2,3,7,8-	2.0e-04	1.2e-09	ID	ID	ID	ID	ID
127-18-4	Tetrachloroethylene	4.1e-01	9.8e-02	ID	8.2e+02	ID	1.7e+02	1.8e+01
79-01-6	Trichloroethylene	2.2e-01	4.7e-02	ID	ID	8.9e+01	1.4e+01	7.9e+01
95-95-4	Trichlorophenol, 2,4,5-	ID	ID	ID	ID	ID	4.0e+00	9.0e+00
88-06-2	Trichlorophenol, 2,4,6-	ID	ID	ID	ID	ID	ID	1.0e+01

Table 9. Lowest Chemical Stressor Concentration Limit (CSCL) Derived for Soil, Sediment, Surface Water, and Plant Tissue

(ID = insufficient data available)

		Media Types								Plant Types	
		Sediment (mg/kg)		Surface water (mg/L)				Soil (mg/kg)		Plant (mg/kg DW)	
CAS Number	Chemical Names		Receptor	Total	Receptor	Dissolved	Receptor		Receptor		Receptor
67-64-1	Acetone	8.7e-03	BC	1.5e+00	FWT	ID		ID		ID	
107-05-1	Allyl chloride	ID		ID		ID		ID		ID	
65-85-0	Benzoic acid	ID		4.2e-02	FWT	ID		ID		ID	
100-51-6	Benzyl alcohol	1.1e-03	BC	8.6e-03	FWT	ID		ID		ID	
111-44-4	Bis (2-chloroethyl) ether	ID		1.0e+03	FWT	ID		ID		ID	
39638-32-9	Bis (2-chloroisopropyl) ether	ID		ID		ID		ID		ID	
117-81-7	Bis(2-ethylhexyl) phthalate	1.8e-01	BC	3.0e-03	FWT	ID		8.9e-01	American Robin	1.6e+01	Northern Bobwhite
75-27-4	Bromodichloromethane	ID		ID		ID		ID		ID	
75-25-2	Bromoform	ID		ID		ID		ID		ID	
75-15-0	Carbon disulfide	8.5e-04	BC	9.2e-04	FWT	ID		ID		ID	
126-99-8	Chloro-1,3-butadiene, 2-	ID		ID		ID		ID		ID	
124-48-1	Chlorodibromomethane	ID		ID		ID		ID		ID	
67-66-3	Chloroform	2.2e-02	BC	2.8e-02	FWT	ID		7.1e+01	Short-tailed Shrew	8.3e+01	Meadow Vole
108-90-7	Chlorobenzene	4.1e-01	BC	6.4e-02	FWT	ID		4.0e+01	Soil Community	ID	
95-48-7	Cresol, o-	ID		4.2e+01	River Otter	ID		1.4e+03	Short-tailed Shrew	1.5e+03	Meadow Vole
106-44-5	Cresol, p-	ID		ID		ID		ID		ID	
107-06-2	Dichloroethane, 1,2-	2.5e-01	BC	9.1e-01	FWT	ID		1.4e+01	American Robin	1.5e+02	Meadow Vole
156-59-2	Dichloroethylene, cis-1,2-	ID		5.9e-01	FWT	ID		ID		ID	
156-60-5	Dichloroethylene, trans-1,2-	ID		5.9e-01	FWT	ID		ID		ID	

		Media Types								Plant Types	
		Sediment (mg/kg)		Surface water (mg/L)				Soil (mg/kg)		Plant (mg/kg DW)	
CAS Number	Chemical Names		Receptor	Total	Receptor	Dissolved	Receptor		Receptor		Receptor
84-66-2	Diethyl phthalate	ID		2.1e-01	FWT	ID		1.2e+04	Short-tailed Shrew	ID	
131-11-3	Dimethyl phthalate	6.0e-01	BC	1.4e-01	FWT	ID		2.0e+02	Soil Community	ID	
117-84-0	Di-n-octyl phthalate	ID		ID		ID		ID		ID	
100-41-4	Ethylbenzene	8.9e-02	BC	7.3e-03	FWT	ID		1.0e-01	Soil Community	ID	
75-00-3	Ethyl chloride	ID		ID		ID		ID		ID	
118-74-1	Hexachlorobenzene	7.7e+01	BC	7.3e-06	Kingfisher	ID		4.9e-01	American Robin	8.3e+00	Meadow Vole
78-93-3	Methyl ethyl ketone	ID		3.6e+03	River Otter	ID		8.4e+03	Short-tailed Shrew	9.4e+03	Meadow Vole
75-09-2	Methylene chloride	3.7e-01	BC	2.2e+00	FWT	ID		ID		ID	
7439-97-6	Methyl mercury	ID		2.0e-08	Great Blue Heron	ID		2.0e-02	American Robin	3.1e-01	Northern Bobwhite
87-86-5	Pentachlorophenol	ID		3.4e-03	River Otter	ID		3.0e+00	Terrestrial Plants	2.2e+01	Meadow Vole
108-95-2	Phenol	3.1e-02	BC	1.1e-01	FWT	ID		3.0e+01	Soil Community	ID	
100-42-5	Styrene	ID		ID		ID		3.0e+02	Terrestrial Plants	ID	
1746-01-6	TCDD 2,3,7,8-	2.0e-04	BC	1.3e-11	River Otter	ID		1.6e-06	Short-tailed Shrew	5.5e-06	Meadow Vole
127-18-4	Tetrachloroethylene	4.1e-01	BC	9.8e-02	FWT	ID		1.8e+01	Soil Community	ID	
79-01-6	Trichloroethylene	2.2e-01	BC	1.6e-02	River Otter	ID		1.8e+00	Short-tailed Shrew	2.1e+00	Meadow Vole
95-95-4	Trichlorophenol, 2,4,5-	ID		ID		ID		9.0e+00	Soil Community	ID	
88-06-2	Trichlorophenol, 2,4,6-	ID		ID		ID		1.0e+01	Soil Community	ID	

