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May 16, 2008 Project No. C246.0101

Mr. Matt McClincy
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Northwest Region
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Subject: **Draft Upland Human Health Risk Assessment**
Arkema Portland Facility

Dear Mr. McClincy:

On behalf of Legacy Site Services Inc., enclosed are three paper copies and one CD of the Draft Upland Human Health Risk Assessment for the Arkema Portland Facility. We look forward to receiving your comments and meeting with you and finalizing the report.

If you have any questions, please feel free to contact me at 503-284-5545.

Sincerely,

David Livermore, R.G., L.H.G.
Principal-in-Charge

Enclosures

cc: Todd Slater, LSS
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HUMAN HEALTH RISK ASSESSMENT
Arkema Site: Upland Areas

DRAFT

Prepared for
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ACRONYMS AND ABBREVIATIONS

ADAF	age-dependent adjustment factor
AF	absorption factor
ALM	adult lead methodology
Arkema	Arkema, Inc.
AS/SVE	air sparging and soil vapor extraction
ATOFINA	ATOFINA Chemicals, Inc.
ATSDR	U.S. Agency for Toxic Substances and Disease Registry
BaP	benzo(a)pyrene
BEHP	bis(2-ethylhexyl)phthalate
bgs	below ground surface
BKSF	biokinetic slope factor
BMP	best management practice
BPA	Bonneville Power Administration
BTEX	benzene, toluene, ethylbenzene, and xylene
CDC	Centers for Disease Control and Prevention
COI	chemical of interest
COPC	chemical of potential concern
CrIII	trivalent chromium
CrVI	hexavalent chromium
CSF	cancer slope factor
CSM	conceptual site model
CTE	central tendency exposure
DCB	dichlorobenzene
DDD	dichlorodiphenyldichloroethane
DDE	dichlorodiphenyldichloroethylene
DDT	dichlorodiphenyltrichloroethane
DEQ	Oregon Department of Environmental Quality
DNAPL	dense, nonaqueous-phase liquid

DRO	diesel range organic hydrocarbon
DSL	Oregon Department of State Lands
ECSI	Environmental Cleanup Site Information
ED	effective dose
EE/CA	engineering evaluation and cost analysis
ELF	Société Nationale Elf Aquitaine
EPA	U.S. Environmental Protection Agency
EPC	exposure point concentration
FCTF	Former Caustic Tank Farm
FFS	focused feasibility study
GLISP	Guild's Lake Industrial Sanctuary Plan
GSD_i	individual geometric standard deviation
HEAST	Health Effects Assessment Summary Tables
HHRA	Human Health Risk Assessment
HI	hazard index
HQ	hazard quotient
Integral	Integral Consulting Inc.
IRIS	Integrated Risk Information System
IRM	interim remedial measure
LADD	lifetime average daily dose
LOAEL	lowest-observed-adverse-effect level
LOF	locality of the facility
LSS	Legacy Site Services
MCB	monochlorobenzene
MPR	manufacturing process residue
MRL	minimal risk level
MSSL	medium-specific screening level
NOAEL	no-observed-adverse-effect level
NPDES	National Pollution Discharge Elimination System
OCTF	Old Caustic Tank Farm

PAH	polycyclic aromatic hydrocarbon
PbB_0	"baseline" blood lead level
PBPK	physiologically-based pharmacokinetic
PCB	polychlorinated biphenyl
PCDD	polychlorinated dibenzo-p-dioxin
PCDD/F	polychlorinated dibenzo-p-dioxin/P dibenzofuran
PCDF	polychlorinated dibenzofuran
PCE	tetrachloroethene (also referred to as "tetrachloroethylene")
PCP	pentachlorophenol
Pennwalt	Pennwalt Corporation
ppm	parts per million
PPRTV	Provisional Peer Reviewed Toxicity Value
PRG	Preliminary Remediation Goal
RAF	relative absorption factor
RAIS	Risk Assessment Information System
RBC	risk-based concentration
RCRA	Resource Conservation and Recovery Act
RfC	reference concentration
RfD	reference dose
RI	remedial investigation
RI/FS	remedial investigation and feasibility study
RM	River Mile
RME	reasonable maximum exposure
ROD	record of decision
RP	Rhône Poulenc
RPF	relative potency factor
RRO	residual range organic hydrocarbons
TCDD	tetrachlorodibenzo-*p*-dioxin
TCE	trichloroethene (also referred to as "trichloroethylene")
TEF	toxic equivalency factor

TEQ	total toxic equivalency
TPH	total petroleum hydrocarbon
UCL	upper confidence limit
URF	unit risk factor
USEPA-ECAO	EPA Environmental Criteria and Assessment Office
VOC	volatile organic compound
WOE	weight-of-evidence

EXECUTIVE SUMMARY

This report presents the results of the Arkema Upland Human Health Risk Assessment (HHRA) conducted by Integral Consulting Inc (Integral) for Legacy Site Services (LSS). The HHRA was performed in conjunction with the Arkema Site Remedial Investigation (RI) that is being conducted under the Oregon Department of Environmental Quality (DEQ) Voluntary Cleanup Program[1]. The HHRA followed the relevant DEQ HHRA guidance and recommendations (DEQ 2000, 2003, 2007).

The Site is located in an industrial area along the southwest bank of the Willamette River in Portland, Oregon. Historically, the Site was occupied by a chloro-alkali plant from 1941 until 2001, when the facility was closed for economic reasons. Decommissioning and removal of the manufacturing infrastructure was completed in 2005.

The Site is composed of four lots. Lots 1 and 2, are located at the northeast end of the Site. Lot 1 was and is undeveloped. Lot 2 was primarily used for the limited storage of manufacturing residue and was relatively undeveloped. Lots 1 and 2 received dredge materials in 1953 from the Port of Portland and U.S. Army Corps of Engineers' work in the Willamette River, and dredged materials may have been placed on the property from the navigation channel by others. Lots 1 and 2 are currently covered by a mixture of grasses, bare soil, and disturbed scrub-shrub vegetation. Historically, the majority of chemical processing occurred on Lots 3 and 4, the developed portion of the Site. Lots 3 and 4 are currently occupied only by an office building at the Site entrance on Front Street. The remainder of Lots 3 and 4 is covered with gravel and some concrete floor slabs that were left in place. Future use for Lots 1 and 2 and Lots 3 and 4 is anticipated to remain industrial.

With the exception of the docks in the Willamette River, the riverbank area (also referred to as Tract A) is currently vacant, but it received miscellaneous fill from various sources for many years. The majority of the riverbank is currently covered in rip rap, other slope-stabilizing materials, or heavy vegetation.

For the purposes of the HHRA, the Site was divided into potential exposure areas based upon historical use, potential future use, and current Site conditions. On this basis, three exposure areas were identified: Lots 1 and 2, Lots 3 and 4, and Tract A (riverbank area). Separate evaluations of potential human health risk were conducted for these three exposure areas.

In general, chemicals of interest (COIs) in environmental media at the Site are primarily associated with site manufacturing process residue, including dichlorodiphenyltrichloroethane (DDT) and its metabolites (dichlorodiphenyldichloroethane [DDD] and

[1] Voluntary Agreement for Remedial Investigation/Feasibility Study, DEQ No. ECVC-WMCVC-NWR-97-14 (Voluntary Agreement).

dichlorodiphenyldichloroethylene [DDE]), chlorobenzene, perchlorate, and chromium. COIs were identified separately for each exposure area. In addition, soil data were grouped into three depth horizons: 0-3 ft below ground surface (bgs), 0-15 ft bgs, and the entire unsaturated zone (designated as 0–Max). All chemicals that were detected at least once in the exposure area and soil horizon of interest were included as COIs. Maximum concentrations of COIs were compared to background concentrations and to risk-based concentrations (RBCs) to determine if they should be included as chemicals of potential concern (COPCs) for the HHRA. COIs that exceeded both background concentrations and RBCs were retained as COPCs. In addition, a COI was retained as a COPC if it was expected to contribute significantly to cumulative exposure. COPC screening for groundwater was conducted on a site-wide basis, rather than by exposure area.

Exposure media evaluated in this HHRA were limited to upland media only: surface and subsurface soil, indoor and outdoor air, and groundwater. No drinking water wells are located on or near the Site and areas of impacted groundwater are relatively deep; therefore, only indirect pathways of exposure were quantified for groundwater.

The Site is located in a heavily industrial area and is anticipated to remain industrial for the foreseeable future.[2] Therefore, the exposure assessment focused on industrial scenarios where individuals could come into contact with COPCs in surface and subsurface soils, groundwater, and air. A total of six types of potential human receptors were identified.

It was assumed that redevelopment of the Site could result in exposure of construction and excavation workers involved in the construction project. Duration of exposure for these receptors would be short (a year or less). Subsequent to redevelopment, it was assumed that there could be three potential exposure scenarios for industrial Site workers. Assuming that soil conditions remain the same, an indoor worker could be exposed to vapors migrating from soil and groundwater into an overlying building. An outdoor worker could be exposed to COPCs in surface soil via direct contact and to vapors migrating from soil and groundwater into outdoor air. However, if it is assumed that excavation activities during construction could result in movement of subsurface soil to the land surface, a future outdoor occupational worker could then become exposed to COPCs relocated from subsurface to surface soils during redevelopment. This hypothetical receptor is termed a "redevelopment worker" to differentiate this scenario from the outdoor worker scenario that assumes undisturbed soil conditions. The redevelopment worker was included at the request of DEQ. A trespasser scenario was also evaluated. Because of the difficulty accessing the site, potential trespassers were assumed to be no younger than 9 years of age and exposed over a period of ten years from age 9 to age 18.

Estimates of cancer risk and noncancer health hazards were calculated for both average (central tendency exposure [CTE]) and reasonable maximum exposure (RME). Mean concentrations in

[2] The site is zoned "IH" for heavy industrial use and lies within the Guild's Lake Industrial Sanctuary Plan whose purpose is to maintain and protect this area as dedicated to heavy and industrial uses (Integral 2006).

environmental media were used to estimate exposure point concentrations (EPCs) under average conditions. The 90 percent upper confidence limit (UCL) was used to estimate EPCs for both the CTE and RME conditions.

Intakes were calculated for each complete exposure pathway. For Lots 1 and 2, exposures were quantified for incidental ingestion of soil, inhalation of wind-blown dust, and dermal contact with soil. Based on the results of the COPC screening, it was determined that volatile COPCs are not present at concentrations that are expected to pose a risk to worker health for exposures via inhalation; therefore, inhalation of vapors in indoor and outdoor air were not quantified.

For Lots 3 and 4, exposure pathways quantified for outdoor receptors included incidental ingestion of soil, inhalation of wind-blown dust, and dermal contact with soil. Exposure pathways quantified for indoor workers included inhalation of vapors migrating from subsurface soil to indoor air and inhalation of vapors migrating from groundwater to indoor air.

For the riverbank area, complete exposure pathways included incidental ingestion of soil, inhalation of soil particulates, and dermal contact with soil. Based on the results of the COPC screening, inhalation of vapors in ambient air was not considered a complete exposure pathway for workers or trespassers. Also, it was assumed that buildings will not be constructed within Tract A because it is not practical to Site buildings directly on the riverbank. Therefore, vapor migration pathways to indoor air were not considered complete for this exposure area.

A toxicity assessment was conducted in accordance with United States Environmental Protection Agency (EPA) and DEQ guidance for each of the COPCs. For noncancer health effects, toxicity values identified included oral, dermal, and inhalation reference doses (RfDs) for both subchronic and chronic exposure periods. To assess cancer risk, cancer slope factors (CSFs) were identified for oral and dermal exposures, while unit risk factors (URFs) and CSFs were identified for inhalation exposures.

For health risks other than cancer, the ratio of the average daily intake to the RfD was calculated to obtain a hazard quotient (HQ). A HQ less than 1 indicates that no adverse health effects are expected. To evaluate the effect of exposure to multiple chemicals, HQs for each exposure pathway for individual chemicals were summed to obtain a hazard index (HI). HIs for multiple chemicals were summed even if the RfDs for the chemicals were based on effects on different target organs; despite that expectation that such effects would not be additive. A more rigorous approach to the HI based on target organs was not required given that HIs greater than 1 were dominated by a single chemical and the more refined approach would not change the risk conclusions.

A cancer risk estimate is an incremental probability that an individual will develop cancer during his or her lifetime due to exposure to a COPC under the assumed exposure conditions.

Cancer risks were calculated for each COPC by multiplying the average daily intake by the CSF. An individual cancer risk at or below 1×10^{-6} was the risk threshold used for screening COPCs. Because cancer risks are assumed to be additive, risks associated with simultaneous exposure to more than one carcinogen and more than one exposure pathway were combined to estimate the total cancer risk for a potential receptor.

For Lots 1 and 2, noncancer and cancer risk estimates were calculated for the outdoor occupational worker, redevelopment worker, construction worker, excavation worker, and trespasser. An indoor worker evaluation was not necessary as subsurface soil and groundwater underlying Lots 1 and 2 have not been shown to be impacted by historical operations that occurred on Lots 3 and 4. Noncancer HIs for all receptors were below a target hazard of 1.

Under average exposure conditions, potential cancer risk to an outdoor worker on Lots 1 and 2 exceeded an acceptable individual COPC risk limit of 1×10^{-6} only for arsenic. Under RME conditions, individual COPCs that exceeded a risk of 1×10^{-6} were limited to arsenic and 4,4'-DDT. Total cancer risk ranged from 2×10^{-6} under average exposure conditions for trespasser exposures to 2×10^{-5} under RME conditions for outdoor workers. The source of arsenic (the risk driver) at the Site is not related to historical site processes and the assumption of 100% bioavailability for arsenic used in the assessment is considered to result in a very conservative estimate of the likely risks.

Lead was retained as a COPC for Lots 1 and 2. Results of the adult lead methodology (ALM) indicate that lead in soil is not a concern for Lots 1 and 2 under assumed exposure conditions.

For Lots 3 and 4, noncancer and cancer risk estimates were calculated for the outdoor worker, redevelopment worker, construction worker, excavation worker, and indoor worker. HQs associated with 4,4'-DDT intake for an outdoor worker, redevelopment worker, and construction worker exceeded a target HQ of 1. HQs for all other chemicals and receptors were below 1.

For Lots 3 and 4, total potential cancer risk associated with direct soil contact was the greatest for outdoor and redevelopment workers. Total cancer risk for an outdoor worker ranged from 2×10^{-5} to 1×10^{-4} under average and RME conditions, respectively. The greatest contributor to total risk was ingestion of soil containing 4,4'-DDT. 4,4'-DDT was also the risk driver for an estimated total cancer risk of 2×10^{-5} for a construction worker and 7×10^{-7} for an excavation worker under RME conditions. Total cancer risk for an indoor worker exposed to vapors migrating from subsurface soil ranged from 2×10^{-6} to 8×10^{-6} under average and RME conditions, respectively. The total cancer risk was dominated by risk associated with inhalation of tetrachloroethene (PCE). PCE in the soil of Lots 3 and 4 was infrequently detected (i.e., 8% of the 77 samples) and the average PCE soil concentration used in the vapor modeling was dominated by an extreme value. Total cancer risk for an indoor worker exposed to vapors

migration from groundwater was below the COPC screening level of 1×10^{-6} for both the average and RME cases.

For the riverbank area, noncancer and cancer risk estimates were calculated for the outdoor worker, construction worker, and trespasser. Noncancer HQs for all COPCs and all receptors were well below a value of 1. Total HIs for potential cumulative effects were all below an acceptable HI of 1 for both the average and RME cases.

Total cancer risk for an outdoor worker ranged from 8×10^{-6} to 7×10^{-5} under average and RME conditions, respectively. These risks are primarily associated with ingestion of soil containing dioxins/furans. Total cancer risks for trespassers were also dominated by exposure to dioxins/furans, with values ranging from 6×10^{-6} to 1×10^{-5}. Total cancer risk for a construction worker under RME conditions was 1×10^{-5}.

Lead was retained as a COPC for the riverbank area. For outdoor workers and trespassers, predicted blood lead levels were well below target risk levels. Results of the ALM indicate that lead in soil is not a concern for receptors on the riverbank under assumed exposure conditions.

The results of the HHRA indicate that the most significant potential risks from site-related COPCs to receptors under anticipated exposure scenarios occur in Lots 3 and 4 due to exposure to DDT. However, the cancer risks for Lots 3 and 4 are within the 10^{-4} to 10^{-6} risk range USEPA (1990) established as a basis for remediation at Superfund sites. The noncancer risks are generally below with only a few slight exceedances of the hazard threshold of 1. Exposures to receptors at other portions of the Site are below the levels estimated for exposures at Lots 3 and 4. As described in the HHRA, the uncertainty inherent in the estimation of human health risk is substantial and the net effect is likely to be an overestimate of potential risks. Therefore, it is likely that actual risks to individuals that may access the Site are less than the estimated values presented in this HHRA.

1 INTRODUCTION

This report presents the results of the Arkema Upland Human Health Risk Assessment (HHRA) conducted by Integral Consulting Inc. (Integral) for Legacy Site Services (LSS). The HHRA is being performed in conjunction with the Arkema site remedial investigation (RI) completed by ERM (2005). This document summarizes the various steps in the HHRA, including development of a conceptual site model (CSM), selection of chemicals of potential concern (COPCs), exposure assessment, toxicity assessment, risk characterization, and uncertainty assessment. The remainder of this section summarizes key aspects of the site setting, including historical operations, sources of contamination, and previous environmental investigations. Subsequent sections will address the remaining steps in the HHRA.

1.1 SITE LOCATION AND ADJACENT PROPERTY USE

The Arkema, Inc. (Arkema) facility (the Site) is located along the southwest bank of the Willamette River between approximately River Mile (RM) 6.9 and 7.6 at 6400 NW Front Avenue in Portland, Oregon (Figure 1-1). The Site occupies approximately 54 acres and is generally flat with surface elevations between 25 and 38 ft (National Geodetic Vertical Datum 1929) (ERM 2005). A steep bank approximately 30-ft high borders the eastern side of the property, forming the bank of the Willamette River.

The Site is composed of 4 lots and one tract along the Willamette River. Lots 1 and 2, at the northeast end of the Site, were used for storage of manufacturing residue, and are a relatively undeveloped portion on the Site that is covered by a mixture of grasses, bare soil, and disturbed scrub-shrub vegetation. Lots 3 and 4 are the developed portion of the Site where the vast majority of chemical manufacturing and processing occurred. The riverbank is steeply sloping and covered with riprap, other slope-stabilizing materials, or heavy vegetation; a limited amount of vegetation grows among the bank armoring material.

The Site is bordered on the east by the Willamette River and to the south by CertainTeed [GS Roofing Product Manufacturing; Oregon Department of Environmental Quality (DEQ)'s Environmental Cleanup Site Information (ECSI) database site 117]. The Willbridge Bulk Fuel Area (ECSI 1549) and Kinder Morgan (ECSI 2104) sites are located immediately south of CertainTeed. Front Avenue borders the Site to the north and west. Four sites are located to the west of Front Avenue, upgradient of the Site. The sites include Starlink (Rhône-Poulenc [RP]; ECSI 155), Gould Industries, Doane Lake (ECSI 36), and Kinder Morgan (ECSI 2104). The Siltronics Inc. site (ECSI 183) is located immediately north of Front Avenue. Additional details on the adjacent properties can be found in DEQ's ECSI database[3].

[3] http://www.deq.state.or.us/lq/ecsi/ecsiquery.asp

Heavy industrial land use surrounds the Site, isolating it from the park and residential areas. The nearest residential structures are located approximately 0.3 miles west of the facility. Forest Park, a large forested public park, is located 0.5 miles to the west of the facility (ERM 2005).

1.2 SITE HISTORY

A summary of the histories of Site ownership and manufacturing are presented below. Further details can be found in the *Upland Remedial Investigation Report Lots 3 & 4 and Tract A – Revision 1* ("*Upland RI Report*", ERM 2005) and *Work Plan: Engineering Evaluation/Cost Analysis* (EE/CA) – *Arkema Removal Action – Portland, Oregon – Revised Draft* ("*Revised Draft EE/CA Work Plan*", Integral 2006).

1.2.1 Arkema Site Ownership History

The facility manufactured inorganic chemicals from 1941 to 2001. It was constructed and operated by Pennsylvania Salt Manufacturing, which later became known as Pennwalt Corporation (Pennwalt). Purchased by Societé Nationale Elf Aquitaine (ELF) in 1989, Pennwalt was combined with two other companies in 1990 to form Elf Atochem North America, Inc. In 2000, ELF merged with TOTALFINA to form TOTALFINA ELF, and Elf Atochem became ATOFINA Chemicals, Inc. (ATOFINA). In 2004, ATOFINA changed its name to Arkema, Inc. In April 2006, the management of Arkema, Inc.'s environmental work for the Portland property was transferred to LSS, the sole agent for Arkema.

1.2.2 Arkema Site Operational History

The Arkema Site operated as a chloro-alkali plant throughout most of its history from 1941 until 2001, when the entire facility was shut down due to escalating electricity costs. The facility used electrolytic cells to reduce concentrated sodium chloride brine to produce chlorine, caustic soda, hydrogen, hydrochloric acid, and sodium chlorate (CH2M Hill 1997).

Other key manufacturing processes that have occurred at the facility are listed below. The major industrial features of the Site and the riverbank features are briefly summarized in this section. The locations of the historical processes at the Site are presented in Figure 1-2. Unless otherwise indicated, the information presented below is summarized from the *Revised Draft EE/CA Work Plan* (Integral 2006).

1.2.2.1 Acid Plant Area

Dichlorodiphenyltrichloroethane (DDT), magnesium chloride hexahydrate, ammonium perchlorate, solid sodium hydroxide, a grass defoliant[4] (produced by chlorinating acetone with chlorine gas), sodium orthosilicate, and hydrochloric acid were manufactured in the Acid Plant Area (Figure 1-2). The dates of manufacture for each product are provided below:

- DDT (1947 to 1954)
- Magnesium chloride hexahydrate (1952 to 1962)
- Ammonium perchlorate (1958 to 1962)
- Solid sodium hydroxide (early 1950s to 1980s)
- Grass defoliant (early 1950s)
- Sodium orthosilicate (1950 to 1980)
- Hydrochloric acid (1966 to 2001).

1.2.2.2 Chlorate Plant Area

Sodium chlorate and potassium chlorate were manufactured in the chlorate plant area on Lot 4 (Figure 1-2). Sodium chlorate was produced by electrolysis of sodium chloride solution from 1941 to 2001. Potassium chlorate production (1941 to 1978) was similar to that of sodium chlorate, except that the salt source was potassium chloride rather than sodium chloride.

1.2.2.3 Chlorine Manufacturing Area

Chlorine manufacturing started in 1946 using cells equipped with graphite anodes in the old chlorine cell room on Lot 4 (LSS 2006, pers. comm.). These cells were operated until 1971, when they were shut down and replaced by a new chlorine cell room (1962 to 2001) located north of the old cell room on Lot 3 (Figure 1-2).

1.2.2.4 Salt Pads

Salt was the primary raw material used at the Site throughout its operational history (1941 to 2001). The salt was stored on asphalt-lined salt pads in the southeastern corner of the Site on Lot 4. The salt was dissolved in water while on the salt pads to produce brine for plant manufacturing operations.

[4] For only a few months in the early 1950s, Pennwalt attempted to produce a grass defoliant material for use in the agricultural industry. The operation consisted of chlorinating acetone with chlorine gas. Based on historical information, this material was never mass produced or sold.

1.2.2.5 Asbestos Ponds and Trenches

The asbestos diaphragm chlorine cells were rebuilt annually. Approximately 12 trenches on Lot 1 (Figure 1-2) were filled with asbestos-containing residue from the diaphragm chlorine cells. The ponds were decommissioned voluntarily to excavate the trenches containing asbestos residue. The asbestos removal work was conducted under a work plan approved by DEQ and under DEQ's oversight. The asbestos removal work was completed in 1992 (Elf Atochem 1999).

1.2.2.6 Former Brine Mud Pond

Calcium and magnesium were precipitated from salt in the form of calcium carbonate and magnesium hydroxide (referred to as dolomite/brine mud) in the later years of plant operation. This material was stored on soil in a diked area and in a pond located in the northern area of the property on Lots 1 and 2 (Figure 1-2) until it was excavated in the early 1990s (Elf Atochem 1999).

1.2.2.7 Old Caustic Tank Farm

The Old Caustic Tank Farm (OCTF) sometimes referred to as the Former Caustic Tank Farm (FCTF), is located just to the south of the Acid Plant Area on Lot 4 (Figure 1-2). Tanks within the OCTF were used to store sodium hydroxide from 1946 to 1996. The OCTF was in use until 1996. The idle tanks were removed from the OCTF during plant demolition activities in the spring of 2002 (ERM 2005).

1.2.2.8 Ammonia Plant

Ammonia was produced (mid-1950s to 1990) by combining nitrogen (stripped from air) with hydrogen that was produced in the chlor-alkali process. The combined gases were compressed and cooled to form anhydrous ammonia. The operation was located in what is known as the New Caustic Tank Farm Area on Lot 3 (Figure 1-2).

1.2.2.9 Transformer Pads

Electrical transformers were historically installed at various locations throughout the Arkema facility (see Figure 1-5 of the *Upland RI Report* [ERM 2005]). During facility demolition, all transformers were properly removed. The pads on which the transformers were located were tested for the presence of polychlorinated biphenyl (PCB) and managed or disposed of in accordance with DEQ oversight (McClincy 2003, pers. comm.) The scope and results of that investigation are summarized in the *Upland RI Report* (Appendix A) (ERM 2005).

1.2.2.10 Bonneville Power Administration Substation

The Bonneville Power Administration (BPA) owned and operated an electrical substation on the Site. The substation, which is divided into the main substation (also referred to as the Pennwalt

Substation) on Lot 3 and a substation annex to the north on Lot 2, occupied a total area of 1.28 acres of the facility (Figure 1-2). The substation was decommissioned following plant closure, and the associated equipment was removed from the property during the fall of 2002 (ERM 2005).

1.2.2.11 Riverbank

The riverbank area, mostly between Docks 1 and 2, received miscellaneous fill for many years. Parties responsible for the filling activities included the City of Portland, private excavation contractors, and Elf Atochem. Fill materials included clean soil, asphalt, concrete, metal piping, and miscellaneous materials from spent chlorine cells (ERM 2005). In addition, dredge spoils were deposited on the riverbank, which may have contained chemicals of interest. Details regarding known and potential dredge fill activities at the Site can be found in the *Revised Draft EE/CA Work Plan* (Section 2.2.4) (Integral 2006).

1.2.2.12 Stormwater Drain System

Many of the Site sewers have been in place since at least the mid-1950s and were designed to carry large volumes of cooling water. Many also were designed to drain building basements and process sumps, and are therefore rather deep (approximately 12 feet below ground surface [bgs] in certain locations).

The facility was issued a National Pollution Discharge Elimination System (NPDES) permit on January 28, 1993, which authorized the discharge of process wastewater, cooling water, and stormwater runoff. In January 2004, a new NPDES permit was issued to Arkema solely for the discharge of stormwater.

1.3 LAND USE AND BENEFICIAL WATER USE

Current and reasonably anticipated land uses, and current and reasonably likely future beneficial uses of groundwater in the locality of the facility (LOF) are described in detail in the *Upland RI Report* (ERM 2005). The following information summarizes the information reported by ERM (2005).

