APPENDIX T – BASELINE HUMAN HEALTH RISK ASSESSMENT

FINAL REMEDIAL INVESTIGATION REPORT
CASMALIA RESOURCES SUPERFUND SITE
CASMALIA, CALIFORNIA

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1×10^{-6}	one in one million
ADD	Average Daily Dose
BERA	Baseline Ecological Risk Assessment
bgs	below ground surface
BHHRA	Baseline Human Health Risk Assessment
Cal Prop 65	Safe Drinking Water and Toxic Enforcement Act of 1986
CalEPA	California Environmental Protection Agency
COPC	chemical of potential concern
CSC	Casmalia Steering Committee
CSF	Cancer Slope Factor
CSM	Conceptual Site Model
DTSC	Department of Toxic Substances Control
EPA	Environmental Protection Agency
EPC	exposure point concentration
FPP	Former Ponds and Pads
ft	feet
HEAST	Health Effects Assessment Summary Tables
HI	Hazard Index
HQ	hazard quotient
HSAA	California Hazardous Substances Account Act
IRIS	Integrated Risk Information System
J&E	Johnson and Ettinger Vapor Intrusion Model
LADD	Lifetime Average Daily Dose
mg/kg-day	milligrams per kilogram of body weight per day
NCEA	National Center for Environmental Assessment
NCP	National Contingency Plan
OEHHA	Office of Environmental Health Hazard Assessment
PAH	polycyclic aromatic hydrocarbon
PCB	polychlorinated biphenyl
PRG	Preliminary Remediation Goal
RAGS	Risk Assessment Guidance for Superfund
RCRA	Resource Conservation and Recovery Act
RfC	reference concentration
RfD	reference dose
RI/FS	Remedial Investigation/Feasibility Study
SB	Soil boring
SIC	Silty Clay
SS	Surface Soil (0 to 6 inches bgs)
SVOC	semi-volatile organic compound
TEF	Toxicity Equivalency Factors
TEQ	toxic equivalent
URF	Inhalation Unit Risk Factors
USEPA	United States Environmental Protection Agency
VOC	volatile organic compound
µg/dl	micrograms per deciliter

1.0 INTRODUCTION

The objective of the baseline human health risk assessment (BHHRA) was to evaluate potential baseline health risks associated with chemicals detected at the Casmalia Resources Superfund Site (Site). The results of the BHHRA in conjunction with the ecological risk assessment findings can be used to identify chemicals and exposure media that may pose an unacceptable risk to current and/or future receptors at the Site and to provide information for remedial planning. This risk assessment was prepared as part of this RI to evaluate potential exposures and "define risks to public health and the environment" related to soil, sediment, soil vapor, and surface water, and to subsequently provide information for the FS.

1.1 Risk Assessment Approach

This BHHRA presents the approach and methodologies that were used to estimate potential human health risks associated with residual chemicals detected in soil, sediment, soil vapor, and surface water samples collected from the Site. The overall approach that was used in this BHHRA was based on United States Environmental Protection Agency (USEPA, 1989; 1991ab; 1997a; 2002; 2004; 2006a,b,c) and Cal-EPA guidance documents (2000; 2003). The BHHRA consists of five major components organized in the following manner:

- **Data Review and Evaluation:** A review of available data to characterize the Site and identify data gaps; to define the nature and extent of environmental contamination identified at the Site; and to identify Site-related chemicals of potential concern (COPCs) (defined as potentially hazardous chemicals associated with the Site that are present at concentrations higher than background levels);
- **Exposure Assessment:** An assessment of the magnitude, frequency, duration, and routes of potential human exposure to Site-related COPCs. The exposure assessment considers both current and likely future Site uses and is based on complete exposure pathways to actual or probable human receptors (i.e., general groups that could come in contact with Site-related COPCs). The exposure scenarios are summarized in the Conceptual Site Model (CSM), which includes the sources, affected media, release mechanisms, and exposure pathways for each identified receptor population;
- **Toxicity Assessment:** A presentation of available information to identify the nature and degree of toxicity and to characterize the dose-response relationship (the relationship between magnitude of exposure and magnitude of potential adverse health effects on each receptor) for each COPC;
- **Risk Characterization:** A synthesis of exposure and toxicity information to yield quantitative estimates of potential cancer risks and noncancer hazards to defined receptor populations; and
- **Uncertainty Analysis:** A discussion of the uncertainties associated with each of the four previous steps to assist decision-makers in evaluating the risk assessment results in the context of the assumptions and variability in the data used.