Note: Benthic Community (BC), Freshwater Community - Total (FWT), Freshwater Community - Dissolved (FWD)

[1] Although there were other soil CSCLs less than the selected value, this value was the lowest value that exceeded mean soil background concentrations for the Eastern United States

Table 10. Modeled Media Concentrations for Constituents Using High-End Management Scenarios for Wastewater Tanks (Baton Rouge, LA)

CAS Number	Chemical Names	Sediment (mg/kg)	Surface water (mg/L)		Soil (mg/kg)	Plant (mg/kg DW)
			Total	Dissolved		
67-64-1	Acetone	1.2e-08	6.0e-07	6.0e-07	3.5e-09	5.0e-07
107-05-1	Allyl chloride	5.2e-08	5.8e-08	5.8e-08	1.5e-09	1.5e-07
65-85-0	Benzoic acid	6.9e-08	3.1e-08	3.1e-08	6.9e-07	4.7e-05
100-51-6	Benzyl alcohol	2.2e-10	5.3e-10	5.3e-10	1.1e-08	9.5e-07
111-44-4	Bis (2-chloroethyl) ether	2.2e-06	1.9e-07	1.9e-07	4.4e-07	3.4e-05
39638-32-9	Bis (2-chloroisopropyl) ether	5.1e-07	9.8e-07	9.8e-07	4.3e-07	3.2e-05
117-81-7	Bis(2-ethylhexyl) phthalate	8.3e-08	1.3e-11	1.2e-13	2.3e-06	7.5e-03
75-27-4	Bromodichloromethane	8.9e-08	1.6e-08	1.6e-08	1.1e-08	4.2e-07
75-25-2	Bromoform	1.2e-07	1.3e-08	1.3e-08	3.3e-08	8.7e-07
75-15-0	Carbon disulfide	6.7e-08	2.2e-08	2.2e-08	1.0e-09	1.0e-07
126-99-8	Chloro-1,3-butadiene, 2-	2.0e-07	5.4e-08	5.4e-08	5.1e-09	5.2e-07
124-48-1	Chlorodibromomethane	1.3e-07	6.1e-09	6.1e-09	8.3e-09	7.6e-07
67-66-3	Chloroform	3.2e-07	5.4e-08	5.4e-08	6.4e-08	2.1e-06
108-90-7	Chlorobenzene	7.8e-06	3.0e-06	3.0e-06	3.9e-07	3.9e-05
95-48-7	Cresol, o-	3.6e-10	1.2e-10	1.2e-10	3.6e-09	3.1e-07
106-44-5	Cresol, p-	3.3e-10	1.2e-10	1.2e-10	5.0e-09	4.4e-07
107-06-2	Dichloroethane, 1,2-	6.0e-07	6.4e-07	6.4e-07	4.3e-08	4.1e-06
156-59-2	Dichloroethylene, cis-1,2-	7.9e-08	3.5e-08	3.5e-08	4.9e-09	3.9e-07
156-60-5	Dichloroethylene, trans-1,2-	4.8e-08	9.6e-09	9.6e-09	1.6e-09	1.2e-07
84-66-2	Diethyl phthalate	4.0e-08	4.2e-09	4.2e-09	1.6e-06	1.0e-04
131-11-3	Dimethyl phthalate	1.2e-11	7.3e-12	7.3e-12	9.1e-10	7.9e-08
117-84-0	Di-n-octyl phthalate	7.2e-06	1.1e-09	1.8e-12	2.0e-04	5.6e-03
100-41-4	Ethylbenzene	2.8e-07	4.8e-09	4.7e-09	8.8e-09	8.0e-07
75-00-3	Ethyl chloride	4.6e-08	5.4e-08	5.4e-08	8.6e-10	1.5e-07
118-74-1	Hexachlorobenzene	6.1e-04	1.1e-07	2.1e-08	1.4e-04	3.7e-04
78-93-3	Methyl ethyl ketone	5.1e-08	8.0e-07	8.0e-07	1.5e-08	1.5e-06
75-09-2	Methylene chloride	3.3e-08	4.2e-08	4.2e-08	1.6e-09	1.3e-07
7439-97-6	Methyl mercury	3.4e-06	3.7e-09	3.4e-09	9.8e-07	2.3e-03
87-86-5	Pentachlorophenol	4.2e-08	1.5e-11	8.6e-12	1.8e-06	2.8e-04
108-95-2	Phenol	5.8e-10	6.1e-10	6.1e-10	1.6e-08	1.4e-06
100-42-5	Styrene	1.6e-06	6.2e-08	6.2e-08	1.2e-07	1.0e-05
1746-01-6	TCDD 2,3,7,8- (TEQ)	1.8e-09	2.8e-13	4.0e-15	4.5e-08	9.0e-07
127-18-4	Tetrachloroethylene	2.9e-07	2.0e-08	2.0e-08	7.5e-09	6.6e-07
79-01-6	Trichloroethylene	3.1e-09	1.7e-10	1.7e-10	9.8e-11	9.2e-09
95-95-4	Trichlorophenol, 2,4,5-	2.5e-07	1.1e-09	1.1e-09	1.8e-06	1.2e-04
88-06-2	Trichlorophenol, 2,4,6-	2.1e-05	1.5e-07	1.5e-07	7.8e-05	4.9e-03