1.3.1 Current and Future Land Use

The current and reasonably likely future land use in the LOF is well defined. Chemical manufacturing operations at the facility ceased in 2001. Decommissioning and removal of the manufacturing infrastructure were completed in early 2005. The only structure remaining is the office building at the Site entrance on Front Street and certain concrete floor slabs left in place as environmental soil caps. Arkema maintains leases from the Oregon Department of State Lands

(DSL) for the docks in the Willamette River, but the docks are not currently in use. In-water and upland remedial activities to address environmental impacts are ongoing.

The facility is located within the Guild's Lake Industrial Sanctuary Plan (GLISP) (formerly the Northwest Portland Industrial Sanctuary), zoned and designated "IH" for heavy industrial use (ERM 2005). The purpose of the GLISP is to maintain and protect this land as a dedicated area for heavy and general industrial uses. The future use of the facility will be heavy industrial, consistent with the GLISP, Portland's Comprehensive Plan, and the City's Zoning Code.

1.3.2 Beneficial Water Use

As reported by ERM (2005), information regarding use of water potentially affected by former manufacturing operations had been collected as part of the Phase 2 Site Characterization (CH2M Hill 1997) and a beneficial water use survey conducted for a nearby facility (Woodward-Clyde 1997). Potential beneficial uses of nearby surface water (the Willamette River) include industrial use, recreational use, and ecological habitat in the LOF.

No drinking water wells are located on or near the LOF. Groundwater is not currently used nor is it reasonably likely to be used in the future as a drinking water source. A survey of wells within a 1-mile radius of the Site was conducted by CH2M Hill (CH2M Hill 1997). The 1997 CH2M Hill survey identified wells within the search radius of the Site, but concluded that there were no water supply wells identified downgradient of the Site.

An updated inventory of wells situated within a 1-mile radius of the facility was conducted for the RI. Nine new wells classified as water supply wells were identified within the 1-mile search radius. Of those nine wells, seven were wells installed at the Arkema facility as monitoring wells (i.e., they were previously incorrectly classified in the Oregon Water Resources Department database). The two remaining wells consisted of one well determined to be a monitoring well situated on Port of Portland property across the Willamette River and one well installed for cathodic protection purposes. No new water supply wells were identified within the search radius.

Because of the proximity of the Site to the Willamette River, future industrial water needs (e.g., noncontact cooling water) are likely to be met by surface water or by the City of Portland municipal water supply, and to a limited extent, the basalt aquifer. The beneficial use for groundwater in the LOF is expected to be recharge to the Willamette River (ERM 2005).

1.4 REGULATORY STATUS

The uplands portion of the Arkema facility evaluated in this risk assessment is located adjacent to the Study Area of the Portland Harbor Superfund Site, which encompasses about 9 miles of the Willamette River from approximately RM 2 to 11. The Study Area does not legally define

the Portland Harbor Superfund Site; the boundaries of the Site will be determined by U.S. Environmental Protection Agency (EPA) upon issuance of a Record of Decision (ROD; Integral and GSI 2004).

In 1995, Arkema (then known as Elf Atochem) submitted an intent to participate in DEQ's voluntary Cleanup Program. In 1996, Arkema entered into a voluntary letter agreement with DEQ that was followed in 1998 by a voluntary agreement with DEQ to complete a remedial investigation and feasibility study (RI/FS) of the former DDT manufacturing area. The RI/FS was later expanded to include other areas and chemicals at the Site (e.g., hexavalent chromium and perchlorate).

Arkema has completed several Interim Remedial Measures (IRMs) to address contaminated soil and groundwater. In 2000 and 2001, Arkema completed two phases of excavation and disposal to remove surface and subsurface soil with elevated DDT concentrations from the former manufacturing process residue pond and trench. Air sparging and soil vapor extraction (AS/SVE) techniques were employed to address residual chlorobenzene in groundwater in the upland portion of the Site. The system operated between December 2004 and June 2006. Calcium polysulfide was injected into groundwater in the Chlorate Plant Area to reduce hexavalent chromium (CrIV) to a trivalent chromium (CrIII) state and immobilize it in groundwater. The full-scale program implemented in June and November 2005 was monitored through April 2006 (Integral 2006). Bench-scale treatability studies of *in situ* treatments for perchlorate in groundwater were conducted between December 2003 and March 2006. Based on these studies, an active (groundwater and nutrient recirculation system) enhanced *in situ* bioremediation was previously recommended for the perchlorate plume (Integral 2006). In 2006, the *in situ* groundwater IRM work was halted and groundwater remediation efforts were redirected to the design and installation of a groundwater slurry wall and pump and treat system. The *ex situ* groundwater and treatment system was selected in lieu of *in situ* groundwater efforts in order to achieve EPA-directed source control efforts in accordance with the in-water EE/CA schedule. The groundwater IRM infrastructure and system will most likely be implemented in 2009.

An IRM to address stormwater is currently being designed for the Site. A feasibility study is being developed for probable implementation in 2008.

1.5 SUMMARY OF PREVIOUS INVESTIGATIONS

A number of sampling efforts have been conducted on the Arkema Site and on riverbanks adjacent to the Arkema site since 1996, as listed in Table 1-1. A summary of each investigation that focuses on the principal chemicals of interest (COIs) at the site (4,4'-DDT and its metabolites, chlorobenzene, perchlorate, chromium, and chloride) is presented in the *Upland RI*

Report (ERM 2005) and the *Revised Draft EE/CA Work Plan* (Section 3.1) (Integral 2006). Soil sample locations from previous investigations and monitoring wells are shown on Figure 1-3.

2 CHEMICAL PATHWAYS ANALYSIS

Based on the preliminary chemical screening presented in the RI (ERM 2005), COIs in environmental media at the Site are primarily associated with Site manufacturing process residue, including DDT and its metabolites dichlorodiphenyldichloroethane (DDD) and dichlorodiphenyldichloroethylene (DDE), chlorobenzene, perchlorate, and chromium. The following section presents information describing various sources (primary and secondary) of these chemicals in environmental media and chemical migration pathways (Integral 2006; ERM 2005). Refer to Figure 2-1 for a preliminary CSM depicting COI sources, migration pathways, and impacted media.

2.1 PRIMARY SOURCES AND RELEASE MECHANISMS

The following list of the primary sources of COIs from the Arkema Site was summarized from the draft Arkema in-water removal action *Revised Draft EE/CA Work Plan* (Integral 2006). Because the Site has undergone extensive remediation and demolition to date, including interim remedial source control measures, many of these areas may no longer function as sources of COIs.

2.1.1 Acid Plant Area

2.1.1.1 Former MPR Waste Pond

A shallow unlined pond was constructed in 1948 northeast of the manufacturing building to receive manufacturing process residue (MPR) and was used until about 1954 when DDT manufacturing operations ceased. Major portions of the former MPR pond soils were removed during a soil IRM in 2000.

2.1.1.2 Former MPR Waste Trench

In 1951 or 1952, a trench 8-ft wide by 285-ft long was constructed north of the MPR pond to increase its capacity. Use of the MPR trench ceased in 1954 when DDT operations ended. Elevated concentrations of monochlorobenzene (MCB) and DDT were found in this area prior to soil removal. A two-phased soil removal and source control interim remedial measure was implemented in 2000 and 2001. Impacted soil was removed in portions of the Acid Plant Area to depths of up to 12 ft bgs.

2.1.1.3 Former DDT Process Building and MCB Recovery Unit

The pesticide DDT was manufactured in the former DDT process building from 1947 to 1954. Chemical base stocks used in the DDT manufacturing process included MCB, chloral, and

sulfuric acid. Some DDT handling took place in Warehouse No. 2, in the northwest corner of the Acid Plant Area. From 1958 to 1962, after DDT manufacturing ceased ammonium perchlorate operations were conducted in the former DDT process building. During this period, sodium perchlorate was produced inside the chlorate cell room. Sodium perchlorate was transferred to the Acid Plant Area where it was converted to ammonium perchlorate by using ammonium chloride to form a solid propellant for guided missiles. The production of sodium perchlorate and ammonium perchlorate ceased in 1962.

2.1.1.4 Lot No. 1 Former DDT Trench

Historical construction activities in the Acid Plant Area generated soils with DDT residues that were reportedly disposed of in a defined trench on Lot No. 1 of the Site. In 1994, Arkema discovered and excavated the trench on Lot 1 that contained DDT MPR. Confirmation samples indicated that low concentrations of DDT, DDD, and DDE (below DEQ industrial soil cleanup levels) are present in shallow soil in a discrete area between 3 and 14 ft bgs around the perimeter of the former trench. None of the 33 confirmation samples contained constituent pesticides greater than EPA Region 9 Preliminary Remediation Goal (PRG) for industrial soil. Arkema handled and disposed of the MPR from the former DDT trench in accordance with a DEQ-approved soil management plan to ensure proper management of these soils.

2.1.1.5 Riverbank Soil

Fill was placed in the Acid Plant Area bordering the Willamette River after DDT manufacturing ceased. It appears that the bank adjacent to the Acid Plant Area has been filled out toward the Willamette River approximately 200 ft since the 1950s (CH2M Hill 1997). Fill thickness ranges from a few feet in the former DDT manufacturing area to approximately 25 ft along the riverbank (Integral and GSI 2005). DDT residuals have been measured in some portions of the fill and are being addressed as part of the ongoing IRMs for the upland portion of the Arkema Site.

2.1.2 Chlorate Plant Area

2.1.2.1 Sodium Chlorate Manufacturing

Sodium chlorate manufacturing started in the Chlorate Plant Area in 1941. Chlorate solutions were shipped by truck or barge. Trucks were loaded on the southern side of the Chlorate Plant Area. Barges were loaded at Dock 2. The production of sodium perchlorate in the chlorate plant is the source of the main perchlorate plume (ERM 2005).

2.1.3 Historical Spills and Releases

2.1.3.1 Historical Spills

Chemical spills when the plant was operational included a 1,200-gallon ammonium hydroxide leak to the sewer system in 1986, a sodium chlorate release of unknown quantity to the sewer system in 1987, and other smaller spills (Integral and GSI 2005).

2.1.3.2 Ammonia Plant

On July 16, 1987, Pennwalt experienced a spill of 30 percent aqueous ammonia. It was estimated that approximately 400 gallons of solution containing approximately 896 pounds of ammonia spilled onto the ground. It is likely, based on the atmospheric conditions at the time (75 degrees Fahrenheit), that most of the ammonia would have volatilized rapidly.

2.1.3.3 Former Storm Sewer Outfall

In 1947, at the initiation of DDT manufacturing, MPR was discharged to floor drains connected to a storm sewer that drained into the Willamette River. The suspected outfall location that received the MPR is located between Docks 1 and 2.

2.1.3.4 Former Caustic Tank Farm

There have been two documented releases of sodium hydroxide in the FCTF, in 1993 and 1996. In each case, approximately 200 gallons of caustic (sodium hydroxide) were spilled to the floor of the tank farm. The caustic was collected in the sumps and pumped to the wastewater treatment system for neutralization. The idle tanks were removed from the FCTF during the demolition activities in the spring of 2002. During Site demolition activities, soil was collected from seven different locations from the base of the tank and submitted as a composite sample for laboratory analysis of total petroleum hydrocarbons (TPH) and PCBs. Petroleum hydrocarbons were detected in the sample, but no PCBs were detected.

Other potential source areas include the salt pads (chloride) and transformer pads (PCBs) formerly located on the Arkema property and former BPA substation.

2.2 SECONDARY AND NON-SITE RELATED SOURCES

Information regarding the possible sources of nonsite-related chemicals is provided where available. COI-containing matrices being evaluated for the upland HHRA include surface and subsurface soil, riverbank soil, and groundwater. Unless otherwise indicated, the information provided below is summarized from *Revised Draft EE/CA Work Plan* (Integral 2006).

Various organic and inorganic compounds were detected in the Site media despite limited or no use of these compounds historically. Metals, including aluminum, cadmium, copper, lead, silver, and zinc were detected in Site soils. There is no indication that these metals were ever used as raw materials or process ingredients during plant operations. Aluminum and copper were components of electrical distribution devices located inside various process buildings at the Site, such as the electrolytic cell rooms. Small quantities of lead may have been used in some of the older chlorine cell components. Some of these decommissioned chlorine cells containing lead may have been used as fill along the riverbank between Docks 1 and 2. Small quantities of silver may have been used for reagent purposes in laboratory chemical analyses conducted at the Site. Zinc was used as a biocide to inhibit biological growth in the cooling towers at the Chlorate Plant and the Chlorine Plant. These noncontact cooling tower waters were discharged by permit through industrial sewers to the river. Similarly, halogenated compounds including hexachlorobenzene and hexachlorobutadiene were detected at the Site despite no indicates that they were ever used as a raw material or process ingredient at the Arkema facility.

Polychlorinated dibenzo-*p*-dioxin P/dibenzofurans (PCDD/Fs) were never used as a raw material or process ingredient; however, PCDD/Fs may be associated with chlorine production from some cells equipped with graphite anodes. In 1946, the plant began using graphite anodes during chlorine production. Cells were dismantled and washed approximately once per year and the associated wash water was discharged to the river. By 1971 all of the graphite anodes were replaced with titanium anodes. A letter from LSS to DEQ (dated May 18, 2006) addresses the potential generation of PCDD/Fs during production processes at the Site (LSS 2006, pers. comm.). Investigations of the chloro-alkali process has indicated that polychlorinated dibenzofurans (PCDFs) are the predominate PCDD/Fs produced in the process and polychlorinated dibenzo-p-dioxins (PCDDs) were below detection limits (Rappe et.al. 1991). PCDD/Fs are ubiquitous in urban environments in large measure from atmospheric dispersion and subsequent deposition of emissions from combustion sources including industrial furnaces, forest fires, and automobile exhausts. Within the vicinity of the Site there are numerous dioxin emission sources that could impact Site media though transport via groundwater migration, air deposition, and surface water runoff. A rigorous dioxin fingerprint analysis to determine the source of the PCDD/Fs detected at this Site was not conducted as part of this risk assessment. Furthermore, the PCDD/Fs detected at the Site were evaluated as a whole using toxic equivalency factors (TEF) that relate the toxicity of each PCDD/F detected to the most toxic PCDD/F (i.e., the dioxin congener 2,3,7,8-tetrachlorodibenzo-*p*-dioxin [TCDD]). The Uncertainty Section of this risk assessment does discuss the distribution of PCDD/Fs found at the Site and the portion of the PCDD/F risk that may be related to transport of PCDD/Fs from nearby sources.

Polycyclic aromatic hydrocarbons (PAHs) are detected at the Site. PAHs were never used as a raw material or process ingredient during plant operations. However, fuels containing PAHs were likely used on Site, though there are no records of spills to the ground surface. In

addition, asphalt paving, a PAH-containing material, was a common component of riverbank fill. Private contractors and the City of Portland transported the material to the Site during road maintenance projects. Other potential sources of PAHs include atmospheric deposition from offsite locations, road runoff, and the transport and deposition of upstream particulate.

Electrical transformers were installed at various locations throughout the Site over the operating history. The PCB-containing transformers were properly removed during facility demolition activities at the Site. The concrete pads supporting these transformers were tested for the presence of PCBs and managed or disposed of in accordance with regulatory guidance and with DEQ oversight (ERM 2005). The BPA also operated an electrical substation on the property for a number of years. The substation was properly decommissioned and all associated equipment removed following the cessation of manufacturing activities at the Site and subsequent plant closure (ERM 2005).

Pesticides including aldrin, dieldrin, endrin, beta-endosulfan, gamma-hexachlorohexane, and heptachlor epoxide were not manufactured at the Site and none were used as a raw material or process ingredient. Many of these pesticides were handled by the upgradient RP facility. DDT was the sole pesticide that was manufactured and handled at the Arkema facility.

Phenols were not used at the Site. Phenolic COIs include phenol, 2-chlorophenol, and pentachlorophenol (PCP). At least some utility poles used onsite were impregnated with wood preservatives such as PCP.

Volatile organic compounds (VOCs) that represent COIs at the Site include carbon disulfide, carbon tetrachloride, chloroform, methylene chloride, trichloroethane, tetrachloroethene (PCE), toluene, m,p-xylene, and chlorobenzene. Carbon disulfide, carbon tetrachloride, chloroform, trichloroethene (also referred to as "trichloroethylene"; TCE), and PCE were not used at the Site. Carbon disulfide may have been a constituent of the asphalt used as fill along the riverbank. Chloroform may have been produced from alkaline reactions with chloral. Methylene chloride was used in the auto shop (near the Front Office) and in the ammonia plant for parts cleaning and as a degreaser, respectively. It appears that methylene chloride was seldom used at the plant, and the auto shop dip tank contents were manifested off the Site shortly after 1990.

2.3 MIGRATION PATHWAYS AND ENVIRONMENTAL MEDIA OF CONCERN

COIs present in groundwater, surface soil, and subsurface soil are of concern for potential human contact during current and future use of the Site and are further evaluated in this HHRA. Human health risks associated with COIs in sediment and surface water currently are being addressed by the *Revised Draft EE/CA Work Plan* (Integral 2006) and the *Portland Harbor In-Water Human Health Risk Assessment* (Kennedy and Jenks 2004) and are not included in this

assessment. A description of COI migration pathways in relevant upland media is provided in this section.

2.3.1 Surface and Subsurface Soil

COIs released directly to surface and subsurface soil via drains, spills, leaks, and/or COIs leaching to subsurface soils may represent a potential exposure source for current and future hypothetical receptors. As depicted in Figure 2-1, volatile COIs in subsurface soil may migrate upward, volatilizing to ambient air, or when a building is present, vapors migrating upward may infiltrate cracks in foundations and infiltrate indoor air. Alternatively, COIs may percolate downward through surface and subsurface soil with rainfall. COIs migrating to subsurface soil then may leach to groundwater.

With the exception of some erosion of bank soils, little overland or airborne transport of chemicals in surface soil is expected via wind and water erosion. The northern third of the property (Lots 1 and 2) were formerly developed to receive process related waste materials and graded dredge spoils, but are now idle industrial areas that have been passively colonized by a mixture of native and introduced plants species. On the northeastern edge of Lots 1 and 2 is a stand of black cottonwood trees, a willow thicket, and an area overgrown with scrub-shrub vegetation. Trees and scrub-shrub vegetation also exist on the western end of Lot 1. The remainder of the property (Lots 1 and 2) is vegetated with grass or is bare. The southern two-thirds of the property (Lots 3 and 4) where chemical manufacturing activities took place, is largely covered by pavement, gravel, or a temporary cover system. Overland runoff to the river is not expected.

The riverbank in the vicinity of Arkema, Tract A, is partially river beach and steep slopes covered with bank stabilization material that includes large chunks of concrete, asphalt, and other impervious material (Integral 2004). There is no evidence of large-scale bank erosion, although there was minor sloughing of the bank between Docks 1 and 2 during a flood in 1996 (Integral 2004).

2.3.2 Groundwater

Groundwater occurs in six distinct groundwater zones beneath the Site including the Shallow Zone, Shallow-Intermediate Silt Zone, Intermediate Zone, Deep Zone, Gravel Zone, and Fractured Basalt (ERM 2007). In general, the depth to groundwater increases from west to east across the Site (from Front Avenue toward the Willamette River). Shallow, unconfined groundwater occurs from 5 to 25 ft bgs in the uppermost fill and sand alluvium. The shallow-Intermediate Silt Zone, comprised of silts, sandy silts, and clays is approximately 1- to 4-ft thick across the Site. The thickness of this zone increases to the west of the Site where it reaches up to 45 ft in the former Doane Lake Area. The Intermediate Zone, consisting of alluvial sands is confined or semi-confined occurring between depths of 36-46 ft bgs. The saturated thickness of

the Intermediate Zone is 5- to 10-ft across the Site. The Deep Zone is characterized by finer-grained deposits; predominantly silt with some clay and fine sand at depths of 40-60 ft bgs. The underlying basalt bedrock controls the depth and saturated thickness of the Deep Zone (up to approximately 60 ft). The Gravel Zone (alluvial gravel), a formation of high hydraulic conductivity is present in some portions of the Site between the Deep Zone and the basalt bedrock. This zone is typically 10 ft thick and with increasing thickness towards the Willamette River. The Fractured Bedrock sits below the alluvial deposits up to 216 bgs, which is the maximum depth explored. The fractured and weathered composition of this zone has relatively low permeability as compared to the overlying Gravel Zone (ERM 2007).

Dissolved groundwater plumes at the Site are relatively stable and well-documented under Lots 3 and 4 (Integral and GSI 2005). A conceptual interpretation of the current approximate configuration of groundwater plumes originating from the upland portion of Lots 3 and 4 and entering into river sediments is provided by Integral (2006) in a draft work plan for in-water removal action. Residual dense, nonaqueous-phase liquid (DNAPL) has been observed in groundwater underlying the Acid Plant Area.

In 2005, low-flow groundwater sampling was conducted for Lot 2 at the RP-2 well cluster along the eastern portion of the Site (ERM 2006). Depth to groundwater was consistent among the wells at approximately 30 ft bgs. Pesticides, perchlorate, and arsenic were not detected in groundwater samples collected from the RP-02 well cluster, although chloride was present at concentrations up to 16,500 mg/L.

A Site-wide groundwater sample collection event was conducted in April of 2007. This event sampled a total of 92 monitoring wells at the Site, including 3 well clusters on Lots 1 and 2 that had not been sampled previously (i.e., RP-08, RP-09 and RP-10). The validated data and related sampling report (ERM 2007) were not available in sufficient time to be included in the quantitative risk assessment presented herein. However, the results from the April 2007 event are evaluated in light of the risks predicted in this assessment based on earlier monitoring data as a means to judge the uncertainty and potential conservatism that results from not using the more current and spatially complete groundwater sampling results.

COI plumes underlying Lots 3 and 4 may migrate through the subsurface environment, including transport across the Site where they may be discharged to the Willamette River in nearshore sediment. Vapors from volatile COIs in groundwater may migrate to outdoor air. Although buildings are not currently present along the eastern portion of Lots 3 and 4 where the plumes are found, volatile COIs may infiltrate the indoor air if buildings are constructed in the future.

2.3.3 Stormwater

Comprehensive stormwater monitoring was conducted monthly in 2004 and 2005 as a requirement for the renewal of an NPDES permit for the facility (ERM 2005). In addition, as part of the stormwater IRM, four rounds of stormwater samples were collected in 2007 following the removal of catch basin sediments and implementation of best management practices (Integral 2007). Low levels of DDT and metabolites, CrVI, and perchlorate were detected in the stormwater samples. Low levels of DDT and metabolites were detected in samples collected from every outfall. The majority of the higher concentrations of CrVI were detected in samples collected from Outfall 004, and the highest perchlorate concentrations were detected in samples collected from Outfalls 001 and 002 (Figure 1-2).

Stormwater runoff from Lots 1 and 2 flows to a fully enclosed conveyance system that discharges directly to the Willamette River. Discharged effluents currently are managed under a NPDES permit and will not be evaluated further in the HHRA.

2.3.4 Sediment

DDT was observed in river sediment adjacent to the property are present primarily because of historical releases from a former process discharge pipe located just north of Dock 1 and groundwater discharges to the sediment. Erosion of sediment from one location to another and bank soils may be secondary sources of other COIs to other portions of the river. COIs may be taken up from sediment by benthic and aquatic biota. However, COIs in sediment and biota are not evaluated further in this HHRA, as they are being addressed by an in-water removal action for the Arkema Site (Integral 2006) and an in-water risk assessment for the lower Willamette River (Kennedy and Jenks 2004).

2.3.5 Surface Water

COIs present in surface water at the Site are a combination of COIs already present in the river water as well as COIs that are in sediment partitioning to surface water. Samples collected from transition zone water adjacent to the Site were found to contain DDT and chloride (Integral 2006). Some transition zone water samples were collected using a Geoprobe® or similar method, which can generate turbid water samples. Reported pesticide and metal concentrations using these sampling methods may not be representative of actual dissolved groundwater concentrations.

COIs in surface water may migrate with currents in the Willamette River and may partition to aquatic biota. However, impacts to surface water and aquatic biota currently are being evaluated by an in-water removal action for the Arkema Site (Integral 2006), and an in-water risk assessment (Kennedy and Jenks 2004) for the lower Willamette River and will not be evaluated further under this HHRA.

2.4 STATUS OF SOURCE MEDIA – SOURCE CONTROL

Since 1989, Arkema has conducted several source control measures to improve soil, stormwater, groundwater, and sediment quality at the Site. Several groundwater source control measures are ongoing and other source control measures are presently under development.
A chronological summary of source control measures that have been completed or that are ongoing at the Site is provided below.

2.4.1.1 Brine Residue Pile and Pond

The brine residue pile and pond were located on Lots 1 and 2. Brine residue, which was composed of calcium carbonate and magnesium hydroxide, were historically disposed of in either the brine residue pile or pond. The brine residue pile was completely removed from the site in February 1989, and the pond was completely removed in August 1992 (Integral and GSI 2005). The material was transported to Hillsboro Landfill and beneficially used as a soil amendment to the final landfill cap.

2.4.1.2 DDT Removal

In 1994, a trench on Lot 1 that contained DDT residues was completely excavated, disposed of, and backfilled with clean material. Approximately, 1,700 tons of soil with DDT residues were excavated and disposed of at the Waste Management Subtitle C landfill in Arlington, Oregon.

2.4.1.3 Phase I and II Soil Removal IRMs

Soil removals were carried out in 2000 and 2001 to address soil containing high concentrations of DDT and chlorobenzene in the Acid Plant Area. The Phase I soil removal IRM was performed at the Site between September and November 2000. During the Phase I removal, approximately 3,800 tons of soil was excavated from the former MPR pond and trench. These soils were disposed of at the Waste Management Subtitle C landfill in Arlington, Oregon. Additionally, a temporary surface cover was constructed in the unpaved area east of the Acid Plant Area, where unpaved surface soil samples had been collected. The Phase II soil removal IRM was completed in November 2001 in the Acid Plant Area. A total of 915 tons of shallow soil were removed from throughout the Acid Plant Area and disposed of at an Arlington, Oregon landfill.

2.4.1.4 Soil Vapor Extraction System

A soil vapor extraction system was installed in December 2000 to extract chlorobenzene mass from subsurface soils. The system was expanded over 2.5 years of operation and included five horizontal extraction wells. This system was shut down in 2003.

2.4.1.5 Stormwater System Improvements

Phase I efforts included the cleanout of stormwater catch basins and subsurface lines, emplacement of filter bags in catch basins, and the removal of some surface soil and placement of temporary cap over a large, unpaved area (ERM 2005). Upon completion of the Phase I and Phase II removal activities, asphalt was placed over the area to direct stormwater directly to surface drains. A temporary impermeable cover was also placed on a fill area on the eastern boundary of the Acid Plant Area to divert stormwater runoff directly to surface drains.

Stormwater IRM fieldwork was conducted in three stages between October 16, 2006 and June 5, 2007 and included best management practices (BMPs) and catch basin sediment and stormwater sampling (Integral 2007). Catch basin and filter sock sediment sampling was conducted from October 16 to 20, 2006. Following catch basin sampling, additional BMPs were implemented including: removal of sediment from each catch basin, rinsing with potable water, and pumping the water out of the catch basin with a sump pump. These clean-out activities were conducted between October 15 and 24, 2006. After catch basin cleaning, additional BMPs were implemented including installing filter socks in the active catch basins and placing biobags on or adjacent to the catch basin grates (October 16 to November 1, 2006). In addition, a number of catch basin pipes were decommissioned (sealed with concrete or expandable plugs) as they were determined to be non-integral to the stormwater system (Integral 2007).