For purposes of this BHHRA, the Site includes both Zone 1 and Zone 2, as depicted on **Figure T-1.** Zone 1 (the Site) includes the inactive Class I hazardous waste management facility and comprises approximately 252 acres. Zone 2 (offsite) includes the area encompassing the extent of Site-related contamination or potential contamination outside the Zone 1 boundary.

Because potential human health effects from exposure to Site-related chemicals are evaluated based on current and potential future land use scenarios, an important step in developing the risk assessment approach was to define baseline conditions. As discussed in **Section 7.1** of the RI Report, the BHHRA was developed assuming that certain remedies are already in place. In this way, any pathways of exposure considered incomplete, because of the existing or presumptive remedies, were not evaluated in the BHHRA. The following areas of the Site have been capped: (1) the P/S Landfill, and (2) the EE/CA Area, which includes the Heavy Metals, Caustics/Cyanides and the Acids Landfill and the areas between these landfills. As discussed earlier in this RI Report, the PCB Landfill located adjacent to the P/S Landfill will also be capped.

In addition, the CSC and USEPA have agreed that the two treated liquids impoundments, Pond A-5 and Pond 18, will be drained as part of Site remediation. As a result, potential exposures to treated liquid impoundment waters were not considered in the BHHRA. However, impoundment sediments were evaluated as exposed surface soils, since the impoundments will be drained. As a part of this assumption, it is assumed that once drained, the treated liquid impoundment area will be graded as appropriate to minimize future collection of water.

1.2 Site Background

A detailed discussion of the Site background, history and use information, as well as previous investigations conducted at the Site, can be found in **Section 2** of the RI Report.

1.3 Risk Assessment Organization

The remainder of this BHHRA is organized in the following sections:

- Section 2 presents the data review and evaluation process, including the identification of chemicals of potential concern (COPCs) and methods for evaluating background concentrations of inorganic chemicals;
- Section 3 presents the exposure assessment approach by describing the conceptual site model, including the identification of potential human receptors, the evaluation of possible exposure pathways, as well as the methods for evaluation of analytical data and exposure point concentrations;
- Section 4 describes the approach for selecting chemical-specific toxicity values;
- Section 5 describes the risk characterization process and proposed risk management criteria;
- Section 6 includes a discussion of the uncertainty analysis;
- Section 7 presents the summary and conclusions for the BHHRA; and
- Section 8 presents the references cited in this document.

1.4 Definitions

Terms used in this RA have specific meaning with respect to the Site or the processes described. The following are definitions of select terms:

1) The 252-acre inactive Class I hazardous waste management facility will herein be referred to as the "Site";
2) A "chemical of potential concern" (COPC) is a potentially site-related chemical with data of sufficient quality for use in a quantitative BHHRA;
3) A "human receptor" is a hypothetical individual who may be exposed to compounds in the environment. Receptors are often identified by the behaviors that determine how or with what intensity they may be exposed, such as "workers" or "residential receptors";
4) An "exposure route" is a mechanism of uptake. Environmentally relevant exposure routes typically include inhalation, ingestion, and absorption through the skin;
5) An "exposure pathway" is defined by USEPA (1989) as consisting of four elements: (a) source and mechanism of chemical release; (b) a retention or transport mechanism through an environmental medium; (c) a point of potential contact with the impacted medium (i.e., an exposure point); and (d) an exposure route at the exposure point. If any of these elements is missing, the exposure pathway is considered "incomplete", and compound uptake via pathway would not occur; and
6) An "exposure point concentration" (EPC) is the concentration of a COPC in a medium at the location where a receptor is assumed to make contact with that medium. Depending on the nature of the exposure, an EPC may be estimated at a specific point, or may be averaged about an "exposure area" (e.g., the soil surface), using the 95% upper confidence limit (UCL).

2.0 DATA REVIEW AND EVALUATION

An initial step in the risk assessment process is an evaluation of available data to develop a data set for use in the BHHRA and identify media-specific chemicals of potential concern (COPCs). This section presents a summary of the data evaluation steps that were conducted for the BHHRA. The methodology that was used to identify the COPCs for the Site and contiguous areas is presented in below. The data evaluation steps that were conducted to develop a risk assessment dataset, identify media-specific COPCs, and calculate exposure point concentrations (EPCs) for evaluation in the BHHRA were previously discussed in **Section 7** of the RI Report.

2.1 Data Evaluation

As discussed in Section 5 of the RI Report, the project database was constructed with several phases of RI sampling that have been conducted since 2004 in addition to limited historical data (background and West Canyon Spray area soil data). The database includes soil, sediment, surface water, soil vapor and groundwater data. To prepare a dataset for quantitative risk assessment purposes, the soil, sediment, surface water and soil vapor data were first evaluated for usability and then processed through several steps.