Table 11. Modeled Media Concentrations for Constituents Using High-End Management Scenarios for Wastewater Tanks (Baltimore, MD)

CAS Number	Chemical Names	Sediment (mg/kg)	Surface water (mg/L)		Soil (mg/kg)	Plant (mg/kg DW)
			Total	Dissolved		
67-64-1	Acetone	1.3e-08	6.0e-07	6.0e-07	3.5e-09	5.0e-07
107-05-1	Allyl chloride	5.2e-08	5.8e-08	5.8e-08	1.5e-09	1.5e-07
65-85-0	Benzoic acid	6.9e-08	3.1e-08	3.1e-08	6.9e-07	4.7e-05
100-51-6	Benzyl alcohol	2.2e-10	5.3e-10	5.3e-10	1.1e-08	9.5e-07
111-44-4	Bis (2-chloroethyl) ether	2.9e-06	1.9e-07	1.9e-07	4.9e-07	3.4e-05
39638-32-9	Bis (2-chloroisopropyl) ether	5.1e-07	9.8e-07	9.8e-07	4.3e-07	3.2e-05
117-81-7	Bis(2-ethylhexyl) phthalate	6.6e-08	1.0e-11	6.0e-14	4.4e-06	7.5e-03
75-27-4	Bromodichloromethane	1.6e-07	1.9e-08	1.9e-08	2.0e-08	5.6e-07
75-25-2	Bromoform	2.2e-07	1.5e-08	1.5e-08	5.6e-08	1.0e-06
75-15-0	Carbon disulfide	7.3e-08	2.2e-08	2.2e-08	1.0e-09	1.0e-07
126-99-8	Chloro-1,3-butadiene, 2-	2.1e-07	5.4e-08	5.4e-08	5.1e-09	5.2e-07
124-48-1	Chlorodibromomethane	2.0e-07	6.1e-09	6.1e-09	1.1e-08	7.6e-07
67-66-3	Chloroform	5.9e-07	5.9e-08	5.9e-08	1.2e-07	2.5e-06
108-90-7	Chlorobenzene	8.9e-06	3.0e-06	3.0e-06	3.9e-07	3.9e-05
95-48-7	Cresol, o-	3.6e-10	1.2e-10	1.2e-10	3.6e-09	3.1e-07
106-44-5	Cresol, p-	3.3e-10	1.2e-10	1.2e-10	5.0e-09	4.4e-07
107-06-2	Dichloroethane, 1,2-	7.0e-07	6.4e-07	6.4e-07	4.3e-08	4.1e-06
156-59-2	Dichloroethylene, cis-1,2-	9.6e-08	3.5e-08	3.5e-08	5.2e-09	3.9e-07
156-60-5	Dichloroethylene, trans-1,2-	7.6e-08	9.6e-09	9.6e-09	3.0e-09	1.4e-07
84-66-2	Diethyl phthalate	5.4e-08	4.2e-09	4.2e-09	2.0e-06	1.0e-04
131-11-3	Dimethyl phthalate	8.6e-12	7.3e-12	7.3e-12	7.5e-10	7.9e-08
117-84-0	Di-n-octyl phthalate	3.3e-06	4.9e-10	5.2e-13	2.1e-04	4.9e-03
100-41-4	Ethylbenzene	4.4e-07	5.0e-09	4.9e-09	1.7e-08	9.5e-07
75-00-3	Ethyl chloride	5.0e-08	5.4e-08	5.4e-08	8.6e-10	1.5e-07
118-74-1	Hexachlorobenzene	8.2e-04	1.4e-07	1.8e-08	1.9e-04	3.7e-04
78-93-3	Methyl ethyl ketone	5.2e-08	8.0e-07	8.0e-07	1.5e-08	1.5e-06
75-09-2	Methylene chloride	6.0e-08	4.8e-08	4.8e-08	3.1e-09	1.8e-07
7439-97-6	Methyl mercury	3.4e-06	3.7e-09	3.4e-09	9.8e-07	2.3e-03
87-86-5	Pentachlorophenol	6.2e-08	1.8e-11	8.3e-12	5.4e-06	2.8e-04
108-95-2	Phenol	5.8e-10	6.1e-10	6.1e-10	1.6e-08	1.4e-06
100-42-5	Styrene	2.2e-06	6.2e-08	6.2e-08	1.4e-07	1.0e-05
1746-01-6	TCDD 2,3,7,8- (TEQ)	3.6e-09	5.5e-13	4.9e-15	1.7e-07	9.1e-07
127-18-4	Tetrachloroethylene	3.1e-07	2.0e-08	2.0e-08	7.5e-09	6.6e-07
79-01-6	Trichloroethylene	3.7e-09	1.7e-10	1.7e-10	1.3e-10	9.2e-09
95-95-4	Trichlorophenol, 2,4,5-	2.8e-07	1.1e-09	1.1e-09	1.8e-06	1.2e-04
88-06-2	Trichlorophenol, 2,4,6-	2.1e-05	1.5e-07	1.5e-07	7.8e-05	4.9e-03