On December 19, 2006, a vacuum truck was utilized to clean the deep catch basins and vaults connected to outfalls 001, 003, and 004 (i.e., Pit 16 and catch basins 1-11, 1-19, 3-2, 3-3, and CB-003-01. In addition, catch basin 1-11 was sealed with concrete (Integral 2007).

An IRM Focused Feasibility Study (FFS) is currently being developed for Site stormwater for probable implementation in 2008.

2.4.1.6 Groundwater Cleanup/Source Control

Pilot studies were conducted for Site groundwater in order to develop remedial strategies for cleanup and source control. AS/SVE techniques were employed to address residual chlorobenzene in groundwater in the upland portion of the Site. The system operated between December 2004 and June 2006. Calcium polysulfide was injected into groundwater in the Chlorate Plant Area to reduce CrIV to a trivalent state and immobilize it in groundwater. The full-scale program implemented in June and November 2005 was monitored through April 2006 (Integral 2006). Bench-scale treatability studies of *in situ* treatments for perchlorate in groundwater were conducted between December 2003 and March 2006. Based on these studies, an active (groundwater and nutrient recirculation system) enhanced *in situ* bioremediation was recommended for the perchlorate plume (Integral 2006). In 2006, the *in situ* groundwater IRM work was halted and groundwater remediation efforts were redirected to the design and installation of a hydraulic control-based approach utilizing a slurry wall and pump and treat

system. The *ex situ* groundwater and treatment system was selected in lieu of *in situ* groundwater efforts in order to achieve EPA-directed source control efforts in accordance with the in-water EE/CA schedule. The groundwater IRM infrastructure and system will most likely be implemented in 2009

Based on the chemical transport pathways and source control measures implemented at the Site, media potentially impacted by former operations can be identified. These media are shown in Figure 2-1.

Identification of impacted media is important in assessing potential exposure pathways and contact media for people at the Arkema Site. Within Lots 1 and 2, Lots 3 and 4, and the riverbank area (Tract A), chemicals may be present in surface and subsurface soil, groundwater, outdoor air, indoor air of buildings constructed in the future, windblown dust, surface water and sediment, and terrestrial and aquatic biota. Exposure media are discussed in more detail in the following section as they relate to specific receptors and their respective exposure routes.

Areas where limited or no manufacturing activities occurred have not been characterized to the same extent as former process areas. Analytical data for process areas may be used to represent media concentrations for other parts of the Site that are less fully characterized although for these other parts of the Site the process areas are likely overestimates and represent "worst case" media concentrations. With respect to groundwater exposures, this potential bias will be evaluated by comparing historical concentrations used in this risk assessment with the more recent and spatially complete monitoring data from the April 2007 sampling event.

2.5 POTENTIAL EXPOSURE MEDIA, EXPOSURE ROUTES, AND RECEPTORS

Depending on the nature of their activities, a variety of people may come into contact with COIs in one or more of the environmental media described above. Groups of people who may contact COIs in Site media are called receptors. Possible receptors present on the Arkema Site and potential routes of exposure are discussed in this section and are depicted on Figure 2-1. Environmental media that contain COIs and are potentially contacted by receptors are referred to as exposure media.

As mentioned previously, surface water, sediment, and aquatic biota are not evaluated under this HHRA; therefore, people who may contact these media, such as recreational river users, divers, recreational and subsistence fishers, dock workers, and others are not presented in Figure 2-1 as possible receptors. A detailed evaluation of exposure pathways for these and other receptors who may contact aquatic media is provided by Kennedy and Jenks (2004).

Stormwater system workers may contact COIs present in stormwater while collecting compliance monitoring samples. However, stormwater workers are not included as a receptor

of concern in this HHRA because the stormwater system currently is managed under a NPDES permit. Samples are expected to be collected by trained professionals who are working under an approved health and safety plan that would require the use of protective clothing to prevent exposures.

Exposure media to be evaluated for this assessment are limited to upland media only, surface and subsurface soil, indoor and outdoor air, and groundwater.

2.5.1 Surface and Subsurface Soil

Exposure routes of primary concern include incidental ingestion and direct contact with surface and subsurface soil. Direct exposures may occur through soil contact with skin, while outdoor workers are engaged in activities such as construction, property maintenance, and excavation. Soil that adheres to skin also may be ingested through hand-to-mouth contact, such as what occurs when eating or smoking. Consistent with DEQ guidance (2000, 2003), it is assumed that outdoor workers may contact COIs in surface soil to a depth of 3 ft bgs. An additional outdoor worker exposure with soils up to 15 ft bgs, identified as the redevelopment scenario, has also been conducted in response to a request from DEQ. The maximum depth to which construction and excavation workers are likely to contact COIs is assumed to be 15 ft bgs.

Currently, most of the riverbank is covered in riprap, heavy vegetation, or other slope stabilizing materials and so only limited contact with COIs in riverbank soil may occur. If trespassers gain access to the riverbank, they may contact COIs where ground cover is not present. In this case, direct exposure to surface soil via skin contact and incidental ingestion are potentially complete exposure pathways for trespassers.

Volatile chemicals in soil may be released to outdoor ambient air or infiltrate indoor air. Additional discussion of the inhalation pathways for indoor and outdoor air is presented in Section 2.5.3

In addition, some fraction of the site-related chemicals may adhere to soil particles and be released as fugitive dust by wind erosion and vehicular traffic. These particles then may be inhaled by people working outdoors or trespassers. Wind-blown dust is negligible throughout the Site due to groundcover (e.g., gravel, grass, and pavement).

2.5.2 Groundwater

The area with the highest concentrations of COIs in groundwater are found in Lots 3 and 4 where depth to shallow groundwater is greatest. The groundwater containing elevated concentrations of COIs is found at depths greater than 15 ft bgs, the maximum depth to which excavators are expected to work. Conversely, groundwater along the western portion of the Site is found at depths as shallow as 8 ft bgs; however, detected concentrations of COIs in

groundwater in this area are orders of magnitude below those in other areas of the Site. COIs found on this portion of the Site are primarily from the adjacent RP facility. Arkema expects RP to conduct additional characterization of RP-contaminated groundwater flow onto the Site and apparent infiltration in the Arkema storm line. Although there is the potential for workers to have direct contact with groundwater that seeps into excavations, it is not expected to be an exposure pathway of concern.

Only limited contact with groundwater by excavation workers (between 8 and 15 ft bgs) is expected to occur at the Site where groundwater and subsurface soil remediation is taking place to reduce COI concentrations. Therefore, direct contact of groundwater by excavation workers will not be evaluated quantitatively in this HHRA. It is anticipated that future excavation workers will work under a health and safety plan that will require donning appropriate personal protective equipment and implementation of administrative and/or engineering controls to prevent exposure.

Vapor inhalation exposures resulting from volatile chemicals in groundwater are discussed in Section 2.5.3.

2.5.3 Indoor and Outdoor Air

Volatile chemicals present in soil and groundwater may migrate to outdoor air and may be inhaled by workers and trespassers. Vapors migrating to indoor air spaces also may be inhaled by workers; however, no buildings or structures are present in areas affected by COIs. In addition, excavation workers constructing or otherwise working in a trench onsite may inhale volatile COIs in trench air. The potential for vapor intrusion or volatilization to air is dependent on soil type and grain size, organic content of the soil, depth to and thickness of the shallow aquifer, soil air- and water-filled porosity, Henry's constant of the chemical, and other physical and chemical parameters.

COIs present in these exposure media are discussed further in Section 3.

3 DATA EVALUATION

Data were evaluated according to *Guidance for Data Usability for Risk Assessment* (USEPA 1992a), which provides minimum data requirements to assure that data will be appropriate for use in risk assessment. The guidance addresses five main issues pertinent to assessing data quality for risk assessment:

- Data sources. Evaluate the type of data collected (e.g., screening data, fixed laboratory data) and whether quality assurance/quality control samples are available for the data to provide data quality information.
- Analytical methods and detection limits. Evaluate methods for appropriateness and sensitivity and determine if detection limits are low enough for risk-based screening.
- Data quality indicators. Review laboratory validation reports for data quality issues.
- Background samples. Assess whether appropriate quantity and location of background samples were collected.
- Consistency of data collection methods. Evaluate sample collection methods for appropriateness for the chemical, media, and analysis; review field trip notes to assess quality of sample collection; and determine if differences in sample collection exist between different sampling events and investigations.

3.1 DATA SOURCES

Arkema Site soil chemistry data were collected from April 1994 through March 2005. Data collected to support the upland remedial investigation of Lots 3 and 4 and Tract A (i.e., riverbank) from September 1998 to March 2005 (ERM 2005) were selected for use in the quantitative portion of the risk assessment. Additional sampling data collected in March 2005 from Lots 1 and 2 (ERM 2006) were also included in the quantitative risk evaluation. These data are maintained by the Lower Willamette Group in the Round 1 Main Database, and samples collected prior to March 2005 are summarized in the remedial investigation report (ERM 2005). At the request of DEQ, a supplemental sampling investigation was conducted along the riverbank. The sampling methodology, locations, and analytical procedures for the supplemental riverbank sampling were approved by DEQ. The supplemental riverbank sampling was conducted in March of 2007, and the data were included in the quantitative risk evaluation. Data collected prior to the remedial investigation (pre-1998) were not included in the risk assessment dataset because the data quality objectives did not conform to the Quality Assurance Project Plan that was subsequently developed for the RI (Exponent 1998). Differences in sample collection, analytical methods, and data validation for data collected prior to 1998 could lead to uncertainty in interpretation of analytical results.

As discussed in the *Upland RI Report* (ERM 2005), the majority of soil investigation samples have been collected from Lots 3 and 4 because the historical operations concentrated on those two parcels. IRMs from 2000 to 2002 on Lots 3 and 4 have resulted in the removal of impacted soils from the Acid Plant and Chlorate Plant Areas. Samples collected from areas where soil was subsequently removed during an IRM were removed from the risk assessment dataset[5] (Appendix A).

Groundwater samples were collected between 1994 and 2005 during various Site investigation activities. Groundwater monitoring conducted for the remedial investigation began in January 1999 and continued through 2005. However, only the most recent groundwater monitoring data are considered representative of current Site conditions due to ongoing remedial actions. Higher quality, low-flow sample collection techniques were implemented after June 2001. For the purpose of quantitative risk evaluation for groundwater exposures, only validated data collected from June 2001 through 2005 were included in the risk assessment dataset. In April 2007, a Site-wide groundwater sampling event was conducted for a total of 92 wells, including 3 well clusters on Lots 1 and 2 that had not been previously sampled. The validated groundwater results from this sampling event were not available in time to be included in the quantitative risk evaluation. However, the results from the 2007 groundwater data have been compared to the data used in the quantitative risk evaluation to examine any potential bias and uncertainty that may be associated with the older and more spatially limited groundwater dataset. The analysis is presented in the Uncertainty Section of this report. Field screening and other unvalidated data were not considered in this risk assessment.

3.2 DATA TREATMENT

Data validation and other quality assurance and quality control practices are outlined in Quality Assurance Project Plan developed for the RI (Exponent 1998). Only validated analytical data from fixed laboratory analyses were evaluated quantitatively in the risk assessment; no results from field and laboratory screening analyses were included in the quantitative assessment of risk. The mean of duplicate and replicate paired samples were included in the dataset. All results flagged with R qualifiers (indicating rejection of data) were excluded from use in the risk assessment.

Detection frequencies were calculated by dividing each analyte's number of detects by the total number of samples in which it was analyzed. If an analyte were not detected in any sample for a particular medium, then it was assumed that the chemical was not present, and the chemical was dropped from further consideration in the risk assessment. If the analyte was detected in at

[5] Samples removed from the database include the following stations and depths: B-49 (2 to 2.5 ft), B-53 (4.5 to 6 ft), B-61 (5 to 7 ft), IB-4 (6 to 7 ft), IB-17 (1 to 2 ft), IB-48 (1 to 2 ft), IB-77 (5 to 6 ft), IB-79 (1 to 1.5 ft), IB-82 (5 to 6 ft), IB-83 (5 to 6 ft), and IB-84 (5 to 6 ft). A partially removed soil sample (AP-2) was included in the analysis.

least one sample, the chemical concentration for all remaining nondetected results was assumed to be one-half of the detection limit.

3.3 GROUPING OF DATA INTO EXPOSURE UNITS

Due to different historical uses, current and potential future uses, and the potential for varying levels of contamination, the Site was divided into the following three categories for the purpose of risk assessment: Lots 1 and 2, Lots 3 and 4, and the riverbank (Tract A).

As described in Section 1, most of the Site's processing facilities and buildings were located on Lots 3 and 4 and other heavy industrial use occurred along the riverbank[6] and in nearshore waters. The former process buildings on Lots 3 and 4 were razed, and much of the area has been paved or covered with gravel. Also, much of the riverbank has been covered in riprap and other shore-stabilizing materials. In contrast, Lots 1 and 2 were relatively undeveloped.

For evaluation of COIs in soil, data were grouped according to three depths depending on the exposure scenario under consideration. The three soil horizons evaluated were 0-3 ft bgs, 0-15 ft bgs, and the entire unsaturated zone (designated as 0-Max). Table 3-1 presents a summary of exposure units and soil horizons that were evaluated for each potential receptor.

Direct contact with nearsurface soil was assumed to apply to a soil horizon of 0-3 ft bgs. Both an outdoor occupational worker and a trespasser were assumed to have direct contact exposure limited to the upper 3 ft of soil. Outdoor occupational worker exposure was evaluated for all three exposure units (Lots 1 and 2, Lots 3 and 4, and the riverbank). Trespasser exposure to nearsurface soil was evaluated for Lots 1 and 2 and the riverbank. Lots 3 and 4 are currently fenced and mostly covered with pavement or gravel; therefore, trespasser exposure to nearsurface soil on Lots 3 and 4 is not an exposure pathway of concern. However, there have been incidents where trespassers have cut the fence and accessed Lots 3 and 4 for skateboarding activities. Potential exposure to COPCs under the skateboarding scenario would be from COPCs in the concrete and fugitive dust. Although these exposures are likely to be small in comparison to direct contact with impacted soils, Arkema has taken measures to prevent future trespass on the Site by making it unattractive to skateboarders. For the purposes of this HHRA, it was assumed that the skateboarding scenario has been eliminated by institutional and engineering controls. Therefore, potential exposure of trespassers on Lots 3 and 4 was not quantified.

Direct contact with both nearsurface and subsurface soils was assumed to apply to a soil horizon of 0-15 ft. A construction worker, an excavation worker, and a future outdoor occupational worker under a redevelopment scenario were assumed to have direct contact

[6] The riverbank zone consists of the area along the Site's eastern boundaries within 15.24 m (50 ft) inland of the estimated mean high water level of the Lower Willamette River.

exposure to soils down to 15 ft bgs. For the construction and excavation workers, it was assumed that the maximum excavation or trench depth would be 15 ft bgs on Lots 1 and 2 and Lots 3 and 4. The maximum depth of 15 ft bgs reflects both the presence of the groundwater table at fairly shallow depths that is expected to preclude deeper construction activities and likely construction scenarios for anticipated future industrial land use. The riverbank area (Tract A) is steep and therefore, it is considered unlikely that future construction/excavation activities deeper than 3 ft will occur in the riverbank area. For this reason, an excavation worker scenario was not applied to the riverbank exposure area. For the construction worker, direct contact with soil in the riverbank area was assumed to be limited to a soil horizon of 0-3 ft bgs. For the future redevelopment worker, it was assumed that future redevelopment activities could bring subsurface soil to the surface. Under this scenario, a future occupational worker could be exposed to COIs that are currently in subsurface soils, even though the worker would only be contacting surface soils. For this reason, COIs from a soil horizon of 0-15 ft bgs were evaluated for the redevelopment worker scenario.

The entire unsaturated zone was evaluated as a potential source of inhalation exposure. Volatile COIs in the unsaturated zone were assumed to be available for volatilization and migration to outdoor ambient air or into overlying buildings. Both a future outdoor occupational worker and a current/future trespasser were assumed to have potential exposure to volatile COIs migrating upward to ambient air. A future indoor occupational worker was assumed to have potential exposure to volatile COIs migrating into overlying buildings. The depth to groundwater ranges from approximately 8-32 ft bgs across the Site. Therefore, future construction and excavation worker inhalation exposures to volatile COIs in subsurface soil was likely to be accounted for by the evaluation of soils in the horizon of 0-15 ft bgs and was not evaluated separately.

Groundwater data were not grouped by exposure area; rather the single groundwater dataset available for the quantitative risk assessment was applied to all areas of the Site (i.e., Lots 1 and 2, Lots 3 and 4, and the riverbank). Groundwater underlying Lots 3 and 4 has been well characterized (Integral 2006; ERM 2005) and is not assumed to be confined by administrative property boundaries. The groundwater dataset used in the quantitative risk assessment was almost exclusively from wells located on Lots 3 and 4. However, groundwater sampling on Lot 2 in 2005 did not indicate the presence of DDT, DDD, DDE, or perchlorate. Thus, the use of the groundwater dataset dominated by wells in Lots 3 and 4 may introduce a conservative bias in the risk estimates for Lots 1 and 2. A limited uncertainty analysis was conducted using the April 2007 groundwater sampling data, which included results from three new well clusters within the boundaries of Lots 1 and 2. The results of this analysis are reported in the Uncertainty Section of this report. There is no current or planned future use of groundwater as a source of drinking water, so direct contact with groundwater is not a complete exposure pathway for most Site workers or for trespassers.

For the excavation and construction scenarios, it was assumed that excavations would not exceed 15 ft bgs. In general, groundwater across the site occurs at depths greater than 15 ft bgs, except along the western boundary of the Site. COIs detected in these western boundary wells were limited to a few chlorinated compounds (e.g., 1,2-dichlorobenzene (DCB), 1,1-dichloroethane, and chlorobenzene), which are likely related to offsite sources. Recent repairs to the storm water system along Front Avenue may exacerbate this problem as previous leaks may have allowed the system to intercept a percentage of the upgradient contaminated groundwater before if flowed onto the Arkema property. The detected COI concentrations were several orders of magnitude below DEQ risk-based concentrations (RBCs) for direct contact of groundwater by an excavation worker. In addition, it is assumed that any excavation activities below the water table would require dewatering. Therefore, it is believed that exposure and potential risks associated with direct contact with COIs in groundwater by excavation and construction workers would be unlikely to occur.

Volatile COIs in groundwater have the potential to migrate upward to the land surface or into overlying buildings. All groundwater monitoring data (e.g., data collected from all depths) were included for evaluation of groundwater vapor migration to indoor and outdoor air. Receptors with potential exposure to vapors migrating from groundwater include an indoor occupational worker, outdoor occupational worker, trespasser, excavation worker, and construction worker. COPC screening for vapor migration from groundwater was conducted for an indoor worker, outdoor occupational worker, and trespasser. The outdoor occupational worker is assumed to have a substantially greater exposure duration than an excavation or construction worker; therefore, any remediation required to address potential risks to outdoor workers would also be protective of excavation and construction workers. For this reason, potential exposure of construction/excavation workers to COIs in groundwater will not be evaluated quantitatively.

3.4 CHEMICAL SCREENING

This section describes the selection of COPCs for media sampled at the Site, which includes soil and groundwater. The purpose of the COPC selection process is to help focus the HHRA on the chemicals that may drive human health risks at the Site, given the knowledge already gained from existing data and evaluation of historical operating practices.

3.4.1 COPC Selection Process

The COPC selection process involves multiple steps that are outlined in federal and state guidance (USEPA 1989; DEQ 2000, 2003). These steps include selecting risk-based screening levels and appropriate natural background levels, and comparing Site concentrations to the screening and background levels.

The first step in the screening process involves the identification of background concentrations of naturally occurring COIs (DEQ 2000, 2003). If the maximum detected concentration is less than the selected background level, then it is not retained as a COPC. Background concentrations for inorganic compounds were obtained from DEQ (2002) and are listed in Table 3-2. EPA (1989) also recommends removing chemicals from further consideration if they are considered "essential nutrients," such as aluminum, magnesium, calcium, sodium, and potassium.

The second step in the selection of COPCs requires comparing concentrations of COIs with RBCs (DEQ 2000, 2003). DEQ provides RBCs for a wide variety of potential receptors and exposure pathways. DEQ RBCs were used to screen COIs for inclusion as COPCs for both soil and groundwater. Where DEQ RBCs were not available, medium-specific screening levels (MSSLs) provided by EPA Region 6 (2007a) were used. In cases where DEQ RBCs were not available for vapor migration pathways, risk-based screening values were calculated using EPA's vapor intrusion worksheets for evaluation of the soil-to-indoor air and groundwater-to-indoor air migration pathways (EPA 2004a; Johnson and Ettinger 1991). Tables 3-3 through 3-23 summarize the results of the COPC screening for each potential receptor and exposure area. The source of the RBC used to screen for each COI is indicated in each table. The remainder of this section provides a description of the screening levels used to select COPCs.

DEQ's RBCs are applicable to occupational, construction, and excavation worker scenarios (DEQ 2000, 2003). For the trespasser, RBCs applicable to a residential scenario were used due to a lack of values specific to trespasser exposures. The use of the residential RBCs represents a conservative approach given the more limited exposures that a trespasser is assumed to experience at the Site. Soil RBCs are based on exposure to soil via direct contact (ingestion, dermal contact) and inhalation. Additional RBCs are provided to screen for inhalation of vapors from soil in indoor (Appendix B1) and outdoor air. RBCs for groundwater are based on inhalation of vapors in indoor and outdoor air. DEQ RBCs correspond to a target cancer risk of one in a million (1×10^{-6}) or a noncancer hazard quotient (HQ) of 1.

The DEQ occupational scenario is similar to the EPA Region 6 MSSL occupational scenario in that they are both based on an exposure duration of 25 years and soil ingestion rate of 100 mg/day. Where DEQ RBCs were not available for the outdoor occupational worker, EPA Region 6 MSSLs were used as RBCs.

The excavation and construction worker scenarios represent subchronic exposures, both with exposure durations of 1 year. The excavation worker exposure frequency is 9 days/year and the construction worker exposure frequency is 250 days/year. The soil ingestion rate for both excavation and construction workers is 330 mg/day. Other exposure parameters are described in DEQ guidance (2003). In cases where DEQ RBCs for construction/excavation were not available, EPA Region 6 MSSLs for an outdoor industrial worker were used as risk-based screening levels. The MSSLs for an outdoor industrial worker are based upon a lower soil

ingestion rate of 100 mg/day and a smaller soil adherence factor of 0.2; however, they also are based upon a substantially greater exposure period of 25 years (as compared to 1 year for the RBCs).

The trespasser exposure scenario represents chronic exposure over a total period of 10 years. Since the trespasser scenario includes potential exposure of a youth over age 12, residential screening values were applied. Residential RBCs are based upon an assumed exposure frequency of 350 days/year and age-adjusted intake rates. The likely exposure frequency for a trespasser would be far less than the 350 days/year value used in the RBC.

RBCs were not available for all COIs that may volatilize to outdoor air from groundwater and subsurface soil. When RBCs for inhalation of outdoor air were not available, risk-based screening levels for vapor migration to indoor air were used as surrogate screening levels.

EPA Region 6 MSSLs for soil were selected to screen analytical results for COIs not addressed by DEQ RBCs. MSSLs are available for a residential soil scenario, an industrial indoor worker (inhalation only), and an industrial outdoor worker. The MSSLs are based on direct contact exposure pathways (ingestion, dermal contact, and inhalation) and correspond to a target cancer risk of one in a million (1×10^{-6}) or a noncancer HQ of 1.

The industrial land use scenario in the Region 6 MSSLs assumes an exposure frequency of 250 days/year and exposure duration of 25 years. The soil ingestion rate for the workers is 100 mg/day. The residential land use scenario for MSSLs assumes an exposure frequency of 350 days/year and exposure duration of 30 years. Age-adjusted intake rates are used to estimate potential intakes from birth to age 30. Additional exposure parameters are described in the EPA Region 6 MSSL background document (EPA 2006).

EPA's Vapor Intrusion SL-SCREEN and GW-SCREEN spreadsheets (USEPA 2004a), based on the Johnson and Ettinger (1991) building infiltration model, were used to calculate soil and groundwater RBCs for volatile COIs when DEQ's generic RBCs for vapor intrusion or volatilization to outdoor air were not available. Volatile COIs were identified as those chemicals with a Henry's constant greater than 1×10^{-5} m^3-atm/mol and a molecular weight less than 200 g/mol (DEQ 2003).

The model inputs were modified to represent a worker with an exposure frequency of 250 days/year and exposure duration of 25 years. Default and site-specific data were used to calculate RBCs based on a target cancer risk of one in a million (1×10^{-6}) or a noncancer HQ of 1. Site-specific values obtained from previous environmental investigation reports (Integral 2006) were used for dry bulk density and total organic carbon, listed in Table 3-24. Default values for sandy silt/silty sand soil were obtained from EPA guidance (2004a).

The upland soil and groundwater data described in Section 2.3 were used in the chemical screening process to determine if the COIs are present at concentrations that would pose a risk

to workers. The maximum concentrations of COIs were compared to the risk-based screening levels described above. As specified in DEQ guidance (2003), the COI was retained as a COPC if the COI concentration exceeded the screening level.

COIs that were detected in soil or groundwater at a frequency of detection less than 5 percent were evaluated for exclusion as COPCs. If the COI was considered to be non-Site-related on the basis of historical Site information (as was the case for a few pesticides and phthalates) and if there were sufficient numbers of samples (N>20), then the COI was not included as a COPC.

To account for exposures to multiple chemicals in soil or groundwater, a COI also was retained as a COPC if the condition of the following Equation was met:

$$\frac{C}{RBC} > \frac{1}{N} \qquad \text{(Eq. 3-1)}$$

Where:

C = Chemical concentration

N = Total number of COIs identified for a particular scenario.

For carcinogens, N was equal to the count of all carcinogenic COIs detected in the pertinent exposure medium and exposure area. For noncarcinogens, N was equal to the count of all noncarcinogenic COIs that affect the same target organ within the respective exposure medium and exposure area. The COIs were grouped for the multiple chemical evaluation in this manner to minimize the artifact of greater numbers of samples automatically resulting in greater numbers of COPCs, even when concentrations remained the same.

3.4.2 Results of COPC Screening

The COPC screening was conducted for all receptors and pathways identified in the CSM (see Figure 2-1). The screening is a conservative tool that is used to focus the risk assessment on the relevant Site chemical. The results of the COPC selection process are presented in Tables 3-3 through 3-23. Each table presents a summary of all COIs detected in the environmental medium, the risk-based screening level or RBC and its source, total number of samples, frequency of detection, minimum and maximum detected concentrations, mean concentration (assuming a normal distribution), location of the maximum detected concentration, and the results of the risk-based screening evaluation. The "individual COPC" column indicates whether or not the maximum detected concentration exceeded the RBC. The "greater than 5% FOD" column indicates whether or not the COI was excluded as not site-related. The "grouped multiple COPC" column indicates whether or not the COI should be retained as a COPC on the basis of potential exposure to multiple chemicals. The results of the COPC screening are

presented below. Given the conservative nature of the COPC screening process, which relies on maximum detected concentrations and upper-bound exposure assumptions, the selection of a chemical as a COPC is no indication of potential risk; rather, it indicates further analysis is warranted.