The data evaluation was conducted in addition to the procedures for field sampling, chain-of-custody, laboratory analysis, reporting and data validation that were conducted in accordance to the QAPP. The data evaluation was consistent with guidance provided by USEPA in *Risk Assessment Guidance for Superfund* (RAGS) (USEPA, 1989), *Guidance for Data Usability in Risk Assessments* (USEPA, 1992), *Data Quality Assessment: Statistical Methods for Practitioners (USEPA, 2006a)* and guidance for calculating EPCs (USEPA, 2007 a,b). The results of the data evaluation and validation were incorporated into the final dataset used for the risk assessment. Data were deemed useable for risk assessment with the exception of R-qualified, rejected data. Rejected data was not included in the risk assessment database but estimated (j-qualified) data was included. In addition, it was determined that sufficient samples were collected for calculating exposure point concentrations.

For cases where a field duplicate sample was present or multiple analyses were present for the same chemical in a sample, a single representative concentration for the sample was selected as follows:

(1) If there was a detection in both samples the higher concentration was selected;
(2) If there was a detection in one sample but not the other, the detected concentration was selected; and
(3) If both samples were nondetect the lowest method detection limit was selected and appropriate techniques for handling nondetect data were applied in calculating statistics later in the data evaluation.

Finally, for dioxin and polychlorinated biphenyls (PCB) congener data, total dioxin and PCB toxic equivalents (TEQ) were calculated. Dioxins/furans and PCBs are complex halogenated aromatic hydrocarbon mixtures made up of chemically-related chemicals. The 2,3,7,8-tetrachlorodibenzo-p-dioxin congener has been the most extensively studied of these halogenated aromatic hydrocarbons and is thought to be the most toxic chemical within the dioxin family. Because of their complex nature and the lack of specific toxicity information for each of the individual chemicals, dioxin/furans and some of the PCB congeners that exhibit dioxin-like behavior are evaluated in terms of their relative toxicity to that of 2,3,7,8-TCDD using Toxicity Equivalency Factors (TEFs). The approach used to calculate the total TEQ concentrations is described below:

Step 1. For each sample, select the detected concentrations of each of the 17 2,3,7,8-substituted dioxin/furan congeners or each of the 12 dioxin-like PCB congeners;
Step 2. Multiply each congener concentration by the appropriate TEF for the specific congener (presented in Section 7 of the RI Report) (e.g., human total TEQs should be calculated with mammalian TEFs); and
Step 3. Sum the resulting values from Step 2 to calculate total TEQ concentrations.

2.2 Selection of COPCs

All data determined to be of sufficient quality were carried forward into the COPC selection process, which was discussed in detail in **Section 7.3** of the RI Report. COPCs were selected for each environmental media (soil, sediment, surface water and soil vapor) for inclusion in the BHHRA. COPCs are defined as chemicals clearly associated with the Site and present at concentrations higher than background levels.

Prior to selecting the COPCs, the chemical dataset was filtered based on media and depth as appropriate. For soil and sediment, samples taken from depths less than or approximately equal to 5 feet below ground surface (including data from 5 to 5.5 feet bgs) were selected. The filtered dataset included all Study Areas (including offsite drainages), excluding background, historical West Canyon data, PCB landfills, and capped landfill areas. There was no division by depth for surface water and soil vapor. Study Areas include the following:

Terrestrial Uncapped Areas:

- RCRA Canyon;
- Liquid Treatment Area;
- West Canyon Spray Area;
- Burial Trench Area;
- Maintenance Shed Area;
- Central Drainage Area;
- Administration Building Area;
- Roadway Areas;
- Remaining Onsite Areas; and
- Former pond and pad areas south of the perimeter source control trench (PSCT).

Onsite Ponds:

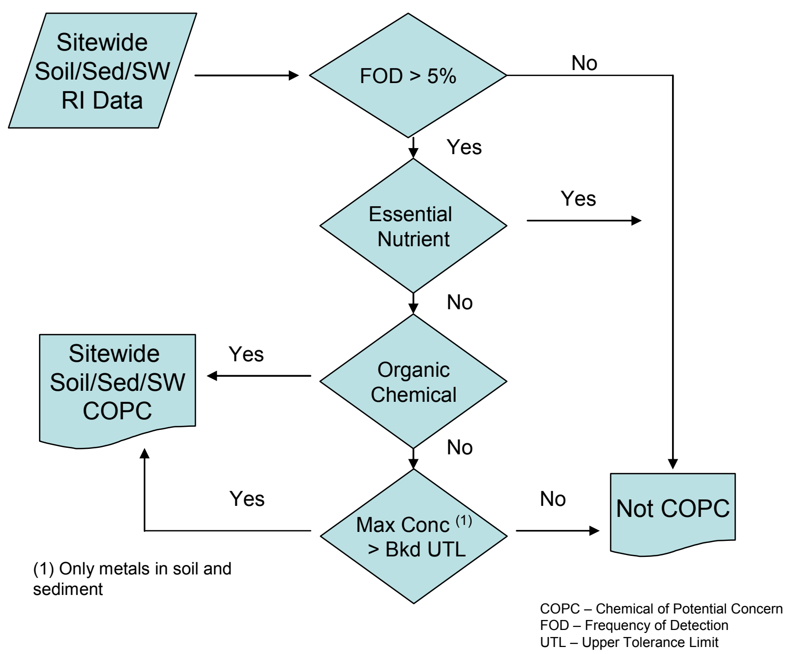
- A-Series Pond;
- RCF Ponds;
- Pond A-5;
- Pond 13; and
- Pond 18.

Offsite Drainages:

- North Drainage;
- A Drainage;
- B Drainage;
- Upper C Drainage; and
- Lower C Drainage.

Sitewide prevalence tables were generated using the Sitewide dataset, which was analyzed on a parameter-by-parameter basis. These tables are presented in **Attachment X-1** in **Appendix X**. Based on these prevalence tables, an analysis was performed to generate Sitewide COPCs. Chemicals were identified as a COPC on a per-matrix basis based on three criteria: (1) prevalence for organic and inorganic chemicals, (2) elimination of essential nutrients, and (3) comparison to background for inorganic chemicals. COPCs for soil and sediment were selected for the depth interval evaluated in this BHHRA, 0 to 5 feet below ground surface (bgs) from the Sitewide dataset (including data from 5 to 5.5 feet bgs). The list of COPCs was then applied both Sitewide and on a Study Area-specific basis. The figure below presents an overview of the Sitewide COPC selection process and is discussed in more detail in **Section 7** of the RI Report. For soil vapor, all detected chemicals were included in the BHHRA.

COPC Selection Process



An additional analysis was conducted for each Study Area to determine if additional "Study Area-specific" COPCs should be added to the BHHRA. The purpose of this more detailed screening process was to address agency concerns that there may be localized detections of a chemical that should be evaluated. Study Area-specific prevalence tables were developed to identify those chemicals with a prevalence of greater than 5% within the Study Area. This list was then compared to the Sitewide COPC list to see if any new chemicals were identified as COPCs. This list was further screened to include only those chemicals with at least three detections in a Study Area. Finally, since many of the chemicals were detected at relatively low concentrations, the list was further screened against the residential PRGs for the BHHRA. The Study Area-specific COPCs are presented in **Attachment X-2 in Appendix X**. Any chemical retained after this additional screening process was included as a COPC only in the Study Area in which it was selected.

Table T-1 presents the list of COPCs by media that were evaluated in this BHHRA.

3.0 EXPOSURE ASSESSMENT

This section describes the receptors and exposure pathways that were evaluated in the BHHRA. The objectives of an exposure assessment are to identify receptor groups (populations) that may be exposed to chemicals in impacted media (e.g., soil and surface water), the exposure pathways, and the route of potential intake. In addition, the chemical concentrations to which the receptors are potentially exposed (exposure point concentrations, EPCs) and the frequency, magnitude, and duration of these potential human exposures (exposure parameters) must be estimated. The exposure assessment focuses on the COPCs detected in soil, sediments, soil vapor, and surface water at the Site. The primary routes of potential human exposure to chemicals detected at the Site include incidental ingestion, dermal contact, inhalation of fugitive dust and inhalation of vapors in indoor and outdoor air.

The following steps are considered in the exposure assessment:

- Identification of potentially exposed receptor populations;
- Identification of complete exposure pathways;
- Estimation of exposure point concentrations for specific pathways; and
- Estimation of chemical intakes for receptor populations associated with each complete exposure pathway.

The end product of the exposure assessment is a measure of chemical intake as an estimated average daily dose (ADD) that integrates the exposure parameters for the receptors of concern (e.g., contact rates, exposure frequency, and duration) with the EPCs for the media of concern. These chemical intakes, or ADDs, are then used in conjunction with chemical-specific toxicity values (e.g., noncancer reference doses and cancer slope factors) to arrive at an estimate of potential health risks for the receptors of concern.