Table 12. Modeled Media Concentrations for Constituents Using Central Tendency Management Scenarios for Wastewater Tanks (Memphis, TN)

CAS Number	Chemical Names	Sediment (mg/kg)	Surface water (mg/L)		Soil (mg/kg)	Plant (mg/kg DW)
			Total	Dissolved		
67-64-1	Acetone	1.6e-09	8.4e-08	8.4e-08	4.9e-10	7.1e-08
107-05-1	Allyl chloride	5.8e-09	6.5e-09	6.5e-09	1.7e-10	1.7e-08
65-85-0	Benzoic acid	7.2e-09	3.2e-09	3.2e-09	7.2e-08	4.9e-06
100-51-6	Benzyl alcohol	4.2e-11	1.0e-10	1.0e-10	2.2e-09	1.8e-07
111-44-4	Bis (2-chloroethyl) ether	3.1e-07	2.7e-08	2.7e-08	6.3e-08	4.8e-06
39638-32-9	Bis (2-chloroisopropyl) ether	4.3e-08	8.4e-08	8.4e-08	3.6e-08	2.7e-06
117-81-7	Bis(2-ethylhexyl) phthalate	3.8e-09	5.8e-13	7.6e-15	8.5e-08	2.2e-03
75-27-4	Bromodichloromethane	2.6e-08	6.7e-09	6.7e-09	3.9e-09	1.8e-07
75-25-2	Bromoform	3.5e-08	5.2e-09	5.2e-09	1.1e-08	3.4e-07
75-15-0	Carbon disulfide	1.1e-08	3.4e-09	3.4e-09	1.6e-10	1.6e-08
126-99-8	Chloro-1,3-butadiene, 2-	3.2e-08	8.7e-09	8.7e-09	8.3e-10	8.5e-08
124-48-1	Chlorodibromomethane	4.1e-08	1.9e-09	1.9e-09	2.6e-09	2.4e-07
67-66-3	Chloroform	8.1e-08	1.8e-08	1.8e-08	1.9e-08	6.7e-07
108-90-7	Chlorobenzene	9.7e-07	3.8e-07	3.8e-07	4.9e-08	4.9e-06
95-48-7	Cresol, o-	6.5e-11	2.1e-11	2.1e-11	6.6e-10	5.7e-08
106-44-5	Cresol, p-	4.4e-11	1.6e-11	1.6e-11	6.6e-10	5.8e-08
107-06-2	Dichloroethane, 1,2-	8.5e-08	9.1e-08	9.1e-08	6.2e-09	5.9e-07
156-59-2	Dichloroethylene, cis-1,2-	1.5e-08	6.6e-09	6.6e-09	9.3e-10	7.3e-08
156-60-5	Dichloroethylene, trans-1,2-	1.2e-08	3.4e-09	3.4e-09	5.4e-10	4.3e-08
84-66-2	Diethyl phthalate	3.4e-09	3.5e-10	3.5e-10	1.4e-07	8.5e-06
131-11-3	Dimethyl phthalate	2.2e-12	1.9e-12	1.9e-12	1.9e-10	2.0e-08
117-84-0	Di-n-octyl phthalate	2.9e-07	4.3e-11	1.0e-13	7.0e-06	1.8e-03
100-41-4	Ethylbenzene	8.8e-08	2.2e-09	2.2e-09	3.9e-09	3.8e-07
75-00-3	Ethyl chloride	8.0e-09	9.4e-09	9.4e-09	1.5e-10	2.6e-08
118-74-1	Hexachlorobenzene	4.4e-05	8.7e-09	2.1e-09	4.3e-05	1.4e-04
78-93-3	Methyl ethyl ketone	4.5e-09	7.1e-08	7.1e-08	1.3e-09	1.3e-07
75-09-2	Methylene chloride	8.9e-09	1.6e-08	1.6e-08	5.4e-10	5.0e-08
7439-97-6	Methyl mercury	5.2e-07	5.6e-10	5.2e-10	1.5e-07	3.4e-04
87-86-5	Pentachlorophenol	3.2e-09	1.4e-12	9.5e-13	8.2e-08	4.0e-05
108-95-2	Phenol	6.9e-11	7.2e-11	7.2e-11	1.9e-09	1.7e-07
100-42-5	Styrene	3.2e-07	1.2e-08	1.2e-08	2.3e-08	2.1e-06
1746-01-6	TCDD 2,3,7,8- (TEQ)	3.7e-11	5.6e-15	7.4e-17	8.3e-10	6.0e-08
127-18-4	Tetrachloroethylene	5.1e-08	3.6e-09	3.6e-09	1.3e-09	1.2e-07
79-01-6	Trichloroethylene	6.2e-10	4.1e-11	4.0e-11	2.3e-11	2.2e-09
95-95-4	Trichlorophenol, 2,4,5-	3.9e-08	1.8e-10	1.7e-10	2.8e-07	1.8e-05
88-06-2	Trichlorophenol, 2,4,6-	2.3e-06	1.7e-08	1.6e-08	8.6e-06	5.4e-04