3.4.2.1 Lots 1 and 2 Soil

Maximum concentrations of COIs in Lots 1 and 2 soils were compared to RBCs and MSSLs for evaluation of five worker scenarios: outdoor occupational worker, construction worker, excavation worker, redevelopment worker, and indoor worker. Maximum concentrations of COIs in Lots 1 and 2 soil were also compared to RBCs and MSSLs for evaluation of a trespasser scenario. No COIs were eliminated based on comparison with natural background levels of inorganic compounds.

For an outdoor occupational worker, the COPC screening is shown in Table 3-3. COPCs selected based on comparison with RBCs include arsenic, benzo(a)pyrene (BaP), and 4,4-DDT. Following further screening to account for exposures to multiple COIs in soil, benz(a)anthracene, benzo(b)fluoranthene, Aroclor 1248, 4,4-DDE, and 4,4-DDD also were selected as COPCs. The same COPCs were identified for the future occupational worker under the redevelopment scenario as shown in Table 3-4.

Two COPCs, arsenic and 4,4-DDT, were selected for the construction worker scenario following comparison with RBCs (see Table 3-5). Additional COPCs selected when considering cumulative exposures to multiple COIs include BaP and Aroclor 1248.

No individual COI exceeded the COPC screening values for the excavation worker scenario. Arsenic and 4,4-DDT were selected as COPCs for the excavation worker scenario following screening to account for exposure to multiple COIs in soil (see Table 3-6).

Worker inhalation exposures were assumed for ambient and indoor air as a result of migration of volatiles in the soil of Lots 1 and 2. As shown in Table 3-7 all but two of the COIs identified in Lots 1 and 2 soil are not considered sufficiently volatile for consideration under the vapor intrusion to indoor air or volatilization to ambient air exposure pathways. Of the two COIs considered sufficiently volatile for consideration of the volatilization pathways, neither exceeded its respective RBC for the occupational scenarios. No COPCs were selected for this pathway; therefore, the indoor worker scenario and inhalation of vapors in outdoor air will not be evaluated further for Lots 1 and 2.

For the potential trespasser the COPC screening is shown in Table 3-8. COPCs selected based on comparison with RBCs include arsenic, benz(a)anthracene, BaP, benzo(b)fluoranthene, indeno(1,2,3-cd)pyrene, Aroclor 1248, 4,4-DDD, 4,4-DDE, and 4,4-DDT. Further screening to account for exposure to multiple COIs in soil resulted in the inclusion of lead and benzo(k)fluoranthene as well.

Trespassers could be exposed to volatile chemicals released into ambient air from soil within Lots 1 and 2. As shown in Table 3-9 all but two of the COIs identified in Lots 1 and 2 soil are not considered sufficiently volatile for consideration via the volatilization to ambient air exposure pathway. Of the two COIs considered sufficiently volatile for consideration of the volatilization pathway, neither exceeded its respective RBC for the residential exposure scenario. No COPCs were selected for this pathway; therefore, the trespasser inhalation of vapors in outdoor air will not be evaluated further for Lots 1 and 2.

3.4.2.2 Lots 3 and 4 Soil

Maximum concentrations of COIs in Lots 3 and 4 soil were compared to DEQ RBCs and EPA MSSLs for evaluation of five worker scenarios. No inorganic COIs were eliminated based on a comparison with natural background levels.

As shown in Table 3-10, COPCs selected for the outdoor occupational worker scenario based upon comparison of maximum detected concentration to RBC included Aroclor 1248, 4,4-DDT, 4,4-DDD, 4,4-DDE, and chlorobenzene. Following additional screening to account for exposures to multiple COIs, Aroclor 1260 and 1,4-DCB also were selected as COPCs.

COPC screening for the outdoor worker under a redevelopment scenario is shown in Table 3-11. COPCs selected on the basis of individual screening include chromium (total), Aroclor 1248, 4,4-DDD, 4,4-DDE, 4,4-DDT, PCE, and chlorobenzene. Screening conducted to account for exposure to multiple COIs in soil resulted in the addition of Aroclor 1260 and 1,4-DCB. Residual range organic hydrocarbons (RRO) were also indicated as a potential contributor to cumulative risk. However, since the toxic constituents of RRO were evaluated separately, RRO was not selected as a COPC.

Screening against RBCs for the construction worker scenario, shown in Table 3-12, resulted in selection of the following COPCs: chromium (total), Aroclor 1248, 4,4-DDT, 4,4-DDD, 4,4-DDE, and chlorobenzene. Further screening conducted to account for exposure to multiple COIs in soil resulted in the addition of PCE. Diesel range organic hydrocarbons (DRO) were also indicated as a potential contributor to cumulative risk. However, potentially toxic constituents of DRO (aromatic hydrocarbons) were also analyzed and evaluated separately; therefore, DRO was not selected as a COPC.

For the excavation worker, shown in Table 3-13, COPCs include chromium (total) and 4,4-DDT. Additional COPCs selected following screening to account for exposure to multiple COIs include 4,4-DDD and 4,4-DDE.

Most COIs were not considered sufficiently volatile to be relevant for the vapor intrusion and volatilization pathways. Of the detected COIs in Lots 3 and 4 soils, none were found to exceed RBCs for volatilization to ambient air for outdoor occupational workers or trespassers (see Table 3-15); therefore, the inhalation exposure scenario for soil volatilization to ambient air was

not evaluated further. For the indoor air worker exposure, 1,4-DCB and PCE exceeded their respective vapor intrusion to indoor air RBCs, as shown in Table 3-15,and were retained as COPCs.

3.4.2.3 Riverbank (Tract A) Soil

Maximum concentrations of COIs in riverbank soil were compared to DEQ RBCs and EPA MSSLs for evaluation of two worker scenarios: the outdoor occupational worker scenario and the construction worker scenario. COPC screening was also conducted for a trespasser scenario. No COIs were eliminated based on comparison with natural background levels of inorganic compounds, although aluminum was eliminated as a COPC because it is considered an essential nutrient (USEPA 1989). The maximum detected concentration of dioxins and furans used for COPC screening was the total toxic equivalency (TEQ) of all applicable dioxins/furans in the sample to 2,3,7,8-TCDD. PCBs were speciated in only one riverbank soil sample and detected concentrations were orders of magnitude below RBCs for total PCBs. Therefore, for the purposes of the HHRA, only Aroclor 1248 and 1260 were identified as COIs for the riverbank.

As shown in Table 3-16, COPCs for the outdoor occupational worker scenario include dioxins/furans, arsenic, chromium (total), lead, BaP, benzo(b)fluoranthene, dibenz(a,h)anthracene, and DDT. Additional COPCs retained for analysis following evaluation of exposure to multiple COIs include Aroclors 1248 and 1260, benz(a)anthracene, benzo(k)fluoranthene, indeno(1,2,3-cd)pyrene, DDD, DDE, PCP, and bis(2-ethylhexyl)phthalate (BEHP). As shown in Table 3-17, no COPCs were identified for outdoor workers via exposure to volatilization from soils to ambient air. The same results are applicable for the future development worker.

As shown in Table 3-18 COPCs selected for the construction worker include dioxins/furans, arsenic, chromium (total), and lead. Further screening to account for exposure to multiple COIs resulted in the selection of Aroclor 1248, benz(a)anthracene, BaP, benzo(b)fluoranthene, dibenz(a,h)anthracene, indeno(1,2,3-cd)pyrene, 4,4-DDE, and 4,4-DDT as COPCs for the construction worker.

As shown in Table 3-19,COPCs selected for the trespasser include dioxins/furans, arsenic, chromium (total), lead, benz(a)anthracene, BaP, benzo(b)fluoranthene, benzo(k)fluoranthene, dibenz(a,h)anthracene, indeno(1,2,3-cd)pyrene, Aroclor 1248, DDE, and DDT. Additional COPCs retained after evaluation of exposure to multiple COIs include Aroclor 1260, chrysene, DDD, PCP, BEHP, hexachloroethane, and hexachlorobenzene. As shown in Table 3-20, no COPCs were identified for soil volatilization to ambient air based on trespasser exposure.

Excavation workers, redevelopment workers, and indoor workers were not included as relevant scenarios for the riverbank exposure area.

3.4.2.4 Groundwater

The maximum concentration of COIs in groundwater were compared to RBCs for evaluation of the vapor intrusion to indoor air and volatilization to outdoor air exposure pathways. Maximum detected concentrations of COIs in groundwater were compared to RBCs for three different potential receptors: an indoor occupational worker, an outdoor occupational worker, and a trespasser. DEQ RBCs for vapor intrusion into buildings (occupational) were used as risk-based screening levels for the indoor worker scenario. As shown in Table 3-21, none of the maximum detected concentrations in groundwater exceeded the RBCs. However, further screening to account for exposure to multiple COIs resulted in the selection of chloroform and PCE as COPCs for the future indoor worker.

For the outdoor occupational worker, DEQ RBCs for volatilization to outdoor air (construction and excavation worker) were used as risk-based screening levels. These RBCs are based upon an exposure frequency of 250 days/year, an exposure duration of 1 year, and an inhalation rate of 7 m^3/day (based on an 8-hour workday). As shown in Table 3-22, maximum detected concentrations of volatile COIs in groundwater did not exceed the RBCs. Screening to account for exposure to multiple COIs identified chloroform and TCE as potential COPCs for outdoor occupational workers.

For the trespasser, DEQ RBCs for volatilization to outdoor air (residential) were used as risk-based screening levels. These RBCs are based upon an exposure frequency of 350 days/year over 30 years and an age-adjusted inhalation rate based upon a 24-hour day. The residential RBCs were selected as screening values because they incorporate potential exposure of children. However, the use of these RBCs to screen for trespasser exposure is conservative, given that the exposure frequency and exposure time of a trespasser is substantially less than that of a resident. In addition, the likely age of a trespasser is greater (9 years old and up) than the ages assumed for residential exposure (birth and up). As shown in Table 3-23, none of the maximum detected concentrations of volatile COIs in groundwater exceeded the residential RBCs. Screening to account for exposure to multiple COIs identified chloroform, PCE, TCE, and vinyl chloride as potential COPCs for the trespasser scenario. However, given that the extremely conservative individual screening did not identify any COPCs for the trespasser, potential risks associated with vapor migration to outdoor air will be assessed qualitatively.

3.5 CALCULATION OF EXPOSURE POINT CONCENTRATIONS

To estimate the magnitude of exposure to a COPC in each exposure medium (soil or groundwater), a representative concentration for each COPC for each exposure area was calculated. This concentration, an exposure point concentration (EPC), is used to calculate intake of each COPC in the exposure assessment (Section 3). EPCs may be derived directly from discrete and/or composite sample data or may be modeled when a particular medium is not sampled directly.

DEQ recommends the use of the upper 90th percentile confidence limit of the arithmetic mean (upper confidence limit [UCL]90) to represent the EPC. The UCL90 is used by DEQ to represent the average exposure to contaminants in a given medium that a receptor is likely to contact over time for the purpose of calculating human health risk.

Methods for calculating UCLs are provided in *Calculating Upper Confidence Limits for Exposure Point Concentrations at Hazardous Waste Sites* (USEPA 2002a) and in *ProUCL Version 4.0 User Guide* (Singh et al. 2004). EPA's ProUCL software (USEPA 2004b) was used to calculate the UCL90 for each COPC. For Lots 1 and 2 and Lots 3 and 4, ProUCL version 3.0 was used. For the riverbank data (with more recently collected samples), ProUCL version 4.0 was used. The more recent software version offers a greater number of statistical functions, but the estimation of a UCL90 in both versions is based upon the same statistical principles. ProUCL was used to assess the distribution type for each analyte and calculate either parametric or nonparametric UCL90s. The appropriate UCL90 was selected based on the dataset distribution. When UCL90 calculations were determined inappropriate (i.e., small sample size or UCL exceeds maximum concentration), the maximum detected concentration was used to represent the EPC.

The following sections describe the calculation of EPCs for specific media using sample data and/or fate and transport models.

3.5.1 EPCs for Soil

EPCs for nearsurface and subsurface soil for all exposure units are shown in Table 3-25. EPCs for both central tendency exposure (CTE) and reasonable maximum exposure (RME) are presented for each soil horizon evaluated.

For Lots 1 and 2, EPCs were generated for an outdoor occupational worker and a trespasser potentially exposed to COPCs in soil from 0 to 3 ft bgs. Given that soil data were not available for deeper than 3 ft, the EPCs were for an excavation worker, a construction worker, and a future redevelopment worker potentially exposed to COPCs in soil from 0 to 15 ft bgs are the same as for soil from 0 to 3 ft bgs. No COPCs were identified for the pathway of vapor migration from subsurface soil to indoor or ambient air; therefore, no EPCs were generated for the soil horizon encompassing the entire unsaturated zone.

For Lots 3 and 4, EPCs were generated for an outdoor occupational worker potentially exposed to COPCs in soil from 0 to 3 ft bgs. EPCs were generated for an excavation worker, a construction worker, and a future redevelopment worker potentially exposed to COPCs in soil from 0 to 15 ft bgs. EPCs were generated for indoor and outdoor occupational workers potentially exposed to vapors migrating from the unsaturated zone (0-Max).

For riverbank soils, EPCs were generated for an outdoor occupational worker, construction worker, redevelopment worker, and trespasser potentially exposed to COPCs in soil from 0 to

3 ft bgs. EPCs were generated for an outdoor occupational worker and trespasser potentially exposed to vapors migrating from the unsaturated zone (0-Max).

3.5.2 EPCs for Groundwater

Groundwater data were not grouped by exposure area. All groundwater data were combined to evaluate vapor migration pathways. EPCs for groundwater are shown in Table 3-26. EPCs for both CTE and RME are presented. The groundwater dataset used to calculate the EPCs was almost exclusively from wells located on Lots 3 and 4, which introduces a conservative bias in the risk estimates for Lots 1 and 2. A limited uncertainty analysis was conducted using the April 2007 groundwater sampling data, which included results from three new well clusters within the boundaries of Lots 1 and 2. The results of this analysis are reported in the Uncertainty Section of this report.

3.5.3 EPCs for Fugitive Dust

Fugitive dust was not sampled directly, so EPCs were calculated using a particulate emission factor. The particulate emission factor relates the chemical concentration in soil to an estimated chemical concentration associated with respirable particles in air due to dust emissions from contaminated soil.

Because risks due to inhalation of particulates are generally low relative to other exposure routes, no attempt was made to calculate a site-specific particulate emission factor. DEQ (2003) recommends using a default value of 7.58×10^{-10} m^3/kg generated by EPA, which is a 90th percentile value for a 0.5-acre site. The default factor is based on a dispersion modeling study conducted by EPA to estimate fugitive dust emission at various sites (USEPA 1996). Factors influencing the particulate emission factor include the amount of ground cover present, soil type, and wind speed. The default particulate emission factor recommended by DEQ is protective for this HHRA, given that large portions of the Site are covered with vegetation, pavement, or gravel.

The generic particulate emission factor was applied in the following equation to calculate EPCs resulting from fugitive dust:

$$C_f = C_s / PEF \qquad \text{(Eq. 3-2)}$$

Where:

C_f	=	Steady-state chemical concentration in outdoor air (mg chemical/m^3 air)
C_s	=	Soil concentration of chemical (mg chemical/kg soil)
PEF	=	Generic particulate emission factor (m^3 air/kg particulate).

3.5.4 EPCs for Vapors in Outdoor Air

As with fugitive dust, vapors in outdoor air were not sampled directly. EPCs for vapors were estimated by modifying the groundwater EPCs by a volatilization factor. The volatilization factors account for the rate at which a chemical volatilizes from water and how well it is dispersed in ambient air.

DEQ (2003) provides recommended methods for calculating the vapor concentration in ambient air resulting from migration of vapors from groundwater, shown in Equation 3-3. The Henry's constant is used to determine the amount of chemical vapor directly above the groundwater. The chemical-specific total effective diffusion coefficient and depth bgs to the water table represent the transport of vapor upwards through the soil to the surface. EPA's modeled chemical flux-concentration relationship, Q/C, also is used to estimate flux of chemical at the ground surface (USEPA 1996).

$$VF_w = \frac{D_{Teff} \times H \times 10^3\, g/kg \times 10^3\, L/m^3}{(Q/C) \times L_w \times 10^2\, cm/m} \qquad \text{(Eq. 3-3)}$$

Where:

VF_w	=	Volatilization factor for groundwater vapor migration to outdoor air (L/m^3)
D_{Teff}	=	Total effective diffusion coefficient (cm^2/s)
H	=	Henry's constant (unitless)
Q/C	=	Modeled flux to concentration ratio (g/m^2-s per kg/m^3)
L_w	=	Depth below ground surface to top of water table (cm).

For this HHRA, EPA's modeled Q/C value was used (USEPA 1996). The total effective diffusion coefficient is calculated using soil characteristic parameters and diffusion coefficients in air and water. This value was calculated during the vapor intrusion to indoor air modeling (Section 3.5.5) and was also used in Equation 3-3. Then, the chemical-specific volatilization factors are applied to the chemical concentration in groundwater to obtain a vapor concentration in ambient air, as shown in Equation 3-4:

$$C_a = VF_w \times C_w \qquad \text{(Eq. 3-4)}$$

Where:

C_a	=	Groundwater vapor concentration in ambient air (mg/m^3)
C_w	=	Chemical concentration in groundwater, EPC (mg/L).

Equation inputs and calculated chemical-specific volatilization factors are provided in Table 3-27.

3.5.5 EPCs for Vapors in Indoor Air

A building infiltration model was used to calculate chemical concentrations in indoor air resulting from volatilization of contaminants from soil or groundwater and migration through cracks in the building foundation into indoor air (Appendix B2). The model is based on an indoor infiltration model developed by Johnson and Ettinger (1991), and modified by EPA (2004a). The model couples both advective and diffusive flow of soil gases and considers the resistance caused by the foundation on the infiltration rate into a building. The EPCs for COPCs in soil and groundwater, calculated as described above, were entered into the model to estimate vapor concentrations.

The amount of building infiltration from soil gas can be determined from the ratio of the contaminant concentration in the indoor air to the soil gas concentration at the source (Johnson and Ettinger 1991; USEPA 2004a):

$$\frac{C_b}{C_a} = \frac{\left[\frac{D_{eff}\,A_b}{Q_b\,L_T}\right] \times \exp\left(\frac{Q_s\,L_{crack}}{D_{crack}\,A_{crack}}\right)}{\exp\left(\frac{Q_s\,L_{crack}}{D_{crack}\,A_{crack}}\right) + \left[\frac{D_{eff}\,A_b}{Q_b\,L_T}\right] + \left[\frac{D_{eff}\,A_b}{Q_s\,L_T}\right] \times \left[\exp\left(\frac{Q_s\,L_{crack}}{D_{crack}\,A_{crack}}\right) - 1\right]} \qquad \text{(Eq. 3-5)}$$

Where:

C_b	=	Chemical concentration in indoor air (g/m^3)
C_a	=	Chemical concentration in soil gas (g/m^3)
D_{eff}	=	Effective diffusion coefficient through soil (cm^2/sec)
A_b	=	Area of building foundation and below grade walls (cm^2)
Q_b	=	Building ventilation rate (cm^3/sec)
L_T	=	Distance from contaminant source to building foundation (distance between the building foundation and the water table or contamination source) (cm)
Q_s	=	Soil gas emission rate into building (cm^3/sec)
L_{crack}	=	Thickness of foundation (cm)

D_{crack} = Effective diffusion coefficient through crack (cm^2/sec)

A_{crack} = Area of cracks in foundation through which vapors can pass (cm^2).

The effective diffusivity, D_{eff} in Equation 3-5 is a function of the soil porosity and moisture content, and can be expressed as:

$$D_{eff} = D_a \left(\frac{\theta_a^{3.33}}{\phi^2} \right) + \left(\frac{D_w}{H} \right) \left(\frac{\theta_w^{3.33}}{\phi^2} \right) \qquad \text{(Eq. 3-6)}$$

Where:

D_{eff} = Effective diffusion coefficient (cm^2/sec)

D_a = Diffusivity in air (cm^2/sec)

θ_a = Air-filled soil porosity (dimensionless)

ϕ = Total soil porosity (dimensionless)

D_w = Diffusivity in water (cm^2/sec)

H = Henry's Law constant at system (soil or groundwater) temperature (dimensionless)

θ_w = Water-filled soil porosity (dimensionless).

In addition to the unsaturated zone, a saturated capillary zone exists directly above the water table. This layer will tend to reduce vapor diffusion. Therefore, when considering vapor infiltration from groundwater, the effective diffusivity must be averaged over all distinct soil layers, including the capillary zone, as follows:

$$D_T^{eff} = \frac{L_T}{\sum_{i=0}^{n} \frac{L_i}{D_i^{eff}}} \qquad \text{(Eq. 3-7)}$$

Where:

D_T^{eff}	=	Total overall effective diffusion coefficient (cm^2/sec)
L_T	=	Distance between source of contamination and bottom of enclosed space floor (cm)
L_i	=	Thickness of soil layer i (cm)
D_i^{eff}	=	Effective diffusion coefficient across soil layer i (cm2/sec).

The soil gas emission rate into the building, Q_s in Equation 3-5, can be estimated using the following equation:

$$Q_s = \frac{2\pi \Delta P k X_{crack}}{\mu \ln\left(\frac{2 Z_{crack}}{r_{crack}}\right)} \qquad \text{(Eq. 3-8)}$$

Where:

ΔP	=	Indoor-outdoor pressure difference (g/cm-sec^2)
k	=	Soil permeability to vapor flow (cm^2)
X_{crack}	=	Total horizontal length of crack (cm)
μ	=	Soil gas viscosity (g/cm-sec)
Z_{crack}	=	Depth of foundation below surface (cm)
r_{crack}	=	Radius of crack (cm).

This equation is an analytical solution for flow to a cylinder of length X_{crack} and radius r_{crack}, located at depth Z_{crack} below ground surface, (assuming $r_{crack} << Z_{crack}$), which represents an idealized model for soil gas flow to cracks located at floor/wall seams.

Note that in the Johnson and Ettinger (1991) paper, the crack radius term (r_{crack}) is incorrectly applied as the total width of the crack when calculating crack area. As a result, the crack radius term (r_{crack}) is incorrectly applied in the EPA revised models. The soil gas emission equation using the r_{crack} term in Johnson and Ettinger (1991) was taken directly from the work of Nazaroff (1988), and is presented as Equation 3-7 above. The original Nazaroff (1988) paper clearly used a horizontal cylinder, with a diameter two times the r_{crack} term, to represent the crack width. However, because the r_{crack} value appears in a logarithmic term (see Equation 3-8), a difference of a factor of two in the r_{crack} term is not critical. Therefore, when the Johnson and Ettinger (1991) suggestion of using the r_{crack} value as the total width of the crack is applied (as is done in the revised EPA model that was used in the risk evaluation), it should be recognized that the resulting soil gas emission rate into the building (Q_s) is slightly higher than the correctly modeled rate.

The chemical concentration in soil gas (C_a) can be determined from either the concentration in soil, or the concentration in soil-water (e.g., at the groundwater table interface).

Input for C_a with concentration in soil as input:

$$C_a = \frac{H\, C_s\, \rho_b}{\theta_w + K_d \rho_b + H\theta_a} \qquad \text{(Eq. 3-9)}$$

Input for C_a with concentration in groundwater as input:

$$C_a = H\, C_w \qquad \text{(Eq. 3-10)}$$

Where:

C_a	=	Chemical concentration in soil gas (mg/m^3)
H	=	Henry's Law constant at the system (soil or groundwater) temperature (dimensionless)
C_s	=	Chemical concentration in soil (mg/kg)
ρ_b	=	Soil dry bulk density (g/cm^3)
θ_w	=	Water-filled soil porosity (dimensionless)
K_d	=	Soil-water partitioning coefficient (mL/g)
θ_a	=	Air-filled soil porosity (dimensionless)

C_w = Chemical concentration in soil water (µg/L).

The soil partition coefficient (K_d) is given by:

$$K_d = K_{oc} \cdot f_{oc} \qquad \text{(Eq. 3-11)}$$

Where:

K_d = Soil-water partition coefficient (cm^3/g)

K_{oc} = Organic-carbon partitioning coefficient (cm^3/g)

f_{oc} = Fraction of organic carbon (unitless).

Typical assumptions for certain input parameters are discussed below. Input parameters are summarized in Tables 3-28 and 3-29 indicating the values that were used in this risk evaluation.

The crack length (x_{crack}) is assumed to be equal to the perimeter of the building. The radius of the crack is calculated from the crack area and crack length:

$$r_{crack} = \eta \left(\frac{A_B}{x_{crack}} \right) \qquad \text{(Eq. 3-12)}$$

where the crack area fraction (η) is:

$$\eta = \mathrm{A}_{\mathrm{crack}}/\mathrm{A}_{\mathrm{B}} = \left[2(L_B \times w) + 2(W_B \times w)\right] / \mathrm{A}_{\mathrm{B}} \cong 0.05\% \qquad \text{(Eq. 3-13)}$$

As discussed earlier, calculating the r_{crack} value as the total width of the crack overestimates the soil gas emission rate into the building.

4 EXPOSURE ANALYSIS

Information regarding the Site setting, current and future land and water use, and knowledge of the nature and extent of contamination was used to develop the chemical pathways analysis (Section 2). This analysis provides a framework for the initial approach to the HHRA, identifying potential receptors and exposure pathways. Following the evaluation of existing Site data and ensuring that data quality objectives were met, data were screened to determine which COIs should be retained as COPCs. Results of the COPC selection process (Section 3) were used to refine the CSM, presented below. Following the refined CSM, methods for calculating intake and intake estimates are presented for all relevant receptors.

4.1 REFINED CONCEPTUAL SITE MODEL

The primary sources and migration pathways for COIs are discussed in Section 2. Figure 2-1 identifies exposure media and potential exposure routes for a variety of Site users. Of interest for this risk evaluation of upland media are COIs in surface and subsurface soil and groundwater. People who may contact COIs in these media include indoor and outdoor workers, construction and excavation workers, and trespassers. Following the COI screening process, complete exposure pathways were identified and are discussed in the following section. The refined CSM is presented in Figure 4-1.

4.1.1 Lots 1 and 2

Lots 1 and 2 are largely undeveloped and contain a mixture of vegetated areas interspersed with bare soil. On the northeastern edge of Lots 1 and 2 is a stand of black cottonwood trees, a willow thicket, and an area overgrown with scrub-shrub vegetation. Trees and scrub-shrub vegetation also exist on the western end of Lot 1. The remainder of the property is vegetated with grass or is bare. Trespassers entering the Site may have contact with chemicals in areas where there is little or no vegetation and the surface soil is exposed. Although these exposures are expected to be very short in duration relative to exposures of onsite workers, risks for the trespasser were quantified for the following:.

- Incidental ingestion of soil
- Inhalation of wind-blown dust
- Dermal contact with soil.

Current or future outdoor occupational workers, excavation workers, and construction workers may have contact with COPCs in surface and subsurface soil. Complete exposure pathways that were quantified for the outdoor workers include the following:

- Incidental ingestion of soil
- Inhalation of wind-blown dust
- Dermal contact with soil.