Table 13. Hazard Quotients (HQs) Calculated for Constituents Using High-End Management Scenarios for Wastewater Tanks (Baton Rouge, LA)

(ID = Insufficient data available)

CAS Number	Chemical Names	Sediment	Surface water		Soil	Plant
			Total	Dissolved		
67-64-1	Acetone	1.3e-06	4.0e-07	ID	ID	ID
107-05-1	Allyl chloride	ID	ID	ID	ID	ID
65-85-0	Benzoic acid	ID	7.3e-07	ID	ID	ID
100-51-6	Benzyl alcohol	2.0e-07	6.1e-08	ID	ID	ID
111-44-4	Bis (2-chloroethyl) ether	ID	1.9e-10	ID	ID	ID
39638-32-9	Bis (2-chloroisopropyl) ether	ID	ID	ID	ID	ID
117-81-7	Bis(2-ethylhexyl) phthalate	4.6e-07	4.2e-09	ID	2.6e-06	4.7e-04
75-27-4	Bromodichloromethane	ID	ID	ID	ID	ID
75-25-2	Bromoform	ID	ID	ID	ID	ID
75-15-0	Carbon disulfide	7.9e-05	2.4e-05	ID	ID	ID
126-99-8	Chloro-1,3-butadiene, 2-	ID	ID	ID	ID	ID
124-48-1	Chlorodibromomethane	ID	ID	ID	ID	ID
67-66-3	Chloroform	1.4e-05	1.9e-06	ID	9.0e-10	2.5e-08
108-90-7	Chlorobenzene	1.9e-05	4.7e-05	ID	9.8e-09	ID
95-48-7	Cresol, o-	ID	2.8e-12	ID	2.6e-12	2.1e-10
106-44-5	Cresol, p-	ID	ID	ID	ID	ID
107-06-2	Dichloroethane, 1,2-	2.4e-06	7.0e-07	ID	3.1e-09	2.8e-08
156-59-2	Dichloroethylene, cis-1,2-	ID	6.0e-08	ID	ID	ID
156-60-5	Dichloroethylene, trans-1,2-	ID	1.6e-08	ID	ID	ID
84-66-2	Diethyl phthalate	ID	2.0e-08	ID	1.4e-10	ID
131-11-3	Dimethyl phthalate	2.0e-11	5.2e-11	ID	4.5e-12	ID
117-84-0	Di-n-octyl phthalate	ID	ID	ID	ID	ID
100-41-4	Ethylbenzene	3.1e-06	6.5e-07	ID	8.8e-08	ID
75-00-3	Ethyl chloride	ID	ID	ID	ID	ID
118-74-1	Hexachlorobenzene	8.0e-06	1.5e-02	ID	2.9e-04	4.4e-05
78-93-3	Methyl ethyl ketone	ID	2.2e-10	ID	1.8e-12	1.6e-10
75-09-2	Methylene chloride	8.8e-08	1.9e-08	ID	ID	ID
7439-97-6	Methyl mercury	ID	1.9e-01	ID	4.9e-05	7.3e-03
87-86-5	Pentachlorophenol	ID	4.4e-09	ID	5.9e-07	1.3e-05
108-95-2	Phenol	1.9e-08	5.5e-09	ID	5.2e-10	ID
100-42-5	Styrene	ID	ID	ID	3.8e-10	ID
1746-01-6	TCDD 2,3,7,8- (TEQ)	9.2e-06	2.1e-02	ID	2.8e-02	1.6e-01
127-18-4	Tetrachloroethylene	7.0e-07	2.1e-07	ID	4.2e-10	ID
79-01-6	Trichloroethylene	1.4e-08	1.1e-08	ID	5.4e-11	4.4e-09
95-95-4	Trichlorophenol, 2,4,5-	ID	ID	ID	2.0e-07	ID
88-06-2	Trichlorophenol, 2,4,6-	ID	ID	ID	7.8e-06	ID

Table 14. Hazard Quotients (HQs) Calculated for Constituents Using High-End Management Scenarios for Wastewater Tanks (Baltimore, MD)

(ID = Insufficient data available)

CAS Number	Chemical Names	Sediment	Surface water		Soil	Plant
			Total	Dissolved		
67-64-1	Acetone	1.4e-06	4.0e-07	ID	ID	ID
107-05-1	Allyl chloride	ID	ID	ID	ID	ID
65-85-0	Benzoic acid	ID	7.3e-07	ID	ID	ID
100-51-6	Benzyl alcohol	2.0e-07	6.1e-08	ID	ID	ID
111-44-4	Bis (2-chloroethyl) ether	ID	1.9e-10	ID	ID	ID
39638-32-9	Bis (2-chloroisopropyl) ether	ID	ID	ID	ID	ID
117-81-7	Bis(2-ethylhexyl) phthalate	3.7e-07	3.3e-09	ID	4.9e-06	4.7e-04
75-27-4	Bromodichloromethane	ID	ID	ID	ID	ID
75-25-2	Bromoform	ID	ID	ID	ID	ID
75-15-0	Carbon disulfide	8.6e-05	2.4e-05	ID	ID	ID
126-99-8	Chloro-1,3-butadiene, 2-	ID	ID	ID	ID	ID
124-48-1	Chlorodibromomethane	ID	ID	ID	ID	ID
67-66-3	Chloroform	2.7e-05	2.1e-06	ID	1.6e-09	3.0e-08
108-90-7	Chlorobenzene	2.2e-05	4.7e-05	ID	9.8e-09	ID
95-48-7	Cresol, o-	ID	2.8e-12	ID	2.6e-12	2.1e-10
106-44-5	Cresol, p-	ID	ID	ID	ID	ID
107-06-2	Dichloroethane, 1,2-	2.8e-06	7.0e-07	ID	3.1e-09	2.8e-08
156-59-2	Dichloroethylene, cis-1,2-	ID	6.0e-08	ID	ID	ID
156-60-5	Dichloroethylene, trans-1,2-	ID	1.6e-08	ID	ID	ID
84-66-2	Diethyl phthalate	ID	2.0e-08	ID	1.7e-10	ID
131-11-3	Dimethyl phthalate	1.4e-11	5.2e-11	ID	3.7e-12	ID
117-84-0	Di-n-octyl phthalate	ID	ID	ID	ID	ID
100-41-4	Ethylbenzene	5.0e-06	6.8e-07	ID	1.7e-07	ID
75-00-3	Ethyl chloride	ID	ID	ID	ID	ID
118-74-1	Hexachlorobenzene	1.1e-05	1.9e-02	ID	3.9e-04	4.4e-05
78-93-3	Methyl ethyl ketone	ID	2.2e-10	ID	1.8e-12	1.6e-10
75-09-2	Methylene chloride	1.6e-07	2.2e-08	ID	ID	ID
7439-97-6	Methyl mercury	ID	1.9e-01	ID	4.9e-05	7.3e-03
87-86-5	Pentachlorophenol	ID	5.2e-09	ID	1.8e-06	1.3e-05
108-95-2	Phenol	1.9e-08	5.5e-09	ID	5.2e-10	ID
100-42-5	Styrene	ID	ID	ID	4.5e-10	ID
1746-01-6	TCDD 2,3,7,8- (TEQ)	1.8e-05	4.2e-02	ID	1.1e-01	1.7e-01
127-18-4	Tetrachloroethylene	7.5e-07	2.1e-07	ID	4.2e-10	ID
79-01-6	Trichloroethylene	1.7e-08	1.1e-08	ID	7.0e-11	4.4e-09
95-95-4	Trichlorophenol, 2,4,5-	ID	ID	ID	2.0e-07	ID
88-06-2	Trichlorophenol, 2,4,6-	ID	ID	ID	7.8e-06	ID

Table 15. Hazard Quotients (HQs) Calculated for Constituents Using Central Tendency Management Scenarios for Wastewater Tanks (Memphis, TN)

(ID = Insufficient data available)

CAS Number	Chemical Names	Sediment	Surface water		Soil	Plant
			Total	Dissolved		
67-64-1	Acetone	1.9e-07	5.6e-08	ID	ID	ID
107-05-1	Allyl chloride	ID	ID	ID	ID	ID
65-85-0	Benzoic acid	ID	7.7e-08	ID	ID	ID
100-51-6	Benzyl alcohol	3.8e-08	1.2e-08	ID	ID	ID
111-44-4	Bis (2-chloroethyl) ether	ID	2.7e-11	ID	ID	ID
39638-32-9	Bis (2-chloroisopropyl) ether	ID	ID	ID	ID	ID
117-81-7	Bis(2-ethylhexyl) phthalate	2.1e-08	1.9e-10	ID	9.6e-08	1.4e-04
75-27-4	Bromodichloromethane	ID	ID	ID	ID	ID
75-25-2	Bromoform	ID	ID	ID	ID	ID
75-15-0	Carbon disulfide	1.2e-05	3.7e-06	ID	ID	ID
126-99-8	Chloro-1,3-butadiene, 2-	ID	ID	ID	ID	ID
124-48-1	Chlorodibromomethane	ID	ID	ID	ID	ID
67-66-3	Chloroform	3.7e-06	6.3e-07	ID	2.7e-10	8.1e-09
108-90-7	Chlorobenzene	2.4e-06	5.9e-06	ID	1.2e-09	ID
95-48-7	Cresol, o-	ID	5.1e-13	ID	4.7e-13	3.8e-11
106-44-5	Cresol, p-	ID	ID	ID	ID	ID
107-06-2	Dichloroethane, 1,2-	3.4e-07	1.0e-07	ID	4.4e-10	3.9e-09
156-59-2	Dichloroethylene, cis-1,2-	ID	1.1e-08	ID	ID	ID
156-60-5	Dichloroethylene, trans-1,2-	ID	5.7e-09	ID	ID	ID
84-66-2	Diethyl phthalate	ID	1.7e-09	ID	1.1e-11	ID
131-11-3	Dimethyl phthalate	3.6e-12	1.3e-11	ID	9.5e-13	ID
117-84-0	Di-n-octyl phthalate	ID	ID	ID	ID	ID
100-41-4	Ethylbenzene	9.9e-07	3.0e-07	ID	3.9e-08	ID
75-00-3	Ethyl chloride	ID	ID	ID	ID	ID
118-74-1	Hexachlorobenzene	5.7e-07	1.2e-03	ID	8.8e-05	1.7e-05
78-93-3	Methyl ethyl ketone	ID	2.0e-11	ID	1.6e-13	1.4e-11
75-09-2	Methylene chloride	2.4e-08	7.2e-09	ID	ID	ID
7439-97-6	Methyl mercury	ID	2.8e-02	ID	7.4e-06	1.1e-03
87-86-5	Pentachlorophenol	ID	4.2e-10	ID	2.7e-08	1.8e-06
108-95-2	Phenol	2.2e-09	6.5e-10	ID	6.2e-11	ID
100-42-5	Styrene	ID	ID	ID	7.6e-11	ID
1746-01-6	TCDD 2,3,7,8- (TEQ)	1.9e-07	4.3e-04	ID	5.2e-04	1.1e-02
127-18-4	Tetrachloroethylene	1.2e-07	3.7e-08	ID	7.4e-11	ID
79-01-6	Trichloroethylene	2.8e-09	2.5e-09	ID	1.3e-11	1.0e-09
95-95-4	Trichlorophenol, 2,4,5-	ID	ID	ID	3.1e-08	ID
88-06-2	Trichlorophenol, 2,4,6-	ID	ID	ID	8.6e-07	ID