Based on the results of the COI screening, it was determined that volatile COPCs are not present at concentrations that are expected to pose a risk to trespassers or worker health for exposures via inhalation; therefore, inhalation of vapors in indoor and outdoor air were not quantified.

Future excavation workers also may contact COPCs in subsurface soil during construction of trenches for installation or repair of utilities. Although trench workers would be expected to wear protective clothing in conformance with a site-specific health and safety plan, it is assumed that protective clothing is not worn and that trench workers may have direct and indirect contact with COPCs in subsurface soil (less than 15 ft bgs) via the following exposure routes:

- Incidental ingestion of trench soil
- Dermal contact with trench soil
- Inhalation of soil as resuspended dust.

According to the beneficial water use survey, groundwater will not be used as a source of drinking water, so direct contact with groundwater by workers at the Arkema Site in the form of ingestion and dermal contact does not represent a complete exposure pathways.

4.1.2 Lots 3 and 4

The decommissioning and removal of chemical manufacturing infrastructure was completed in early 2005, and the only remaining building is the office building located at the Site entrance on Front Street. Currently, one part-time office worker occupies this building. Other current personnel include one part-time outdoor worker who operates and maintains active remediation systems and contract workers who may be present onsite for short periods to conduct removal and other remedial actions. Lots 3 and 4 are enclosed by security fencing and mostly covered by concrete and gravel. Potential exposure of trespassers via direct contact with nearsurface soil is not a concern on this portion of the Site. There have been incidents where trespassers have cut the fence and accessed Lots 3 and 4 for the purpose of skateboarding. However, LSS has taken measures to prevent future trespass on this portion of the Site by making it unattractive to skateboarders. For the purposes of this HHRA, it was assumed that the skateboarding scenario has been eliminated by institutional and engineering controls. Therefore, potential exposure of trespassers on Lots 3 and 4 was not quantified.

The current indoor office worker is not likely to have contact with chemicals in surface soil as the area surrounding the existing office building is paved. Groundwater underlying the Site is not a source of drinking water and so no direct contact with groundwater occurs. Future use of

the Site is likely to be industrial, consistent with the current zoning and use of surrounding properties. Assuming that vapor barriers are not included in future building design, vapors may migrate from subsurface soil and groundwater to indoor air. Vapors in indoor air then may be inhaled by workers. Complete inhalation exposure pathways for the current and future indoor worker include:

- Inhalation of vapors migrating from subsurface soil to indoor air
- Inhalation of vapors migrating from groundwater to indoor air.

Based on the results of the COI screening, inhalation of vapors released from subsurface soil is not a complete exposure pathway for outdoor commercial workers. Future Site development may result in mixing of deeper Site soils and some soil may be left exposed. Outdoor occupational workers and construction workers may have contact with exposed surface soil via the exposure pathways listed below:

- Incidental ingestion of soil
- Inhalation of wind-blown dust
- Dermal contact with soil
- Inhalation of vapors from shallow groundwater.

Other complete exposure pathways are those associated with construction of trenches for installation or repair of utilities. As with the outdoor occupational workers, inhalation of vapors from soil is not a complete exposure pathway for excavation workers. Although excavation workers would be expected to wear protective clothing in conformance with a site-specific health and safety plan, it was assumed that protective clothing is not worn and that excavation workers may have direct contact with COPCs in subsurface soil and through inhalation of volatile COPCs from soil and shallow groundwater via the following exposure routes:

- Incidental ingestion of trench soil
- Dermal contact with trench soil
- Inhalation of vapors from shallow groundwater
- Inhalation of resuspended dust.

According to the beneficial water use survey, groundwater will not be used as a source of drinking water, so contact with groundwater by occupational workers at the Arkema Site is not a complete exposure pathway. The depth to groundwater is greater than 15 ft bgs across the majority of the Site, and excavation work is likely to be restricted to depths above the upper saturated zone. Therefore, direct contact with groundwater in a trench is not expected. In the event that groundwater infiltrates a trench, the excavation would be dewatered to prevent direct contact by workers as a health and safety precaution.

4.1.3 Riverbank Area

Currently, much of the riverbank is covered with riprap, invasive scrub vegetation, or other shore-stabilizing materials that prevent direct contact with bank soil. However, trespassers and outdoor occupational workers may contact COPCs in exposed bank soil. Current or future outdoor occupational workers and construction workers may have contact with COPCs in nearsurface riverbank soil, but are not likely to contact subsurface soils (greater than 3 ft bgs). Complete exposure pathways that will be quantified for these outdoor workers include:

- Incidental ingestion of soil
- Inhalation of particulates
- Dermal contact with soil.

Based on the results of the COI screening, inhalation of vapors in ambient air is not considered a complete exposure pathway for workers or trespassers. Also, it was assumed that buildings will not be constructed within Tract A in the future because it is not practical to Site buildings directly on the riverbank. As a result, exposure to vapors migrating from soil and groundwater to indoor air is not considered a complete exposure pathway for this exposure area.

4.2 QUANTIFICATION OF INTAKE

Exposures to COPCs are estimated by calculating an average daily dose or intake of a chemical taken into the body, averaged over an exposure period. Models, equations, and input parameters used to calculate intake of COPCs from incidental ingestion and dermal contact with soil, inhalation of particulates from soil, inhalation of soil vapors in indoor air, and inhalation of vapors from groundwater are described below.

Intake of COPCs through direct contact with exposure media, such as incidental ingestion of soil and dermal contact with soil and groundwater, is estimated using algorithms provided in EPA guidance (USEPA 1989, 2004c). The generalized equation for calculating chemical intakes is as follows:

$$I = \frac{EPC \times CR \times EF \times ED}{BW \times AT} \qquad \text{(Eq. 4-1)}$$

Where:

I = Intake, the amount of chemical taken in by the worker or other person coming into contact with site-related chemicals (mg chemical/kg body weight-day)

EPC	=	Exposure point concentration, the chemical concentration contacted over the exposure period at the exposure point (e.g., mg/kg soil or mg/L groundwater)
CR	=	Contact rate, the amount of affected medium contacted per unit time or event (e.g., soil ingestion rate [mg/day] or air inhalation rate [m^3/hr])
EF	=	Exposure frequency, describes how often exposure occurs (days/year)
ED	=	Exposure duration, describes how long exposure occurs (year)
BW	=	Body weight, the average body weight over the exposure period (kg)
AT	=	Averaging time, period over which exposure is averaged (days).

The variables shown in the above equation are called exposure factors and vary depending on the population who may come into contact with chemicals at the Site. The exposure factors may vary slightly from those listed above depending on the exposure pathway, and chemical-specific factors and unit conversion factors also may be added when necessary. Exposure pathway-specific equations are shown later in this section as 4-1 through 4-3.

For every exposure pathway, it is expected that there will be differences among individuals in the level of exposure due to differences in intake rates, body weights, exposure frequencies, and exposure durations. This results in a wide range of average daily intakes among different members of an exposed population. Typically, risk assessments focus on intakes that are "average" or near the central portion of the range and also on intakes that are near the upper end of the range. These two exposure estimates are called CTE and RME, respectively. The RME case provides a conservative estimate of exposure that is plausible but still well above the average exposure level. Evaluating two exposure conditions provides more complete risk characterization information for risk evaluation and risk management decision-making.

Some of the exposure factors such as exposure duration, exposure frequency, body weight, and averaging time appear in all of the exposure equations, although their values differ depending on the population being evaluated. The following sections describe these exposure factors as well as those that vary by exposure scenario or exposure route. Values for exposure factors were obtained from state and federal risk assessment guidance.

4.2.1 General Intake Assumptions

Some exposure assumptions used to estimate intake are relevant to equations for all exposure routes, including exposure duration, exposure frequency, body weight, and averaging time. Input values for these factors were obtained from EPA and DEQ guidance.

4.2.1.1 Exposure Duration

The exposure duration is the length of time during which someone may be exposed to a particular medium via a specific exposure pathway. The exposure duration varies depending on the population being evaluated. For a typical indoor or outdoor occupational worker, DEQ (2000) and EPA (1991) recommend a RME exposure duration of 25 years. This value is based on U.S. Census data and represents an upper bound estimate for the length of time a person works at the same location. The average, or CTE, value for an occupational exposure duration is 6 years (DEQ 2003, Appendix H.1).

Construction and excavation workers are expected to work on limited-term projects, such as building construction or subsurface utility installation. If multiple construction projects occur on the Site, it is assumed that different workers will participate on each project. The recommended RME and CTE exposure duration for the utility and construction workers is 1 and 0.5 year, respectively (DEQ 2000, 2003).

People who trespass on the Site are assumed to be residents from outlying neighborhoods and so would potentially have contact with site-related media for longer periods than a short-term contract worker. Trespassers at the Site are assumed to be area residents; however, based on the distance from the nearest residential areas and the industrial nature of the intervening development it was determined to be unlikely that children younger than age 9 would gain access to, and trespass at the Site. Based on best professional judgment it was assumed that individuals might trespass at the site from age 9-18. Therefore, the RME and CTE exposure durations for the trespasser are 10 years.

4.2.1.2 Exposure Frequency

Exposure frequency describes how many days someone may have contact with exposure media (e.g., soil or groundwater) in a typical 1-year period. DEQ (2000) and EPA (1991) recommend an upper-bound exposure frequency of 250 days/year for the RME indoor or outdoor occupational scenario. This value is based on a 5-day work week with 10 days off per year for vacation. DEQ (2003) does not provide a CTE value for the onsite worker and so the RME value of 250 days/year was used. Currently, there are no people working full-time on Lots 1 and 2 or the riverbank. Full-time worker exposures for these areas will be evaluated for future scenarios only.

DEQ's (2000) recommended RME and CTE exposure frequency value for the construction worker also is 250 days/year. The excavation worker is expected to spend fewer days on the Site, with a recommended RME and CTE exposure frequency of 9 days/year (DEQ 2000).

Climatological data from the National Weather Service office in Portland, OR was reviewed to determine the most relevant period of exposure for a trespasser. Based on the typical monthly temperatures and precipitation it was assumed that children or teens trespass at the Site 1

day/week during the months of May and September, and 2 days/week during the drier months of June, July, and August for the CTE case. This resulted in a total CTE exposure frequency of 35 days/year. The RME exposure frequency of 70 days/year was based upon an assumption that children or teens access the Site 2 days/week during May and September, and 4 days/week during June, July, and August. Exposure frequencies for the trespasser scenario were adjusted by an exposure time. The exposure time of 1.5 hours/day was based upon the average amount of time spent outdoors by adult residents, as presented in the EPA Exposure Factors Handbook (USEPA 1997a).

4.2.1.3 Body Weight

A value of 70 kg (154 pounds) represents the body weight for all adults, based on average male and female adult body weights (USEPA 1991). A value of 51 kg, the average body weight of males and females age 9-18 years, was assumed for the RME and CTE for the trespasser (USEPA 1997a).

4.2.1.4 Averaging Time

The averaging time is the period over which an exposure is averaged. The averaging times for evaluating carcinogenic and noncarcinogenic effects are different. For evaluating carcinogenic effects, chemical intakes are averaged over a 70-year lifetime (25,550 days) to be consistent with the way cancer slope factors (CSFs) are derived (USEPA 1989). When evaluating noncarcinogenic effects, chemical intakes are averaged over the exposure duration (USEPA 1989). For noncarcinogenic effects, the exposure duration is converted to days and is used as the averaging time. For example, the RME averaging time for the outdoor occupational and indoor worker is 25 years, or 9,125 days; whereas the RME averaging time for the trespasser is 10 years, or 3,650 days.

4.2.1.5 Age-dependent Adjustment Factor

Exposure to some carcinogens more than others early in life will have a greater impact on the risk of developing cancer later in life (USEPA 2005a; DEQ 2007). Due to this finding, DEQ requires that exposure to carcinogenic PAHs during two different life stages (0-2 years and 2-16 years) be modified by age-adjusted potency factors. Because the trespasser scenario is based on a person from ages 9-18, a weighted age-dependent adjustment factor (ADAF) of 2.4 was applied to the lifetime average daily dose (LADD) of PAHs when determining carcinogenic risks for the trespasser. Consistent with DEQ guidance (2007), the weighted ADAF considered an ADAF of 3 to modify the exposure to carcinogenic PAHs during the trespasser life stage of 9-16 years (years) and an ADAF of 1 to represent exposures for the trespasser life stage of 16-18 years (3 years). The weighted ADAF was applied to both RME and CTE scenarios.

4.2.2 Intake via Soil Ingestion

The algorithm and assumptions for estimating intake of COPCs via incidental ingestion of soil are provided in Exhibit 4-1. Discussion of soil ingestion rates is provided below. Incidental soil ingestion rates for direct exposures to soil vary based on several factors, including the frequency of an individual's hand-to-mouth contact, ground cover (e.g., grass vs. bare ground), amount and type of outdoor activity, and personal hygiene practices (e.g., frequency of hand washing).

Current soil screening guidance (USEPA 2002b) and DEQ risk assessment guidance (2000) recommend default soil ingestion rates of 100 mg/day and 330 mg/day, respectively, for outdoor workers and construction workers. According to EPA guidance, the outdoor worker is defined as:

> …a long-term receptor exposed during the work day who is a full time employee of the company operating on-site and who spends most of the workday conducting maintenance activities outdoors. The activities for this receptor (e.g., moderate digging, landscaping) typically involve on-site exposures to surface and shallow subsurface soils (at depths of 0 to 2 feet). The outdoor worker is expected to have an elevated soil ingestion rate (100 mg per day) and is assumed to be exposed to contaminants via the following pathways: incidental ingestion of soil, dermal absorption of contaminants from soil, inhalation of fugitive dust, inhalation of volatiles outdoors, and ingestion of ground water contaminated by leachate.[2,3] The outdoor worker is expected to be the most highly exposed receptor in the outdoor environment under commercial/industrial conditions.

In contrast, the construction worker is:

> …a short-term adult receptor who is exposed to soil contaminants during the work day for the duration of a single construction project (typically a year or less). If multiple non-concurrent construction projects are anticipated, it is assumed that different workers will be employed for each project. The activities for this receptor typically involve substantial on-site exposures to surface and subsurface soils. The construction worker is expected to have a very high soil ingestion rate and is assumed to be exposed to contaminants via the following direct and indirect pathways: incidental soil ingestion, dermal absorption, inhalation of volatiles outdoors, and inhalation of fugitive dust.

EPA's construction worker rate of 330 mg/day represents the 95th percentile value for adult soil intake rates reported in a soil ingestion tracer-based mass-balance study by Stanek et al. (1997). The 95th percentile estimate is based on use of the median of the best four trace elements (from Al, Si, Ti, Y, and Zr) and the lowest food/soil ratios over the 4-week observation period. In an effort to achieve the best estimates of adult soil ingestion, Stanek et al. (1997) uses the median of the four best trace elements, which is intended to account for some source error attributed to highly variable tracer-specific estimates for the same adults and time periods. Nonetheless, the authors report considerable uncertainty with regard to the best estimate, stating:

> In the judgment of the authors, estimates based on the median of the best four trace elements are most likely to be best. Such estimates indicate that in over half of the study weeks, no soil was ingested by the adults. The average daily soil ingestion (taken over 10 subjects over 4 weeks) was 6 mg/day. The 95th percentile soil ingestion estimate was 331 mg/day, but based on present data, it is substantially uncertain.

EPA notes that this value replaces an earlier default ingestion rate of 480 mg/day (Hawley 1985) that was based on a theoretical calculation for adults engaged in outdoor physical activity[7]. They provide no rationale for selection of this "high-end soil ingestion rate" (USEPA 2002b), nor do they comment on the limitations of the Stanek et al. (1997) study given the small sample size (n=10) and lack of reported information regarding the nature of soil contact activities engaged in by study participants (i.e., the applicability of these activities to the construction worker scenario). They do, however, note that continued research in soil ingestion rates for adults engaged in strenuous activities may result in future changes to this value.

Despite uncertainty associated with adult soil ingestion rates due to a lack of scientific information, default EPA and DEQ values were selected to represent the RME and CTE worker scenarios. Soil ingestion rates of 100 mg/day and 330 mg/day were used for the RME outdoor occupational and construction worker scenarios, respectively (USEPA 2002b, DEQ 2000). A soil ingestion rate of 330 mg/day also was selected for the short-term excavation worker. DEQ (2003) provides central estimates of incidental soil ingestion of 50 mg/day and 100 mg/day for the outdoor occupational worker and construction worker, respectively. A CTE soil ingestion rate of 100 mg/day also is recommended for the excavation worker (DEQ 2003).

A soil ingestion rate of 100 mg/day for the trespasser was obtained from DEQ guidance (2000). The value is recommended for the RME scenario for residential results.

4.2.2.1 Bioavailability

Relative absorption varies among metals. Relative absorption factors (RAFs) for metals in all exposure areas were assumed to be equal to 1.0. In the case of arsenic, oral toxicity values for inorganic arsenic are based on studies of human populations exposed to dissolved arsenic naturally present in drinking water. Arsenic dissolved in water is almost completely absorbed (ATSDR 2000a). Arsenic is soil is typically one-tenth to one-half as bioavailable as arsenic dissolved in water (i.e., the RAF would range from 0.1 to 0.5) (Kelley et al. 2002). At a former smelter site in Anaconda, Montana, arsenic in soil was reported to have an RAF of 0.20 (Freeman et al. 1995). For the soil ingestion pathway at this Site, arsenic in soil was assumed to

[7] An underlying assumption in the Hawley (1985) soil ingestion calculation is a soil adherence rate of 3.5 mg/cm^2 for an individual engaged in outdoor work for extended periods of time and that such an individual ingests "twice daily a quantity of soil corresponding to one half the covering of the inside surface of the fingers and thumbs of both hands." Current EPA guidance (2002b) recommends a 95th percentile soil adherence rate of 0.3 mg/cm^2 for workers engaged in outdoor construction activities.

have a relative bioavailability of 1.0, which is likely to result in an overestimate of potential risks associated with arsenic.

Exhibit 4-1. Incidental Ingestion of Soil

Equation:

$$Intake_{soil} = \frac{EPC_{soil} \times IR_{soil} \times EF \times ED}{AT \times BW \times CF} \qquad \text{(Eq. 4-2)}$$

Where:

$Intake_{soil}$	=	Average daily dose from incidental ingestion of soil (mg/kg-day)
EPC_{soil}	=	Concentration of chemical in soil (mg/kg)
IR_{soil}	=	Soil ingestion rate (mg/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
AT	=	Averaging time (days)
BW	=	Body weight (kg)
CF	=	Unit conversion factor (1E+06 mg/kg).

Exhibit 4-1 Parameter Values

Exposure Factor	Value		Units	Source
	CTE	RME		
Outdoor Occupational Worker				
IR_{soil}	50	100	mg/day	DEQ 2000, USEPA 2002b
EF	250	250	days/year	DEQ 2000, 2003; USEPA 1991
ED	6	25	years	USEPA 1991, DEQ 2003
AT	see text	see text	days	see text
BW	70	70	kg	USEPA 1991
Construction Worker				
IR_{soil}	100	330	mg/day	DEQ 2003, USEPA 2002b
EF	250	250	days/year	DEQ 2000
ED	0.5	1	years	DEQ 2000, 2003
AT	see text	see text	days	see text

Exposure Factor	Value		Units	Source
	CTE	RME		
BW	70	70	kg	USEPA 1991
Excavation Worker				
IR_{soil}	100	330	mg/day	DEQ 2003
EF_{soil}	9	9	days/year	DEQ 2000
ED	0.5	1	years	DEQ 2000, 2003
AT	see text	see text	days	see text
BW	70	70	kg	USEPA 1991
Trespasser				
IR_{soil}	100	100	mg/day	DEQ 2000
EF_{soil}	35	70	days/year	see text
ED	10	10	years	see text
AT	see text	see text	days	see text
BW	51	51	kg	USEPA 1997a

4.2.3 Intake via Dermal Contact with Soil

The algorithms and assumptions used to estimate dermal absorption of COPCs in soil are presented in Exhibit 4-2. Additional discussion is provided below for values selected for skin surface area, soil-to-skin adherence, event frequency, and dermal absorption.

4.2.3.1 Skin Surface Area

Dermal exposure to chemicals is affected by the skin surface area coming into contact with soil. EPA (2004c) provides recommendations for skin surface area that may have contact with soil. Skin surface area estimates are based on population mean values for women and men combined.

EPA recommends that exposure scenarios for worker contact with soil assume that skin contact is limited to the head, hands, and forearms, which have a total surface area of 3,300 cm^2 (USEPA 2004c). DEQ (2003) supports EPA's recommendation for evaluation of outdoor occupational, construction, and excavation workers. A value of 3,300 cm^2 was used for RME and CTE worker scenarios.

Data for males and females 9-18 years of age were used to estimate dermal exposure for trespassers. The median exposed skin surface area of 4,800 cm^2 was used under CTE conditions, and the upper 90th percentile skin surface area of 5,600 cm^2 was used for the RME

scenario (USEPA 1997a). These values are based upon an assumption that the head, hand, forearms, and lower legs would not be covered by clothing.

4.2.3.2 Soil Adherence Factor

The adherence factor describes the amount of soil that sticks to the skin. It is expressed in terms of soil mass per unit surface area (mg/cm^2). Soil adherence to skin is influenced by the properties of the soil, the part of the body exposed, and the activity of the exposed individual (USEPA 2004c).

Adherence factors were selected from DEQ (2003) guidance. DEQ recommends default soil adherence factors for the outdoor occupational worker of 0.02 and 0.1 mg/cm^2 for the CTE and the RME cases, respectively (DEQ 2003). The CTE value is a geometric mean derived for commercial groundskeepers, and the RME value is a geometric mean derived for a construction worker.

DEQ (2003) recommends an adherence factor of 0.1 and 0.3 mg/cm^2 for the CTE and RME construction and excavation worker scenarios. The CTE is a geometric mean derived for a construction worker, and the RME is a 95th percentile derived for a construction worker.

For the trespasser, the recommended soil adherence factor for a residential adult of 0.07 mg/cm^2 was selected for both the RME and CTE scenarios (DEQ 2003). This value is a mean soil adherence value for a residential adult gardener. This is expected to be a conservative soil adherence factor for a trespasser, since it is not considered likely that trespassers will be engaged in digging activities on the Site.

4.2.3.3 Event Frequency

Event frequency is the number of times during one day that someone may have contact with soil. This exposure factor applies only to the dermal exposure route (Exhibit 4-2). EPA (2004c) recommends the assumption that each contact event occurs one time per day. This assumption was selected for both the CTE and RME cases in all scenarios.

4.2.3.4 Dermal Absorption Factors

The dermal absorption factor represents the proportion of chemical in soil that is absorbed across the skin. Values for these factors were obtained from EPA's *RAGS E, Supplemental Guidance for Dermal Risk Assessment* (2004c) and the Oak Ridge National Laboratory's Risk Assessment Information System (RAIS; 2007). Chemical-specific factors for estimating dermal absorption are:

- Arsenic: 0.03 (USEPA 2004c)

- Chromium: 0.001 (RAIS 2007)
- Aroclors: 0.14 (USEPA 2004c)
- PAHs: 0.13 (USEPA 2004c)
- DDT: 0.03 (USEPA 2004c)
- DDD, DDE: 0.01 (RAIS 2007)
- Dioxins/furans: 0.03 (USEPA 2004c)
- PCP: 0.25 (USEPA 2004c)
- VOCs: 0.01 (RAIS 2007)
- Semi-volatile organic compounds: 0.1 (USEPA 2004c).

The dermal absorption value for Aroclor 1254 provided by EPA (2004c) was applied to Aroclor 1248 and 1260. EPA (2004c) does not provide default dermal absorption factors for chromium, DDT metabolites, or VOCs. For many inorganic compounds, including chromium, insufficient data are available to develop a default absorption value and volatile compounds are expected to volatilize from the soil and skin, so exposures to volatiles are better evaluated via the inhalation pathway.

Exhibit 4-2. Dermal Contact with Soil

Equation:

$$DAD_{soil} = \frac{DA_{event} \times EF \times ED \times EV \times SA}{AT \times BW} \qquad \text{(Eq. 4-3)}$$

Where:

DAD_{soil}	=	Dermal absorbed dose (mg/kg-day)
DA_{event}	=	Absorbed dose per event (mg/cm^2-event)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
EV	=	Event frequency (event/day)
SA	=	Skin surface area available for contact (cm^2)
AT	=	Averaging time (days)
BW	=	Body weight (kg).

And

$$DA_{event} = EPC_{soil} \times CF \times AF \times ABS_d \qquad \text{(Eq. 4-4)}$$

Where:

DA_{event}	=	Absorbed dose per event (mg/cm^2-event)
EPC_{soil}	=	Concentration of chemical in soil (mg/kg)
CF	=	Unit conversion factor (1E-06 kg/mg)
AF	=	Adherence factor (mg/cm^2-event)
ABS_d	=	Dermal absorption fraction, chemical-specific (unitless).

Exhibit 4-2 Parameter Values

Exposure Factor	Value		Units	Source
	CTE	RME		
Outdoor Occupational Worker				
AF	0.02	0.1	mg/cm^2	DEQ 2003
EF	250	250	days/year	DEQ 2000, USEPA 1991
ED	6	25	years	USEPA 1991, DEQ 2003
EV	1	1	event/day	USEPA 2004c
SA	3,300	3,300	cm^2	DEQ 2003, USEPA 2004c
AT	see text	see text	days	see text
BW	70	70	kg	USEPA 1991
Construction Worker				
AF	0.1	0.3	mg/cm^2	DEQ 2003
EF	250	250	days/year	DEQ 2000
ED	0.5	1	years	DEQ 2000, 2003
EV	1	1	event/day	USEPA 2004c
SA	3,300	3,300	cm^2	DEQ 2003, USEPA 2004c
AT	see text	see text	days	see text
BW	70	70	kg	USEPA 1991
Excavation Worker				
AF	0.1	0.3	mg/cm^2	DEQ 2003
EF	9	9	days/year	DEQ 2000

Exposure Factor	Value		Units	Source
	CTE	RME		
ED	0.5	1	years	DEQ 2000, 2003
EV	1	1	event/day	USEPA 2004c
SA	3,300	3,300	cm^2	DEQ 2003, USEPA 2004c
AT	see text	see text	days	see text
BW	70	70	kg	USEPA 1991
Trespasser				
AF	0.07	0.07	mg/cm^2	DEQ 2003
EF	35	70	days/year	see text
ED	10	10	years	see text
EV	1	1	event/day	USEPA 2004c
SA	4,800	5,600	cm^2	USEPA 1997a
AT	see text	see text	days	see text
BW	51	51	kg	USEPA 1997a

4.2.4 Intake via Inhalation of Particulates from Soil and Vapors from Groundwater in Outdoor Air

The algorithm and assumptions used to estimate inhalation of COPCs in soil as resuspended dust and vapors from groundwater are presented in Exhibit 4-3. Note that in addition to inhalation of particulates from soil and vapors from groundwater by outdoor workers, the excavation worker may inhale vapors from shallow groundwater while working in a trench.

For the purposes of the HHRA, inhalation is expressed as a daily rate (m^3/day). DEQ (2003) recommends an inhalation rate of 7 m^3/day for the CTE and RME outdoor occupational, construction, and excavation worker scenarios. The RME and CTE trespasser inhalation rates are 3.5 m^3/day and 2.1 m^3/day, respectively (USEPA 1997a). These inhalation rates are based upon an assumed exposure time of 1.5 hours/day and are representative of light to moderate activity that would be expected for a trespasser on the Site.