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Attachment 3

Analyses to Support the Development of a 2,3,7,8-TCDD Toxicity Equivalent (TEQ) Limit for Wastewaters

Table 1, Attachment 3.
Wastewater Tank Scenario
Single High End Ingestion Risk Results for the Farmer
Based on Data from Sample PL-02 (TCDD I-TEQ = 0.66 ng/L)

High End Parameter(s)	TCDD, 2,3,7,8-	OCDD, 1,2,3,4,5, 7,8,9-	HxCDD, 1,2,3,7,8, 9-	HpCDD, 1,2,3,4,6, 7,8,-	OCDF, 1,2,3,4,6, 7,8,9-	HxCDD, 1,2,3,4,7, 8-	TCDF, 2,3,7,8-	HpCDF,1, 2,3,4,7,8, 9-	PeCDF, 2,3,4,7,8-	HxCDF, 1,2,3,6,7, 8-	HxCDD, 1,2,3,6,7, 8-	HxCDF, 2,3,4,6,7, 8-	HpCDF,1, 2,3,4,6,7, 8-	HxCDF, 1,2,3,4,7, 8-	HxCDF, 1,2,3,7,8, 9-	TEQ
Central Tendency	1.E-08	8.E-13	3.E-08	8.E-10	7.E-11	1.E-08	3.E-09	7.E-08	***1.E-06***	1.E-07	3.E-08	1.E-07	3.E-08	7.E-07	7.E-08	***3.E-06***
Single High End Parameter																
Exposure Duration	NA	NA	NA	NA	3.E-10	NA	NA	3.E-07	***6.E-06***	6.E-07	NA	5.E-07	1.E-07	***3.E-06***	4.E-07	***1.E-05***
Beef intake	NA	NA	NA	NA	1.E-10	NA	NA	1.E-07	***3.E-06***	3.E-07	NA	2.E-07	6.E-08	***2.E-06***	2.E-07	***5.E-06***
Dairy Intake	NA	NA	NA	NA	1.E-10	NA	NA	1.E-07	***2.E-06***	3.E-07	NA	2.E-07	6.E-08	***1.E-06***	1.E-07	***5.E-06***
Exposed Veg. Intake	NA	NA	NA	NA	7.E-11	NA	NA	7.E-08	***1.E-06***	1.E-07	NA	1.E-07	3.E-08	7.E-07	7.E-08	***2.E-06***
Root Veg. Intake	NA	NA	NA	NA	7.E-11	NA	NA	7.E-08	***1.E-06***	1.E-07	NA	1.E-07	3.E-08	7.E-07	7.E-08	***2.E-06***
Fruit Intake	NA	NA	NA	NA	8.E-11	NA	NA	7.E-08	***1.E-06***	1.E-07	NA	1.E-07	3.E-08	7.E-07	7.E-08	***2.E-06***
Meteorological Location	NA	NA	NA	NA	9.E-11	NA	NA	9.E-08	***2.E-06***	2.E-07	NA	1.E-07	4.E-08	9.E-07	1.E-07	***3.E-06***
Distance to Receptor	NA	NA	NA	NA	3.E-10	NA	NA	3.E-07	***6.E-06***	6.E-07	NA	5.E-07	1.E-07	***3.E-06***	3.E-07	***1.E-05***
Waste Quantity	NA	NA	NA	NA	2.E-10	NA	NA	2.E-07	***3.E-06***	3.E-07	NA	3.E-07	8.E-08	***2.E-06***	2.E-07	***6.E-06***

Table 2, Attachment 3.
Wastewater Tank Scenario
Single High End Ingestion Risk Results for the Adult Resident
Based on Data from Sample PL-02 (TCDD I-TEQ = 0.66 ng/L)