Exhibit 4-3. Inhalation of Particulates from Soil and Vapors from Groundwater in Outdoor Air

Equation:

$$Intake_{inh} = \frac{EPC_{part-vap} \times InhR \times EF \times ED}{AT \times BW} \qquad \text{(Eq. 4-5)}$$

Where:

$Intake_{inh}$	=	Intake from inhalation of particulates in outdoor air (mg/kg-day)
$EPC_{part\text{-}vap}$	=	Concentration of chemical in particulates (mg/m^3) = EPC_{soil} ÷ PEF, where PEF = particulate emission factor (kg/m^3), default value of 1.58E+10 kg/m^3 (DEQ 2003) or in vapor (mg/m^3) = EPC_{gw} * VF, where VF = volatilization factor (L/m^3) (DEQ 2003)
InhR	=	Inhalation rate (m^3/day)
EF	=	Exposure frequency (days/year)
ED	=	Exposure duration (years)
AT	=	Averaging time (days)
BW	=	Body weight (kg).

Exhibit 4-3 Parameter Values

Exposure Factor	Value		Units	Source
	CTE	RME		
Occupational Worker				
InhR	7	7	m^3/day	DEQ 2003
EF	250	250	days/year	DEQ 2000, 2003; USEPA 1991
ED	6	25	years	USEPA 1991, DEQ 2003
AT	see text	see text	days	see text
BW	70	70	kg	USEPA 1991
Construction Worker				
InhR	7	7	m^3/day	DEQ 2003
EF	250	250	days/year	DEQ 2000
ED	0.5	1	years	DEQ 2000, 2003
AT	see text	see text	days	see text
BW	70	70	kg	USEPA 1991
Excavation Worker				
InhR	7	7	m^3/day	DEQ 2003
EF	9	9	days/year	DEQ 2000
ED	0.5	1	years	DEQ 2000, 2003
AT	see text	see text	days	see text

Exposure Factor	Value		Units	Source
	CTE	RME		
BW	70	70	kg	USEPA 1991
Trespasser				
InhR	2.1	3.5	m^3/day	USEPA 1997a
EF	35	70	days/year	see text
ED	10	10	years	see text
AT	see text	see text	days	see text
BW	51	51	kg	USEPA 1997a

4.3 LEAD INTAKE ASSESSMENT

USEPA (2003a) recommends use of a toxicokinetic model called the adult lead methodology (ALM) to assess exposures of adults to lead. The ALM is based on the assumption that soil is the main source of lead exposures at contaminated sites. The ALM includes a module to predict fetal blood lead levels to support estimates of exposures that are protective of a "fetus of a worker who develops a body burden as a result of non-residential exposure to lead." According to USEPA (2003a), protection of the fetus is the most health-sensitive endpoint for adults. The ALM target risk level is no more than a 5 percent probability that a fetus exposed to lead will exceed a blood lead level of 10 μg/dL (USEPA 2003a).

The ALM uses a technical approach described by Bowers et al. (1994) that predicts the blood lead level in an adult with a site-related lead exposure by summing the "baseline" blood lead level (PbB_0) (i.e., that which would occur in the absence of any site-related exposures) with the increment in blood lead concentration that is expected as a result of increased exposure due to contact with lead-contaminated soil at the Site. The latter is estimated by multiplying the average daily absorbed dose of lead from soil by a biokinetic slope factor (BKSF). Thus, the basic equation for exposure to lead in soil is:

$$PbB = PbB_0 + BKSF \times \left(\frac{PbS \times IR_s \times AF_s \times EF_s}{365} \right) \qquad \text{(Eq. 4-6)}$$

Where:

PbB = Geometric mean blood lead concentration (μg/dL) in women of childbearing age that are exposed at the Site

PbB_0 = "Background" geometric mean blood lead concentration (μg/dL) in women of childbearing age in the absence of exposures to Site soil

BKSF = Biokinetic slope factor (μg/dL blood lead increase per μg/day lead absorbed)

PbS = Soil lead concentration (μg/g)

IR_s = Intake rate of soil, including both outdoor soil and indoor soil-derived dust (g/day)

AF_s = Absolute gastrointestinal absorption fraction for lead in soil and dust (dimensionless). The value of AF_s is given by:

$$AF_s = AF_{soluble} \times RBF_{soil/soluble} \qquad \text{(Eq. 4-7)}$$

Where:

$AF_{soluble}$ = absorption factor for soluble lead

$RBF_{soil/soluble}$ = Relative bioavailability of lead in soil compared to soluble lead (dimensionless).

EF_s = Exposure frequency for contact with site soils and dusts (days per year)

If a factor K_{SD} (which represents a mass fraction of soil in dust) and a weighting factor W_s (which represents the fraction of IR_s ingested as outdoor soil) are introduced to the basic equation for exposure to lead in soil, the basic equation can be rearranged as follows (USEPA 2003a):

$$PbB = PbB_0 + BKSF \times PbS \times \frac{\left[\left(IR_s \times AF_s \times EF_s \times W_s\right) + \left(K_{SD} \times IR_s \times \left(1 - W_s\right) \times AF_s \times EF_s\right)\right]}{365}$$

(Eq. 4-8)

Where:

K_{SD} = Mass fraction of soil in dust (dimensionless)

W_s = Fraction of IR_s ingested as outdoor soil (dimensionless).

This rearranged equation was used for adult blood lead modeling for this study.

Once the geometric mean blood lead value is calculated, the full distribution of likely blood lead values in the population of exposed people can then be estimated by assuming the distribution

is lognormal with a specified individual geometric standard deviation (GSD_i). The 95th percentile of the predicted distribution is given by the following equation from Aitchison and Brown (1957):

$$95^{th}\ percentile = GM \times GSD_i^{1.645} \qquad \text{(Eq. 4-9)}$$

Where:

GM = Geometric mean

GSD_i = Individual geometric standard deviation.

The ALM model uses the following equations to calculate the mean and 95th percentile of fetal blood concentrations:

$$PbB_{fetal} = R_{fetal-maternal} \times PbB_{adult} \qquad \text{(Eq. 4-10)}$$

$$PbB_{fetal,0.95} = R_{fetal/maternal} \times PbB_{adult,central} \times GSD_{i,adult}^{1.645} \qquad \text{(Eq. 4-11)}$$

Where:

PbB_{fetal} = Fetal blood lead concentration (µg/dL) (which, like PbB_{adult}, is a variable quantity having the specified probability distribution)

$R_{fetal/maternal}$ = Constant of proportionality between fetal and maternal blood lead concentrations

PbB_{adult} = Adult blood lead concentration (µg/dL), estimated with parameters appropriate to women of child bearing age

$PbB_{fetal,\ 0.95}$ = Fetal blood lead concentration (µg/dL) among fetus born to women having exposure to the specified site.

There is evidence that fetal blood lead concentrations are consistently lower than maternal blood lead concentrations by a factor of 0.9. Consequently, for this study the ALM default value, 0.9, for $R_{fetal/maternal}$ was used. The use of a point estimate for this value implies a deterministic (nonrandom) relationship between maternal and fetal blood lead concentrations. This assumption omits a source of variability (varying individual-specific ratios of fetal to maternal blood lead) that would tend to increase the variance of fetal blood lead concentrations (USEPA 2003a).

Model input values for soil ingestion rate, exposure frequency, and averaging time were the same as those provided in Exhibit 4-1, above. Other model inputs are described below. The full

set of input parameters and the results of the ALM used in this HHRA are provided in Table 6-27.

4.3.1 Soil Parameters

The soil EPCs used for the ALM were the geometric mean concentrations for each exposure area as recommended by USEPA, rather than the UCL90 used for the other soil related exposures. A sensitivity analysis was conducted to evaluate the impact in the ALM results from using the UCL90. This analysis is reported in the Uncertainty Section of this report. The ALM includes an assumption that indoor dust lead concentrations are due to tracking in lead-containing soil into homes. A weighting factor, W_s, was used to apportion the soil ingestion rate between outdoor soil and indoor dust. It was assumed that 100 percent of the soil ingested is from outdoor soil because the outdoor occupational worker, excavation worker, and construction worker are not assumed to work indoors (i.e., a weighting factor of 1.0).

EPA assumes that the absorption fraction for lead from soil is 0.12 (USEPA 2003a). This parameter represents the fraction of lead in soil ingested daily that is absorbed from the gastrointestinal tract. The default value is based on an absorption factor for soluble lead ($AF_{soluble}$) of 0.2 and a relative bioavailability of lead in soil compared to soluble lead ($RBF_{soil/soluble}$) of 0.6. The default value of 0.2 for $AF_{soluble}$ represents a weight-of-evidence (WOE) determination from various experimental studies, and considers major sources of variability that are likely to be present in human populations.

4.3.2 Biokinetic Slope Factor

EPA adopts a default biokinetic slope factor (BKSF) of 0.4 μg/dL per μg/day. The value is derived using data from Pocock et al. (1983) on lead concentrations in tap water and blood lead levels in 910 adult males, an absorption factor for lead in drinking water (AF_w), and assumptions regarding drinking water patterns. Using the same data and different assumptions regarding drinking water intake, lead absorption, and drinking water patterns, Bowers and Cohens (1998) derived a BKSF of 0.375 μg/dL per μg/day. There are some uncertainties regarding the extrapolation between the study population and exposed population of interest (women of child bearing age) and the assumption of linearity between lead intake and blood lead concentration. Experimental data on the pharmacokinetics of lead in human adults described by EPA (2003a) support the default value of 0.4 μg/dL per μg/day. This default value was applied to the ALM in this risk assessment.

4.3.3 Blood Parameters

The baseline blood lead concentration is intended to represent the best estimate of a reasonable central value of blood concentration in women of childbearing age that are not exposed to lead-contaminated soil in the study area (AGEISS 1996). In cases in which site-specific blood

lead is not available, the ALM guidance recommends that results of Phase I and II of the *National Health and Nutrition Survey (NHANES III)* be used in combination with information on the specific demographic or geographic characteristics of the Site (USEPA 2002c). Estimated geometric means and standard deviations for women ages 17-45 in the Western US were assumed for PbB_0, and GSD_i for women of child bearing age at the Site. A PbB_0 of 1.4 μg/dL and GSD_i of 2.11 were used for CTE and RME scenarios.

5 TOXICITY ASSESSMENT

The purpose of a toxicity assessment is to summarize health effects that may be associated with exposure to the chemicals included in the risk assessment and to identify doses that may be associated with those effects. The focus is on effects associated with long-term exposures and on effects that could be associated with the chemical concentrations and pathways of exposure that are relevant in environmental settings. Toxicity values developed based on dose-response assessments for these relevant adverse effects are identified. These toxicity values are numerical expressions of chemical dose and response, and vary based on factors such as the route of exposure (e.g., oral or inhalation) and duration of exposure.

Toxicity values for carcinogenic and noncarcinogenic health effects have been developed for many chemicals by government agencies, including EPA and the U.S. Agency for Toxic Substances and Disease Registry (ATSDR). Chemicals being evaluated in this risk assessment include two PCB mixtures (Aroclor 1248 and Aroclor 1260), carcinogenic PAHs (assessed using the toxicity value for BaP), metals (arsenic, lead, and chromium), chlorinated pesticides (4,4-DDT, 4,4-DDD, and 4,4-DDE), chlorinated solvents, and DROs.

Duration of exposure is an important factor because the exposure levels that can cause toxic effects are usually lower when exposures continue for a longer period of time. For example, with continuous exposure to a chemical for many years (typically referred to as chronic exposure), much lower concentrations (and resulting doses) of a chemical could be associated with toxic effects, compared with concentrations that would be identified as causing toxic effects in a person who is exposed to a chemical for only 1 day (referred to as an acute exposure). Intermediate duration exposures (referred to as subchronic exposures) are more likely to suggest toxic effects at intermediate concentrations. This HHRA evaluates risks associated with potential exposures to COPCs on and around the Site for scenarios involving chronic exposures, which represent a duration of more than 7 years, and subchronic exposures, which represent a duration ranging from 2 weeks to 7 years; acute exposures, which have a duration of less than 2 weeks, are not considered. This approach is health-protective because the concentrations of COPCs in the environment that can lead to chronic or subchronic effects are typically much lower than those that result in acute effects.

The following section describes the procedures used to identify and assess toxicity information and provides an overview of toxicity values selected. Subsequent sections briefly summarize the basis for the toxicity values for the chemicals evaluated and describe some of the key methodological issues, assumptions, and uncertainties that underlie the toxicity information. Additional discussion is provided for the approach used to assess the toxicity of mixtures of carcinogenic PAHs.

5.1 METHODS FOR TOXICITY ASSESSMENT

Standard procedures were followed per EPA (1989) to identify and assess toxicity factors and other relevant toxicity information, such as the WOE category for carcinogenic potential. As recommended in a recent EPA memorandum, *Human Health Toxicity Values in Superfund Risk Assessments* (USEPA 2003b), the primary sources that were consulted for toxicity values are, in order of priority, EPA's Integrated Risk Information System (IRIS) and EPA's Provisional Peer Reviewed Toxicity Values (PPRTVs) from the National Center for Environmental Assessment/Superfund Health Risk Technical Support Center. If neither IRIS toxicity values nor PPRTVs were available, then toxicity values were obtained from other documented sources, such as ATSDR, minimal risk levels (MRLs), Oak Ridge National Laboratory's Risk Assessment Information System RAIS, and EPA's Health Effects Assessment Summary Tables (HEAST).

The following two subsections describe the toxicity values used to assess noncancer and carcinogenic effects of chemicals.

5.1.1 Noncancer Effects

The potential for noncancer health effects from long duration or chronic exposures (i.e., greater than 7 years) is evaluated by comparing the estimated daily intake with a chronic oral or inhalation reference dose (RfD). These toxicity values represent average daily exposure levels at which no adverse effects are expected to occur with chronic exposures. Subchronic RfDs are applied when exposures are less than 7 years, as is the case with construction and excavation workers (i.e., <1 year). RfDs reflect the underlying assumption that systemic toxicity occurs as a result of processes that have a threshold (i.e., that a safe level of exposure exists and that toxic effects will not be observed until this level has been exceeded).

The RfDs for many noncarcinogenic effects are generally derived based on laboratory animal studies or epidemiological studies in humans. In such studies, the RfD is typically calculated by first identifying the highest concentration or dose that does not cause observable adverse effects (the no-observed-adverse-effect level, or NOAEL) in the study subject. If a NOAEL cannot be identified from the study, a lowest-observed-adverse-effect level (LOAEL) may be used. This dose or concentration is then divided by uncertainty factors to calculate a RfD.

The uncertainty factors are applied to account for limitations of the underlying data and are intended to ensure that the toxicity value calculated based on the data will be unlikely to result in adverse health effects in exposed human populations. For example, an uncertainty factor of 10 is used to account for interspecies differences (if animal studies were used as the basis for the calculation), and another factor of 10 is used to address the potential that human subpopulations such as children or the elderly may have increased sensitivity to the chemical's adverse effects. Thus, variations in the strength of the underlying data are reflected in the uncertainty factors

used to calculate the toxicity values and in the low, medium, or high confidence ratings assigned to those values (USEPA 2007b).

For cases in which toxicity values are not available for an exposure route being evaluated, existing values for other routes may be adjusted and used. For example, no RfDs are available for assessing the dermal exposure route. Oral toxicity values are typically used instead. Because oral toxicity values are usually derived from administered doses, while dermal exposure estimates are expressed as absorbed doses, the oral toxicity values must be adjusted to reflect absorbed dose. This adjustment is accomplished by multiplying the oral RfD by the chemical-specific oral absorption rate.

Route-to-route extrapolation assumes that once a chemical is absorbed into the bloodstream, the health effects are similar regardless of whether the route of exposure is oral, dermal, or inhalation. This assumption may be valid for some chemicals with pharmacokinetic characteristics that are similar regardless of route of administration; however, for many chemicals, factors such as absorption, metabolism, distribution, and elimination vary by exposure route, leading to substantial differences in toxicity.

The toxicity values used to estimate potential noncancer hazards in the HHRA are summarized in Table 5-1 for oral and inhalation exposure routes. Noncancer toxicity values for the dermal exposure pathway are provided in Table 5-2.

5.1.2 Carcinogenic Effects

To assess carcinogenic health effects, CSFs are used for oral and dermal exposures, while unit risk factors (URFs) are used for inhalation exposures. CSFs and URFs are upper-bound estimates of the carcinogenic potency of chemicals. They are used to estimate the incremental risk of developing cancer, corresponding to a lifetime of exposure at the levels described in the exposure assessment. In standard risk assessment procedures, estimates of carcinogenic potency reflect the conservative assumption that no threshold exists for carcinogenic effects (i.e., that any exposure to a carcinogenic chemical will contribute an incremental amount to an individual's overall risk of developing cancer). The specific CSF and URF values described in Section 4.2 are conservative upper-bound estimates of potential risk. In this HHRA, URF values also are provided as inhalation CSFs.

Another component of assessing carcinogenic health effects is a qualitative evaluation of the extent to which a chemical is a human carcinogen. For most chemicals listed in IRIS, this evaluation was conducted by EPA using a classification system called WOE determination.[8] A chemical is assigned a WOE classification based on data obtained from both human and animal

[8] The WOE categories described in the final *Guidelines for Carcinogen Risk Assessment* (USEPA 2005b) as "standard hazard descriptors" differ from and may eventually supersede those used in IRIS (USEPA 2007b). These descriptors include "carcinogenic to humans," "likely to be carcinogenic to humans," "suggestive evidence of carcinogenic potential," "inadequate information to assess carcinogenic potential," and "not likely to be carcinogenic to humans."

studies. Once a WOE is assigned to a chemical, a CSF, URF, or both are derived. Chemicals for which EPA considers adequate human data indicating carcinogenicity are available are categorized as "known human carcinogens" (WOE class A), while other chemicals with various levels of supporting data may be classified as "probable human carcinogens" (WOE class B1 or B2), or "possible human carcinogens" (WOE class C). Where EPA considers that data are inadequate for determining carcinogenicity, the chemical is "not classifiable as to human carcinogenicity" (WOE class D). When studies provide evidence of noncarcinogenicity, a chemical is assigned a WOE class E (USEPA 2007b).

The toxicity values and WOE used to estimate potential cancer risks in the HHRA are summarized in Table 5-2 for the dermal exposure route and Table 5-3 for oral and inhalation exposure routes.

5.1.3 Relative Potency Approach for PAHs

The cancer potencies of individual carcinogenic PAH chemicals are expressed relative to the cancer potency of BaP. This procedure involves applying chemical-specific relative potency factors (RPFs) to the CSF for BaP, resulting in a CSF adjusted for the toxicity of each PAH relative to BaP. Table 5-4 presents the RPFs provided by USEPA (1993).

Despite wide use RPFs in health risk assessments at Superfund and Resource Conservation and Recovery Act (RCRA) sites to express the toxicity of carcinogenic PAHs in relation to the toxicity of BaP, numerous limitations of its use have been identified. These limitations contribute to uncertainty in the estimation of risks for the Site. In 2002, a panel of experts in PAH toxicology and chemistry and risk assessment of chemical mixtures were convened by the National Center for Environmental Assessment to evaluate available approaches to PAH health risk assessment (USEPA 2002d). With regard to their evaluation of the RPF approach, these experts noted the following major issues: 1) the assumption that individual PAH risks are additive may not be accurate; 2) human toxicity data on individual PAHs are lacking; and 3) the approach is not based on a reference PAH mixture with known animal or human toxicity (USEPA 2002d).

In particular, with regard to additivity and interactions, uncertainty arises because some PAHs can both initiate and promote tumors. An approach in which the toxicities of individual PAHs are defined relative to BaP is complicated by the ability of PAH components to act via distinct pathways to produce a single endpoint of cancer (Reeves et al. 2001). Further, because PAH interactions in mixtures are concentration-dependent, the kinds of interactions observed in experimental settings using high concentrations of PAHs may not be the same as those occurring with lower levels more typical of environmental settings (USEPA 2002d). Thus, a large amount of uncertainty may be introduced when attempts to predict responses of biological systems due to mixtures are based on relative potency of a single PAH.

5.1.4 Toxicity Equivalency Approach for PCDDs/PCDFs

PCDDs and PCDFs are two groups of structurally similar, tricyclic, almost planar, organic compounds that exhibit similar physical and chemical properties. There are 75 dioxins and 135 furans, called congeners, which are differentiated by their number and position of chlorine atoms. Researchers in the early 1980s concluded that a subset of PCDDs, PCDFs, and PCB congeners shared a common mechanism of action and induced comparable biological and toxic responses (USEPA 2003d). However, the potency of the different congeners varies considerably. As discussed earlier, the chlor-alkali process, which is the source of PCDD/Fs from site-related activities, will result in a furan dominated profile in which dioxins are below detection limits (Rappe et.al. 1991).

Seventeen PCDD and PCDF congeners (7 PCDDs, 10 PCDFs) exhibit what is termed "dioxin-like" toxicity. These 17 congeners have chlorine atoms present in the 2, 3, 7, and 8 positions on the ring structure of the molecule and are more toxic than other congeners with fewer chlorine atoms or with chlorine atoms in different positions on the ring structure. The congener 2,3,7,8- TCDD is the most widely studied and has been found to exhibit the most potent toxic response. Similarly, 12 coplanar PCB congeners have been shown to exhibit dioxinlike toxicity and are grouped with the 17 dioxin/furan congeners that exhibit toxicity similar to TCDD (USEPA 2003c).

Human health risk estimates for exposures to PCDDs/PCDFs traditionally require conversion of concentrations of individual dioxin and furan congeners to their 2,3,7,8-TCDD TEQ concentration using congener-specific TEFs. The 2,3,7,8-TCDD TEQ concentration for each sample is calculated by multiplying concentrations of individual congeners by their congener-specific TEFs, and summing the results for all congeners. The 2,3,7,8-TCDD TEQ concentration is assumed to express the total potency of the mixture of PCDDs/PCDFs in a sample to exert the toxicity of 2,3,7,8-TCDD.

For assessment of human health risks, TEFs developed by the World Health Organization (Van den Berg et al. 1998) and recommended by EPA (USEPA 2003c) were used to calculate TEQs. These TEFs, which are the most widely accepted equivalency factors and are typically expressed as "WHO98 TEFs," are shown in Table 5-4.

5.2 TOXICITY PROFILES

The following text provides a general summary of the noncancer and cancer toxicity values for each COPC.

5.2.1 PAHs: Benzo(a)pyrene

Although the PAH family consists of over 100 chemicals, there is a core group of about 17 chemicals that are routinely analyzed in environmental samples. Noncancer oral toxicity values (RfDs) are available for eight of these PAHs, none of which is present on the Arkema Site.[9]

Seven of the PAHs are considered to cause cancer,[10] but EPA provides a CSF for only one of them, BaP, as discussed in Section 5.1.3. EPA (2007b) classifies BaP as a Group "B2," probable human carcinogen, based on multiple studies involving all three primary exposure pathways and both rodent and primate models. In animal models, BaP administered by gavage or in diet increased the incidence of tumors in the stomach, esophagus, and larynx, while inhalation studies involving BaP resulted in higher numbers of respiratory tract and upper digestive tract tumors. In addition, dermal exposure to BaP has been shown to cause skin tumors. There is evidence that exposure to mixtures of PAHs known to contain BaP induces lung cancer in humans; however, there is not enough data to conclude that BaP is the causative agent (USEPA 2007b).

EPA (2007b) recommends an oral slope factor of 7.3 per mg/kg-day for BaP. The risk estimate for BaP is based on the geometric mean of four slope factors derived from multiple data sets. This value is used to estimate cancer risk for BaP and six other carcinogenic PAHs (Section 5.1.3); the CSF is multiplied by each chemical-specific RPF to obtain a toxicity adjusted CSF. EPA Region 6 provides a URF of 0.88 per mg/m^3, which can also be presented as an inhalation CSF of 0.31 per mg/kg-day.

5.2.2 PCDDs/PCDFs: 2,3,7,8-TCDD

EPA does not provide a noncancer toxicity assessment for TCDD. ATSDR's current MRL of 1 pg/kg-day for chronic exposure is based on the LOAEL for reproductive effects in rhesus monkeys, adjusted by a 90-fold uncertainty factor (ATSDR 1998a). ATSDR's subchronic MRL of 20 pg/kg-day is based on the NOAEL for decreased thymus weight in guinea pigs (ATSDR 1998a). The ATSDR chronic and subchronic MRLs were applied in this HHRA to assess the noncancer hazard potential of TCDD TEQ exposures at the Site.

In the 2003 draft dioxin reassessment, EPA relied on one point of departure (the effective dose at the 1 percent response level, or ED_{01}) from data collected for one tumor site and one animal species in a single animal bioassay to calculate its proposed CSF of 1E+06 per mg/kg-day. In

9 The PAHs with noncancer toxicity values are naphthalene, 2-methylnaphthalene, acenaphthene, dibenzofuran, fluorene, anthracene, fluoranthene, and pyrene.
10 The cancer-causing PAHs are benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, BaP, chrysene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene.

selecting this approach, EPA chose from a limited number of animal studies and focused on tumor types for which increased incidence with dose was observed.

5.2.3 Aroclors 1248 and 1260

Arclors are commercial mixtures of PCB congeners, of which there are 209 individual congeners. Aroclor 1248 and Aroclor 1260 are approximately 48 percent and 60 percent chlorine by weight, respectively. The EPA RfD/Reference Concentration (RfC) Work Group reviewed health effects data for Aroclor 1248 in November 1996 and determined that the existing database was inadequate for derivation of an oral RfD (USEPA 2007b). An RfD specific to Aroclor 1260 also is not available at this time from EPA. More recently, ATSDR evaluated noncancer health effects data for PCBs, including commercial Aroclor mixtures (ATSDR 2000b). ATSDR broadly applies the subchronic and chronic MRLs to all PCB mixtures, so the MRLs were selected for representation of the noncancer toxicity of Aroclors 1248 and 1260.

ATSDR's chronic MRL of 0.00002 mg/kg-day is based on an immunotoxicity study involving monkeys tested at 23 and 55 months of exposure to Aroclor 1254. Decreased antibody response was observed at the lowest dose level. Although findings for other tested endpoints did not confirm immunotoxic effects at the lowest dose level, the MRL is supported by the observation of mild changes in eyelids and finger- and toenails at the lowest dose level. This study also was used by EPA (2007b) to develop an RfD for Aroclor 1254 of 0.00002 mg/kg-day.

The ATSDR identified a MRL for subchronic oral exposure to PCBs of 0.00003 mg/kg-day. The MRL is based on neurobehavioral changes in infant rhesus monkeys that were exposed to PCB mixtures in breast milk from birth to 20 weeks old to simulate human infant exposure to PCBs in breast milk. Deficiency in learning and performance was observed in the monkeys at later stages of development although no overt signs of toxicity were observed.

EPA has not conducted a complete carcinogenicity evaluation of Aroclors 1248 and 1260 although EPA has classified PCBs as Group "B2," or probable human carcinogens, based on several animal studies in which rats developed liver tumors after exposure to PCB mixtures (USEPA 2007b). EPA (2007b) considers available data based on human epidemiologic studies to be inadequate due to limited studies and small sample sizes. These studies included reports of increased incidence of various cancers, including gastrointestinal, hematologic, liver, gall bladder, biliary tract, and skin cancers.

For quantitative evaluation of PCB mixtures, including Aroclors 1248 and 1260, EPA (2007b) recommends upper-bound and central estimate slope factors of 2.0 per mg/kg-day and 1.0 per mg/kg-day, respectively, for ingestion via food, soil, or sediment, inhalation of dust or aerosols, and dermal contact (if an absorption factor is applied). The central estimate of 1.0 per mg/kg-day was selected for use in this HHRA because no early life exposures or high-risk populations are expected to be receptors of concern for the Site.

5.2.4 Metals

The following section summarizes available toxicity values and critical study information for inorganic COPCs, arsenic, chromium, and lead.

5.2.4.1 Arsenic

Characteristic changes in the skin are the primary effect on which EPA (2007b) has based a chronic oral RfD of 0.0003 mg/kg-day. These skin changes are noted to be the earliest observable sign of chronic arsenic over-exposure via ingestion. The changes include thickening of the skin and formation of small "corns" or "warts," particularly on the palms and soles, as well as development of darkened areas of the skin interspersed with light spots. ATSDR (2000a) does not provide a subchronic MRL for arsenic; therefore, the chronic value is used in this HHRA.

The EPA (2007b) has classified arsenic as a Group "A" known human carcinogen. For oral exposures, the carcinogenic classification is based largely on increased mortality from multiple internal organ cancers (liver, kidney, lung, and bladder) and an increased incidence of skin cancer in epidemiological studies of Taiwanese populations consuming high levels of arsenic (several hundred micrograms per day) in drinking water (Tseng et al. 1968; Tseng 1977). Based on these studies, EPA derived an oral CSF of 1.5 per mg/kg-day.

Inhalation of arsenic also is associated with increased lung cancer mortality observed in multiple studies of inhalation exposures to arsenic by smelter workers (Enterline and Marsh 1982; Lee-Feldstein 1983; Axelson et al. 1978; Tokudome and Kuratsune 1976; Rencher et al. 1977) and pesticide manufacturing workers (Ott et al. 1974; Mabuchi et al. 1979) in occupational settings. Further support for excess lung cancer risk associated with arsenic inhalation was derived from a single study of residents living near a pesticide manufacturing plant (Matanoski et al. 1981). Based on some of these studies, EPA derived a unit risk estimate for arsenic exposure in air of 4.3 per mg/m^3 (USEPA 2007b). The unit cancer risk represents the excess lifetime risk of lung cancer due to a continuous, constant lifetime exposure to one unit of carcinogenic arsenic concentration in air. This unit air risk for arsenic corresponds to an inhalation CSF of 15 per mg/kg-day (USEPA 2007b).

Numerous weaknesses have been identified regarding the studies underlying the EPA CSF. For example, information contained in the Taiwan studies included arsenic well concentrations in groups (villages) that often vary over a wide range within the same village, as well as over time. Thus, exposure estimates derived from the studies have been criticized as being highly uncertain. Other weaknesses identified include inadequate consideration of differences in genetic, nutritional, cultural, social, economic, and lifestyle characteristics between the Taiwanese study population and populations outside this region.

Estimates of incremental cancer risk that are based on linear extrapolation from observed effects at high doses are inaccurate. There is now substantial mode of action evidence that the dose response for induction of cancer by arsenic is nonlinear (e.g., Germolec et al. 1996; Shimizu et al. 1998; Barchowsky et al. 1999; Kaltreider et al. 1999; Kirkpatrick et al. 2000; Menzel et al. 2000; Trouba et al. 2000; Andrew et al. 2003; Kitchin and Ahmad 2003). Specifically, arsenic carcinogenesis is postulated to involve indirect, rather than direct, interactions with DNA. Because arsenic would have to be present at a sufficiently high level for a sufficient duration to induce these types of indirect effects, postulated modes of action of arsenic carcinogenesis would be expected to have a nonlinear dose-response relationship. In other words, low doses of arsenic are likely to be relatively less effective than higher doses, and may, in fact, be associated with zero risk.

5.2.4.2 Chromium

EPA provides toxicological reviews of both CrIII and CrVI (USEPA 2007b). Although CrVI data are available for the Site, CrVI concentrations did not exceed screening values and CrVI was not retained as a COPC. EPA's RfD for CrIII of 1.5 mg/kg-day is based a study in which rats were exposed to chromic oxide in bread for 5 days/week, for a total of 840 days (USEPA 2007b). No adverse effects were observed in the exposed rats. In fact, CrIII is an essential element for lipid, protein, and fat metabolism and adverse effects are more commonly reported with CrIII deficiencies (USEPA 2007b). EPA (2007b) indicates that there are limited data suggesting that CrIII may affect the human respiratory system but this is not supported by animal studies. The database is inadequate to support development of an inhalation RfD (USEPA 2007b).

EPA (1997b) provides a subchronic oral RfD for CrIII of 1.5 mg/kg-day. Noncancer toxicity values for CrIII are recommended for evaluating total chromium data at the Site.

Epidemiological studies of workers exposed to both CrIII and CrVI report excess lung cancer cases although this increased incidence was found to be associated with exposure to CrVI only. Animal studies investigating the carcinogenicity of CrIII following oral and inhalation exposure do not indicate an increased incidence of tumors whereas laboratory studies confirm an increased incidence of cancer with inhalation exposure to CrVI (USEPA 2007b). EPA has designated trivalent chromium as a Group "D" chemical, not classifiable as to its carcinogenicity due to limitations in the toxicity database.

EPA (2007b) has characterized hexavalent chromium as a Group "A" known human carcinogen for the inhalation route of exposure, due to an increased incidence of lung cancer in workers. Animal studies do not provide evidence that CrVI is carcinogenic via the oral exposure route (USEPA 2007b). EPA's air unit risk of 12 per mg/m^3 translates to an inhalation CSF of 42 per mg/kg-day. An assumption underlying this value is that the ratio of CrVI to CrIII is 1 to 6. This assumption is generally consistent with chromium concentrations found on Lots 3 and 4 where

CrVI was speciated in five samples. This value is used to evaluate inhalation exposures to total chromium at the Site.

5.2.4.3 Lead

Health risks associated with lead exposures are assessed by determining the potential to exceed an absorbed dose of lead, measured as a blood lead concentration that is associated with increased potential for adverse health effects (CDC 1997, 2002; USEPA 1998). As a result of numerous studies of occupationally exposed groups and the general population, much dose-effect data for lead has been published. In the U.S., the Centers for Disease Control and Prevention (CDC) (1991, 2002) has identified a blood lead level of 10 μg/dL as the concentration above which further evaluation may be warranted for an individual child. The 10-μg/dL blood lead level was selected based on studies indicating that exposures resulting in blood lead levels at or above this concentration may present an increased health risk to children (CDC 1991, 1997, 2002; USEPA 1998). Because children are more susceptible to lead exposures and effects than adults, the CDC's blood lead level of concern in children also is considered health-protective of adults.

Many of lead's health effects may occur without overt signs of toxicity. For instance, effects on heme metabolism can be detected at blood lead levels below 10 μg/dL in all ages. With prolonged exposures to much higher levels, clinically significant effects on heme synthesis may occur. These effects include decreased hemoglobin production and destruction of red blood cells. In occupationally exposed adults, EPA estimates the threshold blood lead level for a decrease in hemoglobin to be 50 μg/dL.

EPA (2007b) has determined that lead is a probable human carcinogen based on sufficient evidence of carcinogenicity in animals. Evidence of lead carcinogenicity in human studies was found to be inadequate. Despite the finding that lead is a probable human carcinogen, EPA has determined that noncancer effects of lead provide a more sensitive toxicity endpoint than cancer effects, and no toxicity values have been derived for cancer endpoints.

5.2.5 DDT, DDD, DDE

Toxicity studies conducted in rats indicate exposure to DDT administered in rat chow can induce liver lesions (USEPA 2007b). An oral RfD of 0.0005 mg/kg-day was derived based on a dose at which no adverse effects were seen in the rats (USEPA 2007b). An inhalation RfD has not been derived by EPA and noncancer toxicity values for DDD or DDE are not available from EPA at this time (USEPA 2007b). A provisional value of 0.002 mg/kg-day for DDD is provided by EPA (RAIS 2007).

ATSDR (2003) concluded that the dose-response relationship for DDT has not been well defined and is insufficient for derivation of a chronic oral MRL. However, it was concluded that a

subchronic MRL of 0.0005 mg/kg-day could be calculated based on a no effects level estimated in a rat toxicity study (ATSDR 2003). Rats exposed to higher concentrations of DDT exhibited signs of liver toxicity. Based on evaluations by EPA (2007b) and ATSDR (2003), a value of 0.0005 mg/kg-day is used for both chronic and subchronic exposure scenarios.

EPA (2007b) has classified DDT, DDD, and DDE as Group "B2" probable human carcinogens due to the results of seven studies in various mouse and rat strains in which exposures resulted in liver tumor formation. Animals exposed to DDD and DDE also developed lung and thyroid tumors. There is inadequate epidemiological data to confirm that DDT, DDD, and DDE are carcinogenic in humans.

The oral CSF for DDT and DDE of 0.34 per mg/kg-day is the geometric mean of six slope factors derived from rat and mouse toxicity studies (USEPA 2007b). EPA (2007b) assigned an oral CSF for DDD of 0.24 per mg/kg-day based on the formation of liver tumors in mice. An inhalation unit risk value for DDT of 0.097 per mg/m^3 is translated to an inhalation CSF of 0.34 per mg/kg-day (USEPA 2007b). Inhalation unit risks for DDE and DDD are not provided by EPA (2007b).

5.2.6 Volatile and Semivolatile Organic Compounds

Eight chlorinated solvents were retained as COPCs for the HHRA: chlorobenzene, 1,2-DCB, 1,4-DCB, chloroform, PCE, TCE, vinyl chloride, hexachloroethane, hexachlorobenzene, and BEHP. A toxicity summary for each COPC is provided in this section.

In cases where ATSDR provides inhalation MRLs in units of parts per million (ppm), these values were assumed to be equivalent to μL/L. The gas was assumed to be present at standard temperature and pressure, resulting in a volume of 22.4 L/mol. It was also assumed that the MRL could be modified by an inhalation rate of 20 m^3/day and a body weight of 70 kg. The MRL concentrations were converted to units of mg/kg-day according to the following equation:

$$C(mg/kg-day) = C(\frac{\mu L}{L}) \times MolarMass(\frac{g}{mol}) \times 20(\frac{m^3}{day}) \times \frac{1}{70kg} \times \frac{1mol}{22.4L} \times \frac{mg \times L^2}{g \times m^3 \times \mu L}$$

(Eq. 5-1)

5.2.6.1 Chlorobenzene

Exposure to chlorobenzene results in adverse effects to the liver, kidney, and central nervous system. EPA calculated an oral RfD of 0.02 mg/kg-day based on a level at which no adverse effects were seen in dogs following 13 weeks of oral exposure to chlorobenzene (USEPA 2007b). Administration of chlorobenzene at higher doses resulted in histopathologic changes in the liver, a finding replicated in several other rat, mouse, and rabbit studies (USEPA 2007b). A noncancer toxicity value for exposure via inhalation is not provided by EPA, and ATSDR does not provide oral or inhalation subchronic MRLs for chlorobenzene.

EPA (2007b) classifies chlorobenzene as a Group "D" chemical, not classifiable as to its carcinogenicity, due to a lack of human data and inadequate animal bioassay database.

5.2.6.2 1,4-Dichlorobenzene

Inhalation of 1,4-DCB may irritate the eyes, nose, mouth, and lungs. If exposed for long periods of time, these chemicals may adversely affect the kidneys and blood. An oral RfD for 1,4-DCB is not available from EPA (2007b); although, ATSDR provides a chronic oral MRL of 0.07 mg/kg-day (ATSDR 2006). The MRL is based on increased liver enzyme activity in beagles and also is recommended by ATSDR for use as a subchronic oral MRL (ATSDR 2006). EPA provides an RfC of 0.8 mg/m^3, which is converted to an inhalation RfD of 0.23 mg/kg-day. The RfC is based on a level at which no adverse effects were observed in rats exposed to 1,4-DCB vapors. Animals exposed to higher concentrations exhibited increased liver weights (USEPA 2007b). A subchronic inhalation MRL of 0.2 ppm for 1,4-DCB is based on the critical study used to develop EPA's RfC. Using the equation provided above, ATSDR's subchronic inhalation MRL was converted to a concentration of 0.38 mg/kg-day.

EPA has not conducted a complete evaluation of the carcinogenic potential of 1,4-DCB (USEPA 2007b). However, HEAST provides an oral CSF for 1,4-DCB of 0.024 per mg/kg-day (USEPA 1997b). No classification is provided. This value was used for both oral and inhalation exposures to 1,4-DCB.

5.2.6.3 Chloroform

Exposure to chloroform for long periods of time may adversely affect the kidneys and liver. EPA (2007b) calculated an oral RfD of 0.01 mg/kg-day based on the lowest dose at which an adverse effect was observed in an animal study. The critical endpoint of the study was formation of fatty cysts in the liver and elevated liver enzyme levels in dogs exposed to chloroform in toothpaste for 7 years (USEPA 2007b). This study also forms the basis for the ATSDR (1997) chronic and subchronic oral MRLs of 0.01 mg/kg-day and 0.1 mg/kg-day, respectively.

EPA (2007b) does not provide inhalation noncancer toxicity values although ATSDR (1997) provides values for chronic and subchronic inhalation MRLs of 0.02 ppm and 0.05 ppm, respectively. The chronic inhalation MRL is converted to a value of 0.03 mg/kg-day, assuming an inhalation rate of 20 m^3/day and body weight of 70 kg. This chronic inhalation MRL is based on the lowest dose at which factory workers developed toxic hepatitis following exposure to chloroform in air for 1-4 years (ARSDR 1997). The subchronic inhalation MRL may be converted to a value of 0.076 mg/kg-day, assuming an inhalation rate of 20 mg/m^3 and body weight of 70 kg. This subchronic MRL presented by ATSDR (1997) is based on toxic hepatitis, anorexia, nausea, and vomiting in female factory workers exposed to chloroform vapors for up to 6 months.

The animal toxicity study database is considered sufficient to support the classification of chloroform as a Group "B2," probable human carcinogen (USEPA 2007b). EPA (2007b) indicates that chloroform is considered carcinogenic via any route of exposure (e.g., oral, inhalation, dermal). The results of toxicity studies indicate that animals develop liver and/or kidney tumors only after receiving sustained exposures at a concentration sufficiently high to cause cell toxicity and excessive cell regeneration. Because carcinogenicity is secondary to cellular toxicity and proliferative cell regeneration produced by chloroform exposure, EPA does not rely on an assumption of a linear mode of action for estimating cancer risk (USEPA 2007b).

Alternatively, a margin-of-exposure approach is preferred by EPA to estimate cancer risk, which is similar to the use of a noncancer toxicity value, or RfD (USEPA 2007b). The estimated dose above which kidney tumor development is observed in humans consuming chloroform in water is 23 mg/kg-day, a value 2,000 times higher than the noncancer toxicity value described above (USEPA 2007b). Based on this comparison, the RfD of 0.01 mg/kg-day is protective of increased cancer risk from exposure to chloroform (i.e., cancer risk is considered negligible at exposure concentrations below the RfD) (USEPA 2007b).

An inhalation unit risk of 0.023 per mg/m^3 is provided by EPA (2007b) with the caveat that this value, developed in 1987, has not been revisited since more recent data have become available. This unit risk is based on mice exposed to chloroform via gavage and observation of tumors in the liver (USEPA 2007b). The inhalation unit risk is converted to an inhalation CSF of 0.081 per mg/kg-day.

5.2.6.4 Tetrachloroethene

The primary noncancer health effect associated with exposure to PCE is liver toxicity. The EPA (2007b) has established a noncancer toxicity value of 0.01 mg/kg-day based on a 6-week study in which mice were fed PCE in corn oil. Although the exposure duration of the principal study is relatively short, other studies in which animals were exposed orally or via inhalation support the established RfD. Subchronic oral toxicity is estimated using a value of 0.1 mg/kg-day (USEPA 1997b).

A noncancer toxicity value for inhalation exposures is not provided by EPA (2007b). ATSDR (1996a) provides a chronic inhalation MRL of 0.04 ppm, which is translated to a concentration of 0.085 mg/kg-day. The subchronic inhalation RfC of 3 mg/m^3 was converted to a value of 0.86 mg/kg-day (USEPA 1997b).

In 1989, the EPA withdrew the quantitative cancer toxicity values that it had previously issued for PCE. Since that time, the data have been the subject of extensive review regarding the implications of the available data and appropriate carcinogenicity classification. The EPA does not currently endorse quantitative toxicity factors for PCE; however, as a matter of practice, the previously recommended values are commonly used to generate quantitative risk estimates.

In order to include PCE in the risk evaluation, toxicity factors obtained from the EPA Environmental Criteria and Assessment Office (USEPA-ECAO) were used (USEPA-ECAO 1992, 1994). USEPA-ECAO provided the toxicity factors from the 1985 Health Assessment Documents and 1987 Addenda for PCE. The CSF used to estimate cancer risk for PCE is 0.54 per mg/kg-day. EPA Region 6 provides an inhalation URF of 0.0059 per mg/m^3, which is converted to an inhalation CSF of 0.021 per mg/kg-day.

5.2.6.5 Trichloroethene

Breathing TCE over long time periods may cause nerve, kidney, and liver damage. EPA (2007b) does not provide a quantitative assessment of noncancer toxicity of TCE. EPA Region 6's website lists a provisional oral RfD of 0.0003 mg/kg-day developed by the National Center for Environmental Assessment. A provisional inhalation RfD of 0.011 mg/kg-day also is provided. ATSDR's (1998b) subchronic inhalation MRL of 0.1 ppm was converted to a subchronic inhalation MRL of 0.17 mg/kg-day. The MRL is based on decreased heart rate and disturbed sleep in rats exposed to TCE vapors for 6 weeks. A subchronic MRL for oral exposures is not provided by ATSDR.

EPA has classified TCE as likely to be carcinogenic in humans, although EPA (2007b) does not provide a quantitative assessment of TCE toxicity at this time. In 1989, the EPA withdrew the quantitative toxicity values that it had previously issued for this compound. Since that time, the data for TCE have been the subject of extensive review regarding the implications of the available data and appropriate carcinogenicity classifications.

EPA has recently issued a draft reassessment of TCE toxicity that increases the estimated potency of TCE by 5- to 65-fold (USEPA 2001). The EPA Science Advisory Board has supported the EPA reassessment, and the updated assessment is currently undergoing peer review.

EPA has proposed a range of slope factors for assessing both oral and inhalation exposures to TCE on the basis that the studies used to derive CSFs resulted in a range of risks depending on the exposure route. The oral CSF of 0.4 per mg/kg-day is presented in the HHRA. Inhaled TCE produces higher levels of the less-toxic metabolite trichloroacetate, while ingested TCE yields higher levels of the more toxic dichloroacetate (USEPA 2001). Because of these differences, the lower end of the proposed range of slope factors, 0.0016 per mg/kg-day, is more appropriate to use for evaluation of inhalation exposures and was used in this HHRA to assess groundwater vapor inhalation risks.

5.2.6.6 Vinyl Chloride

Exposure to vinyl chloride for long periods of time may result in liver and nerve damage. EPA (2007b) provides quantitative estimates of noncancer and cancer toxicity for vinyl chloride, which are based on data from animal studies and physiologically-based pharmacokinetic

(PBPK) modeling. EPA's oral RfD of 0.003 mg/kg-day is based on a level at which no adverse effects were observed in rats fed vinyl chloride in food. The no-effect level of 0.13 mg/kg-day for rats was converted to an equivalent human dose using a PBPK model that predicts time-integrated liver concentrations of reactive vinyl chloride metabolites. In rats, adverse effects observed at higher doses included angiosarcoma, hepatocellular carcinoma, liver cell polymorphism, and cysts (USEPA 2007b). EPA (2007b) also provides a reference concentration of 0.1 mg/m^3, which may be converted to an inhalation reference dose of 0.029 mg/kg-day.

ATSDR (1998c) provides a subchronic inhalation MRL of 0.03 ppm for rats exposed to vinyl chloride vapors for 10 weeks prior to mating. Investigators observed increased liver weights in rats in the two highest dose groups. The lowest dose at which an adverse effect was observed was used to derive the MRL. The MRL was converted to a subchronic inhalation MRL of 0.024 mg/kg-day.

EPA (2007b) has classified vinyl chloride as a Group "A," known human carcinogen, based on human epidemiological studies of workers exposed to vapors in the workplace. The studies report a rare type of liver tumor, angiosarcoma, in exposed workers. This finding is supported by oral and inhalation animal studies, *in vitro* testing, and PBPK modeling (USEPA 2007b). In addition, vinyl chloride is considered carcinogenic via dermal absorption, as it is readily absorbed and is rapidly distributed throughout body. Vinyl chloride metabolite, chloroethylene oxide, has been shown to form DNA adducts, resulting in tumor formation.

EPA (2007b) provides a CSF of 0.72 per mg/kg-day, based on the linearized multistage model for exposure throughout adulthood. Similarly, an inhalation unit risk factor of 0.0044 per mg/m^3 for continuous lifetime exposure during adulthood was estimated and was converted to an inhalation CSF of 0.015 per mg/kg-day.

5.2.6.7 Hexachloroethane

Skin, nose, lung, and eye irritation may result from exposure to hexachloroethane. Hexachloroethane fed to rats for 16 weeks induced kidney damage, including lesions, atrophy, and degeneration (USEPA 2007b). An oral RfD of 0.001 mg/kg-day was derived from the dose at which no adverse effects were seen in rats (USEPA 2007b). Kidney and liver damage also has been observed in other subchronic rodent and dog studies for exposures via ingestion and inhalation. ATSDR (1996b) has developed a subchronic oral MRL of 0.01 mg/kg-day that is based on the same principal study that EPA uses for its oral RfD.

EPA does not provide noncancer toxicity values for exposures via inhalation. A subchronic inhalation MRL was developed by ATSDR (1996b) based on a subchronic inhalation study on rats. Adverse effects in the exposed rats included reduced resistance to infection, reduced body weight, and eye irritation. The dose at which no adverse effects were observed was used to derive the MRL of 6 ppm, or 18 mg/kg-day.

EPA classifies hexachloroethane as a Group "C" possible human carcinogen, due to one study in which mice developed tumors in the liver, although rats exposed to hexachloroethane in the same study did not develop tumors (USEPA 2007b). An oral CSF for hexachloroethane of 0.014 per mg/kg-day is provided by EPA (2007b). The oral CSF was used to derive an inhalation unit risk of 0.004 per mg/m^3.

5.2.6.8 Hexachlorobenzene

Hexachlorobenzene can cause liver and thyroid damage, as well as adverse effects in the nervous system, bones, kidneys, blood, and immune and endocrine systems if exposure occurs over a long period of time. EPA provides an oral RfD of 0.0008 mg/kg-day, based on the level at which no adverse affects were observed in a 130-week study in which rats were exposed orally to hexachlorobenzene (USEPA 2007b). At higher doses, adverse effects were observed in the liver. A subchronic MRL of 0.0001 mg/kg-day is provided by ATSDR (2002a); however, the oral RfD will be used to evaluate subchronic exposures. EPA and ATSDR do not provide an RfC or MRL for inhalation exposures.

EPA classifies hexachlorobenzene as a Group "B2" probable human carcinogen due to the formation of tumors in the liver, kidney, and thyroid in three rodent species following oral exposure (USEPA 2007b). An oral CSF of 1.6 per mg/kg-day is provided by EPA and was used by EPA to develop the inhalation URF of 0.46 per mg/m^3 (USEPA 2007b).

5.2.6.9 Pentachlorophenol

Exposure to high levels of PCP may cause damage to the liver and immune system. The chronic RfD of 0.03 mg/kg-day is based on a study in which rats were administered PCP in the diet for 2 years, resulting in pigmentation of the liver and kidneys (USEPA 2007b). The ATSDR (2001) provides a subchronic oral MRL of 0.001 mg/kg-day, which was derived from a study on reproductive effects in mink. There is currently no established RfC or inhalation MRL for PCP (ATSDR 2001; USEPA 2007b).

Based on studies in mice, USEPA (2007b) has classified PCP as a "B2," or probable human carcinogen. The CSF derived for PCP is based on a single diet study in mice. Mice administered high doses of PCP in feed for 2 years developed hepatocellular adenomas and carcinomas, adrenal medulla and malignant pheochromocytomas, and hemangiosarcomas and hemangiomas. No similar effect has yet been shown in humans, and USEPA (2007b) considers the one available investigation to be inadequate due to study design flaws. The recommended value for the oral CSF for PCP is 0.12 per mg/kg-day (USEPA 2007b). A URF is not available; therefore, the oral CSF is used to estimate cancer risks from inhalation exposures.

5.2.6.10 Bis(2-ethylhexyl)phthalate

BEHP is not known to cause adverse health effects normally found in the environment (ATSDR 2002b). Animals exposed to high levels of BEHP for long periods of time caused damage to the liver of rats and mice; however, it is not known if BEHP also causes liver damage in humans (ATSDR 2002b). Nevertheless, EPA developed an oral RfD of 0.02 mg/kg-day based on the lowest level that caused increased liver weights in guinea pig (USEPA 2007b). ATSDR provides a subchronic MRL of 0.1 mg/kg-day based on the level at which no adverse effects were observed in mice exposed to BEHP (ATSDR 2002b). Noncancer toxicity values for inhalation exposures are not provided by EPA or ATSDR.

There is insufficient human data to evaluate the carcinogenicity of BEHP (USEPA 2007b). EPA (2007b) has classified BEHP as a Group "B2" probable human carcinogen based on liver tumors observed in rat and mice studies. An oral CSF of 0.014 per mg/kg-day is provided by EPA (2007b). A URF is not available; therefore, the oral CSF is used to estimate cancer risks from inhalation exposures.

5.2.7 Diesel and Residual Range Organic Hydrocarbons

Petroleum products consist of a varying mixture of petroleum hydrocarbons, such as benzene, toluene, ethylbenzene, and xylene (BTEX), PAHs, and aliphatic and aromatic hydrocarbons. Diesel fuel is a mixture of the heavier aliphatic and aromatic hydrocarbon fractions in the C12 to C21 range. RROs generally are heavier than diesel fuel, dominated by aliphatic and aromatic hydrocarbon fractions in the C16 to C34 range. The mixture varies depending on the age and source of the petroleum product. Sometimes, one fraction or component is more toxic than the others, but it may or may not dominate the mixture in terms of its quantity. For that reason, the toxicity and quantity of the various components typically is determined in order to evaluate the toxicity of the entire mixture (DEQ 2003). Toxicity values are assigned to the various components and fractions to be used in quantifying the toxicity of the entire mixture.

For this risk assessment, the toxicity of diesel and residual range organics is estimated by evaluating the toxicity of individual components that were selected as COPCs, such as PAHs.

6 RISK CHARACTERIZATION

To characterize risks, quantitative estimates of exposure and toxicity are combined to yield numerical estimates of potential health risk for noncarcinogenic and carcinogenic COPCs. This phase of a risk assessment also involves interpreting and qualifying the derived risk estimates and the uncertainty associated with them.