High End Parameter(s)	TCDD, 2,3,7,8-	OCDD, 1,2,3,4,5,7,8,9-	HxCDD, 1,2,3,7,8,9-	HpCDD, 1,2,3,4,6,7,8,-	OCDF, 1,2,3,4,6,7,8,9-	HxCDD, 1,2,3,4,7,8-	TCDF, 2,3,7,8-	HpCDF,1,2,3,4,7,8,9-	PeCDF, 2,3,4,7,8-	HxCDF, 1,2,3,6,7,8-	HxCDD, 1,2,3,6,7,8-	HxCDF, 2,3,4,6,7,8-	HpCDF,1,2,3,4,6,7,8-	HxCDF, 1,2,3,4,7,8-	HxCDF, 1,2,3,7,8,9-	TEQ
Central Tendency	NA	NA	NA	NA	2.E-15	NA	NA	2.E-12	9.E-12	5.E-12	NA	4.E-12	2.E-12	2.E-11	3.E-12	4.E-11
Single High End Parameter																
Exposure Duration	NA	NA	NA	NA	5.E-15	NA	NA	6.E-12	3.E-11	2.E-11	NA	1.E-11	6.E-12	7.E-11	8.E-12	1.E-10
Meteorological Location	NA	NA	NA	NA	6.E-14	NA	NA	6.E-11	3.E-10	2.E-10	NA	1.E-10	6.E-11	6.E-10	8.E-11	1.E-09
Distance to Receptor	NA	NA	NA	NA	5.E-15	NA	NA	6.E-12	3.E-11	1.E-11	NA	1.E-11	6.E-12	6.E-11	8.E-12	1.E-10
Waste Quantity	NA	NA	NA	NA	4.E-15	NA	NA	5.E-12	2.E-11	1.E-11	NA	1.E-11	5.E-12	5.E-11	6.E-12	1.E-10

Table 3, Attachment 3.
Wastewater Tank Scenario
Single High End Ingestion Risk Results for the Farmer
Based on Data From Sample PL-01 (TCDD I-TEQ = 0.31 ng/L)

High End Parameter(s)	**TCDD, 2,3,7,8-**	**OCDD, 1,2,3,4,5, 7,8,9-**	**HxCDD, 1,2,3,7,8, 9-**	**HpCDD, 1,2,3,4,6, 7,8,-**	**OCDF, 1,2,3,4,6, 7,8,9-**	**HxCDD, 1,2,3,4,7, 8-**	**TCDF, 2,3,7,8-**	**HpCDF,1, 2,3,4,7,8, 9-**	**PeCDF, 2,3,4,7,8-**	**HxCDF, 1,2,3,6,7, 8-**	**HxCDD, 1,2,3,6,7, 8-**	**HxCDF, 2,3,4,6,7, 8-**	**HpCDF,1, 2,3,4,6,7, 8-**	**HxCDF, 1,2,3,4,7, 8-**	**HxCDF, 1,2,3,7,8, 9-**	**TEQ**
Central Tendency	NA	8.E-13	NA	5.E-10	7.E-11	NA	NA	4.E-08	NA	3.E-08	NA	2.E-08	3.E-08	2.E-07	2.E-08	4.E-07
Single High End Parameter																
Exposure Duration	NA	4.E-12	NA	2.E-09	3.E-10	NA	NA	2.E-07	NA	2.E-07	NA	1.E-07	2.E-07	***1.E-06***	7.E-08	***2.E-06***
Beef intake	NA	2.E-12	NA	1.E-09	1.E-10	NA	NA	8.E-08	NA	7.E-08	NA	5.E-08	7.E-08	4.E-07	3.E-08	7.E-07
Dairy Intake	NA	1.E-12	NA	1.E-09	1.E-10	NA	NA	9.E-08	NA	7.E-08	NA	4.E-08	6.E-08	4.E-07	3.E-08	7.E-07
Exposed Veg. Intake	NA	8.E-13	NA	5.E-10	7.E-11	NA	NA	4.E-08	NA	3.E-08	NA	2.E-08	3.E-08	2.E-07	2.E-08	4.E-07
Root Veg. Intake	NA	8.E-13	NA	5.E-10	7.E-11	NA	NA	4.E-08	NA	3.E-08	NA	2.E-08	3.E-08	2.E-07	2.E-08	4.E-07
Fruit Intake	NA	9.E-13	NA	6.E-10	8.E-11	NA	NA	4.E-08	NA	3.E-08	NA	2.E-08	3.E-08	2.E-07	2.E-08	4.E-07
Meteorological Location	NA	1.E-12	NA	7.E-10	9.E-11	NA	NA	5.E-08	NA	4.E-08	NA	3.E-08	4.E-08	3.E-07	2.E-08	5.E-07
Distance to Receptor	NA	5.E-12	NA	3.E-09	4.E-10	NA	NA	3.E-07	NA	2.E-07	NA	1.E-07	2.E-07	***1.E-06***	1.E-07	***2.E-06***
Waste Quantity	NA	2.E-12	NA	1.E-09	2.E-10	NA	NA	1.E-07	NA	9.E-08	NA	5.E-08	8.E-08	5.E-07	4.E-08	9.E-07