6.1 NONCANCER RISKS

Health risks other than cancer are characterized as the increased likelihood that an individual will suffer adverse health effects as a result of chemical exposure. To evaluate noncancer risks, the ratio of the average daily intake to the RfD is calculated. This ratio is referred to as the HQ. If the calculated value of the HQ is less than or equal to 1, no adverse health effects are expected. If the calculated value of the HQ is greater than 1, then further risk evaluation is needed. The HQ will be calculated using the following equation:

$$HQ = \frac{Intake}{RfD} \qquad \text{(Eq. 6-1)}$$

Where:

HQ = Hazard quotient associated with exposure to the chemical via the specified exposure route (dimensionless)

Intake = Estimated average daily intake of the chemical via the specified exposure route (mg/kg-day)

RfD = Reference dose for the COPC (mg/kg-day).

To evaluate the effect of exposure to multiple chemicals that act on the body in a similar manner, the HQs for each exposure pathway for individual chemicals are typically summed to determine a noncancer hazard index (HI) using the following formula:

$$HI = \frac{Intake_1}{RfD_1} + \frac{Intake_2}{RfD_2} + \ldots + \frac{Intake_i}{RfD_i} \qquad \text{(Eq. 6-2)}$$

Where:

HI = hazard index

$Intake_i$ = Intake for chemical *i* (mg/kg-day)

RfD_i = Reference dose for the ith chemical (mg/kg-day).

HIs for multiple chemicals are generally not summed if the reference doses for the chemicals are based on effects on different target organs. This is because the noncancer health risks associated with chemicals that affect different target organs are unlikely to be additive.

6.2 CANCER RISKS

The cancer risk estimates derived using standard risk assessment methods are characterized as the incremental probability that an individual will develop cancer during his or her lifetime due to exposure to site-related chemicals resulting from the specific exposure scenarios that are going to be evaluated. The term "incremental" reflects the fact that the calculated risk associated with site-related exposure is in addition to the background risk of cancer experienced by all individuals in the course of daily life.

Excess incremental lifetime cancer risks were calculated using the following equation:

$$Cancer\ Risk = Intake\left(\frac{mg}{kg \cdot day}\right) \times CSF\left(\frac{mg}{kg \cdot day}\right)^{-1} \qquad \text{(Eq. 6-3)}$$

Because cancer risks are assumed to be additive, risks associated with simultaneous exposure to more than one carcinogen in a given medium are typically combined to estimate the total cancer risk associated with each exposure pathway (USEPA 1989). Where exposures may occur via multiple exposure routes, total cancer risks for each exposure pathway may be summed for reasonable combinations of exposure pathways to determine the total cancer risk for the population of concern.

6.3 RISK RESULTS FOR LOTS 1 AND 2

Noncancer and cancer risk estimates were calculated for the outdoor occupational worker, outdoor occupational worker under a redevelopment scenario, construction worker, excavation worker, and trespasser for Lots 1 and 2. An indoor worker was not required for the quantitative risk assessment of Lots 1 and 2. No COPCs were identified for the indoor air inhalation exposure via volatiles from subsurface soil in Lots 1 and 2. Groundwater underlying Lots 1 and 2 has not been shown to be impacted by operations that occurred on Lots 3 and 4. The indoor air inhalation exposure via volatiles in groundwater conducted for a future worker in Lots 3 and 4 is a conservative surrogate for the future indoor worker at Lots 1 and 2. Noncancer and cancer risks are presented in Sections 6.3.1 and 6.3.2, below.

6.3.1 Noncancer Risks

Noncancer hazards for all workers (outdoor occupational, outdoor occupational after redevelopment, construction, and excavation workers) and the trespasser were below a target HQ of 1 (Tables 6-1 through 6-5). The greatest HQs were calculated for the construction worker, ranging from 0.01 to 0.1 for CTE and 0.04 to 0.4 for RME exposures. The risk drivers for all receptors were DDT and arsenic. For both arsenic and DDT, the EPC used in the risk evaluation was biased high by a single extreme soil concentration. However, the extreme value for each chemical was not from the same sample location. Thus, the risk results are considered to be an upper-bound estimate of the potential health risks.

The soil ingestion pathway contributed the majority of the hazard for the soil exposure pathways. Exposure via incidental ingestion of pesticides (DDD and DDT) in soil resulted in the greatest HIs when HQ results were summed across an exposure pathway for a given receptor. Each pathway HI as well as the total HI for a specific receptor was less than 1; therefore, adverse noncancer health effects are not expected for soil exposures at Lots 1 and 2.

6.3.2 Cancer Risks

Potential cancer risks for all workers and a trespasser are shown in Tables 6-6 through 6-10. For the outdoor occupational worker under both current and future redevelopment scenarios, potential cancer risks were the same because there are no data available for deeper soils that a redevelopment worker may contact. Potential risk to an outdoor worker associated with CTE conditions exceeded a cancer risk of 1×10^{-6} only for arsenic. Under RME conditions, individual COPCs that exceeded a cancer risk of 1×10^{-6} were limited to arsenic and 4,4′-DDT. The total cancer risk for all COPCs and pathways combined ranged from 2×10^{-6} for CTE to 2×10^{-5} for RME. Ingestion of arsenic in soil represents the greatest contribution (65 percent) to total cancer risk. The outdoor occupational worker was the receptor with the greatest potential cancer risk associated with soil exposures at Lots 1 and 2. Total cancer risk for a construction worker ranged from 5×10^{-7} to 3×10^{-6} for the CTE and RME exposures, respectively. Total cancer risk for an excavation worker ranged from 2×10^{-8} to 1×10^{-7} for the CTE and RME exposures, respectively. The soil ingestion pathway dominated the risk for all workers, and arsenic and 4,4-DDT were the risk driving chemicals.

The total cancer risk for a trespasser on Lots 1 and 2 ranged from 2×10^{-6} for CTE to 4×10^{-6} for RME. The soil ingestion pathway dominated the risk results contributing approximately 80 percent of the total risk. Arsenic and 4,4′-DDT were the risk drivers for the trespasser exposures with total individual cancer risks under the RME of 2×10^{-6} and 1×10^{-6}, respectively.

As discussed earlier arsenic and 4,4′-DDT EPCs used in the risk evaluation was biased high by a single extreme soil concentration and are therefore considered to be an upper-bound estimate of the potential health risks

6.4 RISK RESULTS FOR LOTS 3 AND 4

Noncancer and cancer risk estimates were calculated for five worker receptor groups at Lots 3 and 4: the outdoor occupational worker; an outdoor occupational worker under the redevelopment scenario; a construction worker; an excavation worker; and an indoor worker. The potential risks were not quantified for an indoor worker via direct soil contact as they would be much lower than the potential risks calculated for the other worker receptors. Noncancer and cancer risks are presented in Sections 6.4.1 and 6.4.2, below.

6.4.1 Noncancer Risks

Noncancer hazard estimates for the worker exposures are provided in Tables 6-11 through 6-14. The potential risks for the excavation worker were the lowest of the workers with significant direct contact exposures. Noncancer HQs did not exceed a threshold of 1.0 for contact with COPCs by the excavation worker in Lots 3 and 4. The risk for the soil ingestion pathway dominated the direct soil contact exposures with DDT as the risk driving COPC. Similarly, for outdoor occupational workers and construction workers, DDT exposure via soil ingestion was the risk driver with HQs ranging from 1 to 2 and from 2 to 7 for the CTE and RME, respectively. HIs calculated by grouping chemicals with common target organ toxicity exceeded the threshold of 1 for pesticides (DDD and DDT) only. Because these risks are dominated by intake of DDT, HIs are the same as the HQs listed above for DDT intake. Hazards for intake of Aroclor 1248 and 1260, total chromium, and chlorinated organic compounds were below an HQ of 1.

Noncancer HQs for indoor workers via inhalation of vapors in indoor air from subsurface soil and groundwater are presented in Tables 6-15 and 6-16, respectively. Individual HQs for inhalation of volatile COPCs from subsurface soil (PCE and 1,4-DCB) were well below 1. The total HI for indoor worker inhalation of COPCs from subsurface soil was 0.01 under both CTE and RME conditions. Likewise, individual HQs for inhalation of volatile COPCs from groundwater (PCE and chloroform) were all well below 1. The total HI for indoor worker inhalation of COPCs from groundwater was 0.0007 under both CTE and RME conditions.

6.4.2 Cancer Risks

Individual and total cancer risks for the outdoor worker, redevelopment worker, construction worker, and excavation worker in Lots 3 and 4 are shown in Tables 6-17 through 6-20. Total potential cancer risk is the greatest for an outdoor occupational worker under both current and future redevelopment conditions. Under CTE conditions, individual cancer risk exceeded a value of 1×10^{-6} only for 4,4′-DDT, which had a value of 2×10^{-5}. Under RME conditions, 4,4′-DDD, 4,4′-DDE, and 4,4′-DDT exceeded a cancer risk of 1×10^{-6}. Total cancer risk for an outdoor occupational worker ranged from 2×10^{-5} to 1×10^{-4} under CTE and RME conditions, respectively. The greatest contributor to total risk was ingestion of soil containing 4,4′-DDT. Incidental

ingestion of 4,4′-DDT was also the risk driver for the other occupational exposures with an estimated total cancer risk ranging from 3×10^{-6} (CTE) and 2×10^{-5} (RME) for a construction worker and 1×10^{-7} (CTE) to 7×10^{-7} (RME) for an excavation worker at Lots 3 and 4.

Individual and total cancer risks for the indoor worker at Lots 3 and 4 are shown in Tables 6-15 and 6-16. Total excess lifetime cancer risk for the subsurface soil vapor intrusion pathway ranged from 2×10^{-6} to $8\text{x}10^{-6}$ for the indoor worker. The total cancer risk is dominated by the risk associated with PCE (i.e., 2×10^{-6} for the CTE and to $6\text{x}10^{-6}$ for the RME). The PCE results are driven by a single elevated concentration in the subsurface soil that is more than one hundred times greater than the next highest detection. Because of the low frequency of detection for PCE in the subsurface soil (i.e., 8 percent of the 77 samples) the cancer risk results are considered to be an upper-bound estimate of the potential risk.

Potential total excess lifetime cancer risk for an indoor worker exposed via vapor intrusion from groundwater ranged from 1×10^{-7} under CTE to 5×10^{-7} under RME. Estimated cancer risk associated with individual COPCs ranged from 9×10^{-8} to 4×10^{-7} for chloroform and 4×10^{-8} to 2×10^{-7} for PCE. All individual cancer risks were below a cancer risk of 1×10^{-6} for both CTE and RME conditions.

6.5 RISK RESULTS FOR THE RIVERBANK

Noncancer and cancer risk estimates were calculated for the current outdoor occupational worker, construction worker, and trespasser for the riverbank area (Tract A). Noncancer and cancer risks are presented in Sections 6.5.1 and 6.5.2, below.

6.5.1 Noncancer Risks

Noncancer hazard estimates for workers and trespassers in the riverbank area are presented in Tables 6-21 through 6-23. HQs for all COPCs and receptors were less than 0.1 for both the CTE and RME cases. Total HIs for potential cumulative effects were highest for the worker exposures and ranged from 0.1 for the CTE to 0.3 for the RME. The trespasser HIs ranged from 0.05 for the CTE to 0.1 for the RME. Risks for all receptors were dominated by ingestion of soil containing dioxin/furans and arsenic.

6.5.2 Cancer Risks

Estimated cancer risk for a current outdoor occupational worker, a construction worker and a trespasser in the riverbank area are presented in Tables 6-24 through 6-26. For an outdoor worker, individual cancer risks for arsenic and dioxin/furans exceeded a cancer risk of 1×10^{-6}. The cancer risk was primarily associated with ingestion of soil containing dioxins/furans, which ranged from 6×10^{-6} under CTE conditions to 5×10^{-5} under RME conditions. Total cancer risk for

an outdoor worker in the riverbank area ranged from 8×10^{-6} to 7×10^{-5} under the CTE and RME cases, respectively.

For a construction worker, individual cancer risks for arsenic and dioxin/furans exceeded a cancer risk of 1×10^{-6} under the RME case but not the CTE case. Once again, the cancer risk was primarily associated with ingestion of soil containing dioxins/furans, which ranged from 1×10^{-6} under CTE conditions to 7×10^{-6} under RME conditions. Total cancer risk for an outdoor worker in the riverbank area ranged from 1×10^{-6} to 1×10^{-5} under the CTE and RME cases, respectively.

Potential cancer risks were similar for trespassers, with total cancer risk ranging from 6×10^{-6} under CTE conditions to 1×10^{-5} under RME conditions. The major contributor (75 percent) to total risk was ingestion of soil containing dioxins/furans, which showed a cancer risk of 4×10^{-6} for the CTE case and 8×10^{-6} for the RME case.

6.6 RESULTS OF ADULT LEAD METHODOLOGY

Lead was retained as a COPC in soil for Lots 1 and 2 and for the riverbank. Risks were evaluated using the ALM and are summarized in Table 6-27.

Receptors evaluated for exposure to lead in soil at Lots 1 and 2 were a construction worker, outdoor occupational worker, excavation worker, and trespasser. At this location, the results of the ALM predicted that 95th percentiles of fetal blood lead associated with maternal exposures would range from 4.3 to 5.2 μg/dL, well below the target blood lead level of concern of 10 μg/dL. The probability that fetal blood lead associated with maternal exposure would exceed the target level ranged from 0.3-0.6 percent for all receptors for CTE and RME conditions. These results were well below the target risk level of a 5 percent probability that fetal blood lead will exceed a level of 10 μg/dL (USEPA 2003a). Protection of the fetus is the most health-sensitive endpoint for adults (USEPA 2003a), and therefore, additionally, no risks to the receptors themselves are anticipated with exposure to lead in Lots 1 and 2. Assuming a lognormal distribution the 95th percentile of blood lead for the most highly exposed receptor at Sites 1 and 2 (construction worker, RME, geometric mean blood lead concentration of 1.7 μg/dL,) is predicted as 5.8 μg/dL (Eq. 4-9). Therefore, the results of the ALM indicate that exposure to lead in soil is not a concern for Lots 1 and 2 under assumed exposure conditions.

There are uncertainties associated with risks modeled for the excavation worker in Lots 1 and 2. The default BKSF applied in the ALM is applicable to exposures that result in a quasi-steady state for blood lead concentration. Available evidence shows that for exposures shorter than 90 days, lead concentrations in the body may not reach steady state. Therefore the BKSF may not be directly applicable for determining the uptake of lead, and the results of the ALM model uncertain. The exposure frequency for the excavation worker is only 9 days/year, and therefore it is anticipated that concentrations of blood within the body of this receptor would not reach the quasi-steady state required by the ALM model. Given the fact that risks predicted by the

ALM for the excavation worker are significantly below risk thresholds of concern, even given these uncertainties, it is not anticipated that significant risk to this receptor is likely.

In addition there are uncertainties associated with the risks modeled for the adolescent trespasser. During adolescence, growth spurts may result in an increase in bone deposition and increase in blood volume which may result in lower concentrations of lead in blood. Furthermore, potential lead absorption may be higher in adolescents than in adults These factors make selecting the appropriate baseline blood lead (PbB_0) and AF values for the adolescent trespasser difficult. For the trespasser in this risk assessment PbB_0 and AF parameters were assumed to equal those adopted for the adult worker receptors. These assumptions add some uncertainty to the results.

Given the fact that risks predicted by the ALM for the excavation worker and trespasser are significantly below risk thresholds of concern, it is not anticipated that significant risk to this receptor is likely despite the model uncertainties.

Receptors evaluated for exposure to lead in riverbank soil were the trespasser, outdoor occupational worker, and construction worker. The results of the ALM predicted that the 95th percentile of fetal blood lead associated with maternal exposure for the trespasser would range from 4.4.μg/dL (trespasser CTE) to 5.8 μg/dL (construction worker RME). The probability that fetal blood lead levels associated with maternal exposure at the Site would exceed the target level of 10 μg/dL is between 0.3 and 0.9 percent for the range of CTE and RME scenarios. In addition, assuming a lognormal distribution of blood lead levels in adults, the 95th percentile of blood lead for the most highly exposed receptor at the riverbank (i.e., construction worker RME, geometric mean blood lead concentration = 1.9 μg/dL) is predicted as 6.5 μg/dL (equation 4-9). The results of the ALM indicate that exposure to lead in soil is not a concern for any of the receptors evaluated for the riverbank area under the assumed exposure conditions.

6.7 UNCERTAINTY EVALUATION

The process of evaluating human health risks involves multiple steps. Uncertainties exist in each step, including the collection of samples at a site, laboratory analysis of collected samples, derivation of toxicity values, and estimation of potential site exposures.

This HHRA employed a deterministic approach, in which single fixed input values (i.e., point estimates) are used to represent exposure and toxicity parameters in the risk assessment equations. The output of this approach is a single value of risk for each exposure pathway and scenario. However, the risk estimates are based on a considerable number of assumptions and do not characterize quantitatively the variability inherent in population exposures and responses or the uncertainty associated with the assumptions made (USEPA 1989). An examination of generic and site-specific uncertainties associated with the HHRA is a key element to providing a comprehensive characterization of risk.

Variability for some of the exposure parameters was measured in the HHRA by generating risks for both a CTE and a RME case. CTE and RME risk results are discussed in Sections 6.3 through 6.6. The difference between the CTE and the RME gives an indication of degree of variability between an individual with a central tendency risk and an individual with a high end risk. A limitation of this approach is the lack of knowledge of where the CTE and RME points lie on the distribution of risk for the population of concern. For example, though the goal of the RME case is to represent the 95th percentile of the risk probability distribution, one may find that the RME risk for a receptor may actually lie above the 99th percentile if a full distribution of risk were generated via a probabilistic analysis. The reason for this is that the RME risk combines multiple conservative assumptions that together may result in risk estimates that exceed the 99th percentile.

Table 6-28 provides a list of uncertainties thought to be most significant in this HHRA. Uncertainties are provided for each of the components of the HHRA (i.e., Data Evaluation/COPC Selection, Exposure Assessment, Toxicity Assessment, and Risk Characterization). This table also provides an overview of the nature and magnitude of effects of these sources of uncertainty on the HHRA results.

The potential impact of newer and more spatially complete groundwater data is another source of uncertainty in this risk assessment. The validated groundwater data for the sampling collected in April of 2007 (ERM 2007) was not available in time to be included in the quantitative risk estimates provided above. To address this uncertainty, an analysis was conducted of the April 2007 groundwater data results relative to the groundwater data used in the quantitative risk estimates.

A detailed comparison of the historic groundwater data to the April 2007 samples has shown that concentrations are generally trending downwards (Integral 2008). For example, decreases in concentrations of TCE and PCE by factors of 14, and 5, respectively were observed in the vicinity of Lots 3 and 4. The uncertainty analysis presented in this Section may obscure this trend because a very conservative approach was taken to provide an upper-bound on the potential impact of including the newer groundwater data in the risk assessment. This analysis focused on a comparison of the maximum concentration for a chemical from each dataset without regard to location. Therefore, a higher maximum in the April 2007 data does not indicate that concentrations are increasing at a particular location because the spatial aspects were not considered in this analysis. Similarly, an increase in the maximum would not indicate that the average groundwater concentrations at the Site were increasing because only the maximum concentrations were evaluated.

The exposure pathway for groundwater at the Site was via inhalation of volatile chemicals migrating into ambient or indoor air. The historic groundwater data, dominated by sampling wells in and around Lots 3 and 4, was applied universally to all potential receptors. This assumption had the potential to overestimate potential health risks for receptors in Lots 1 and 2,

especially because of the lesser impact to groundwater in this area relative to Lots 3 and 4. The April 2007 groundwater data included three monitoring well clusters in Lots 1 and 2 that were not part of the historic groundwater data used in the risk assessment, which allowed for evaluation of this potential bias.

The groundwater uncertainty evaluation was based on the maximum concentrations detected in the April 2007 monitoring event. For each COI identified as a sufficiently volatile to migrate into ambient or indoor air from groundwater (See Tables 3-21 and 3-22), the maximum detected concentration for the April 2007 monitoring event was identified. For this evaluation the April 2007 data was separated into wells from Lots 1 and 2 and wells from Lots 3 and 4. The ratio of the maximum from April 2007 to the maximum used for the risk assessment was calculated to determine if the newer data would reflect a higher concentration than considered in the risk assessment. As discussed earlier, groundwater data from Lots 3 and 4 were used to characterize conditions at Lots 1 and 2 due to a lack of wells in this area. A comparison of maximum detected concentrations from April 2007 at Lots 1 and 2 with the historic data from Lots 3 and 4 indicates that maximums are higher for the following volatile chemicals (i.e., 1,2-DCB, 1,4-DCB, benzene, and vinyl chloride). These chemicals are likely to be related to offsite sources along the western boundary of the Site. Thus the detected concentrations of these offsite chemicals in Lots 1 and 2 relative to groundwater concentrations from Lots 3 and 4 are consistent with this source characterization.

The comparison of the historic and April 2007 data for Lots 3 and 4 indicated that maximum concentrations of volatile chemicals that were above the maximum used in the risk assessment for seven chemicals (i.e., 1,1-dichloroethane, benzene, bromodichloromethane, chloroethane, chloroform, PCE, and toluene).

The risk implications of the increased groundwater concentrations observed in the April 2007 monitoring event was evaluated by comparing to the relevant RBCs for the indoor and outdoor worker inhalation exposure as shown in Tables 3-21 and 3-22. For the volatiles indicated above, only a subset had RBCs below the solubility limit. For Lots 1 and 2 the chemicals were 1,4-DCB, benzene, and vinyl chloride. The comparison to the RBCs for each chemical indicated that the concentration observed in April 2007 data would still not cause them to be selected as COPCs. For Lots 3 and 4, the relevant chemicals were benzene, bromodichloromethane, chloroethane, chloroform, PCE, and TCE. Of these chemicals, three had been selected as COPCs and evaluated in the risk assessment (i.e., chloroform, PCE, and TCE). For the remaining chemicals, the concentration observed in the April 2007 data would not cause them to be selected as COPCs.

The risks related to inhalation of volatiles released from groundwater to ambient and indoor air presented a negligible risk for the COPCs evaluated in this risk assessment, based on historic groundwater data collected from 2001 through 2005. Use of the April 2007 groundwater

monitoring data in this risk assessment would not change those conclusions based on a comparison of the maximum concentrations from the two groundwater datasets.

7 CONCLUSIONS

This HHRA evaluated potential risk to three exposure areas of the Site: Lots 1 and 2, Lots 3 and 4, and the riverbank area (Tract A). For each exposure area, COPCs in soil and groundwater were identified for all potential exposure pathways and possible receptors at the Site. Incremental lifetime cancer risks and noncancer health effects for the COPCs were assessed for all relevant exposure pathways and potential receptor combinations. The results of the risk assessment are presented in Table 7-1. The conclusions of the risk assessment are discussed below for each of the three exposure areas evaluated at the Site.

For Lots 1 and 2, the results of the ALM indicated that exposure to lead in soil does not exceed regulatory thresholds for any receptor evaluated. For cancer and noncancer endpoints, the exposure to arsenic and DDT via the incidental ingestion of soil dominated the risks. The total HI for every receptor evaluated for Lots 1 and 2 was less than 1 indicating that adverse health affects are not expected for noncancer endpoints. The total cancer risks for the outdoor worker exposures were the highest of the receptors evaluated at Lots 1 and 2. The total cancer risk was $2x10^{-6}$ for the CTE case and $2x10^{-5}$ for the RME case. For construction workers the total cancer risk was $5x10^{-7}$ and $3x10^{-6}$ for the CTE and RME cases, respectively. For potential trespassers, the total cancer risk was $2x10^{-6}$ and $4x10^{-6}$ for the CTE and RME cases, respectively. The majority of this cancer risk for all receptors is associated with ingestion of arsenic in soil. Estimated cancer risks associated with arsenic in soil are likely be overestimates, since the risk calculations assumed 100 percent bioavailability, an extremely unlikely condition. In addition, the source of arsenic at the Site is not related to site processes and therefore is either naturally occurring (i.e., geogenic), has an anthropogenic background, or is from offsite sources. Furthermore, the EPC used in this risk assessment was driven by an extreme detected concentration.

For Lots 3 and 4, the lead concentrations in soil were below the screening-levels for soil and were therefore not considered to be a COPC for this area. The dominant chemical and exposure route for the cancer and noncancer evaluations was DDT via incidental soil ingestion. For all receptors except the construction worker, the total HI under the CTE case was less than 1, indicating that adverse health effects are not expected for these exposure scenarios. The total HI for the construction worker exposure resulted in a value of 2, indicating that adverse health effects could be associated with this exposure scenario. For the RME case, the total HI for the trespasser was less than 1. All other receptors had total HIs greater than 1, with the construction worker value of 8 as the highest, followed by the outdoor worker at 3 and the outdoor worker under the redevelopment scenario at an HI of 2. These RME results for the worker receptors are slightly above the threshold of 1 and indicate that adverse health effects could occur for the assumed exposures. The highest total incremental lifetime cancer risk of 1×10^{-4} was estimated for an outdoor occupational worker under RME conditions. The CTE cancer risk for this receptor was $2x10^{-5}$ for the outdoor worker. For the construction worker the

total cancer risk were $3x10^{-6}$ and $2x10^{-5}$ for the CTE and RME cases, respectively. Total cancer risk for the excavation worker was well below $1x10^{-6}$ under both the CTE and RME conditions.

For the riverbank area, the blood lead levels predicted for all receptors were below target a level, indicating that exposure to lead in soils is not expected to lead to adverse health effects. The total HI for all receptors under the CTE exposure was 0.1 or less. For the RME case the highest total HI was 0.3. Thus adverse health effects are not expected for any of the receptors evaluated for the riverbank. The highest total incremental lifetime cancer risks were predicted for the outdoor worker with values of $8x10^{-6}$ and $7x10^{-5}$ for the CTE and RME cases, respectively. The total cancer risk for the construction worker was $1x10^{-6}$ for the CTE case and $1x10^{-5}$ for the RME case. For the trespasser exposures, the total cancer risk was $6x10^{-6}$ for the CTE case and $1x10^{-5}$ for the RME case. The cancer risks for all receptors were dominated by the incidental ingestion of dioxin/furan and arsenic in soils. As discussed earlier, the risk predicted for arsenic are likely to be overestimates.

Total cancer risk for an indoor worker was $2x10^{-6}$ for the CTE case and 8×10^{-6} for the RME case. These risks were driven by a single extreme concentration of PCE in subsurface soil and are considered an upper-bound estimate of the potential cancer risks. The total HI for the indoor worker was well less than 0.01 for both the CTE and RME case, indicating that potential adverse health effects are not expected.

The results of the HHRA indicate that potential risks from site-related COPCs to receptors under anticipated exposure scenarios are within typical USEPA acceptable risk limits. As shown in Table 6-28, there is a substantial amount of uncertainty inherent in the estimation of human health risk and the net effect is likely to be an overestimate of potential risks. Therefore, it is likely that actual risks to individuals that may access the Site are less than the estimated values presented in this HHRA. Furthermore, future remedial activities planned for the Site will reduce if not address the potential risks predicted in this assessment. For example, paving to be conducted in Lots 1 and 2 for storm water control will reduce if not eliminate potential exposures to soil. Hot spot evaluations are being planned for soils at the Site, which could substantially reduce the total risk predicted for the worker exposures in Lots 3 and 4. Finally, the removal actions that result from the EE/CA being conducted for the in-water portions of the Site could address even the deminimus risks predicted for exposure scenarios on the riverbank.

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