The exposure assessment follows USEPA recommendations (USEPA, 1995) to develop “reasonable maximum exposure” (RME or upper-bound) for the identified exposure scenarios. The RME incorporates a number of conservative assumptions in estimating chemical intake rates and characteristics of the receptor population. The RME is thus an estimate of the highest exposure that is reasonably expected to occur at the Site and may overestimate the actual risk for the majority of the population. For RME estimates of exposure, reasonable conservative modeling assumptions (those which tend to overestimate exposure point concentrations) and upper-bound default values for most exposure parameters were used.

This section describes the steps that were followed in the exposure assessment.

3.1 Conceptual Site Model

The Conceptual Site Model (CSM) identifies potential chemical sources, release mechanisms, transport media, routes of chemical migration through the environment, exposure media, and potential receptors. Receptors that may be potentially exposed to Site-related chemicals are identified and the likelihood of their potential exposures assessed through consideration of the current and the anticipated future use of the Site.

The CSMs for uncapped areas and surface water at the Site, presented in **Figures T-2** and **T-3**, respectively, represents the understanding of the sources of chemicals of potential concern, the means by which they are released and transported within and among media, and the exposure pathways and routes by which both human receptors may contact them. The major components of the CSMs are discussed below.

3.1.1 Sources

Review of previous investigation results helped to identify potential sources of contamination including, the five landfills, burial trenches, injection wells, active ponds and former ponds and pads (FPP), former RCRA Landfill, underground storage tanks, CNS Area, Liquids Treatment Areas, and roadways. Once chemicals are released into the environment, secondary sources of contamination may include contamination in surface water and sediment, surface and subsurface soil, and groundwater.

As discussed in earlier sections of the RI Report, portions of Zone 1 will have undergone remediation prior to completion of the RI. Therefore, in this BHHRA, assumptions are made that the P/S Landfill, PCB Landfill and the EE/CA Area will be capped and the two treated liquids impoundments (Ponds A-5 and 18) will be drained.

3.1.2 Mechanisms of Release and Transport

Several primary and secondary release and transport mechanisms may exist at the Site. Chemicals may volatilize from surface water, soil, or groundwater. Chemicals absorbed to soil may become wind-blown as fugitive dust or transported in surface water runoff. Chemicals may migrate to groundwater from direct contact with waste or contaminated soil and may subsequently be transported in dissolved phase to other parts of the Site. Each one of these conditions can possibly occur within Zone 1.

3.1.3 Exposure Pathways

An exposure pathway describes a specific environmental mechanism by which an individual (receptor) can be exposed to COPCs present at or originating from a source. The following five elements comprise a complete exposure pathway:

- A source of chemical;
- A mechanism of chemical release to the environment;
- An environmental transport medium (e.g., soil or air);
- A point of potential human contact with the medium; and/or
- A means of entry (i.e., intake route) into the body (e.g., ingestion).

There must be a complete exposure pathway from the source (i.e., from soil, air, or surface water) to human receptors in order for chemical intake to occur. If a potential exposure pathway is considered incomplete for human receptors, no chemical intake occurs and hence, no human health effects are associated with Site-related COPCs.

Given the characteristics of the COPCs and conditions at the Site and adjacent areas, several exposure pathways may be potentially complete. Exposure pathways were selected based on current and future use of the Site. The CSMs, as presented in **Figures T-2** and **T-3**, depict potential exposure pathways and a determination as to their completeness.

Based on current, available information, the following exposure pathways were considered potentially complete for human receptors at the Site:

- Incidental ingestion of COPCs in soil, sediment, or surface water;
- Contact with soil, sediment, or surface water and absorption of COPCs through the skin;
- Inhalation of COPCs in windborne dust generated from soil or sediment;
- Inhalation of vapors emanating from soil, sediment, or surface water into outdoor air;
- Inhalation of vapors emanating from soil vapor into outdoor air;
- Inhalation of vapors emanating from soil vapor into indoor air; and
- Ingestion of beef.

Incomplete exposure pathways are those pathways in which constituent intakes are considered to be nonexistent. Insignificant exposure pathways are those pathways in which constituent intakes are considered to be relatively insignificant in comparison to other exposure pathways. USEPA guidance defines an insignificant pathway as one that has an exposure estimated to be two or more orders of magnitude less than by other pathways (for the same receptor); a pathway is also considered insignificant if the risks are much less for that pathway, or if the likelihood of exposure by that pathway is very small (USEPA, 1989). Potential exposure pathways that are significant are indicated as being complete and potential exposure pathways that may occur under certain Site conditions are indicated as being potentially complete.

A well survey was conducted as a part of the RI/FS (Appendix N). Of the 38 known wells in the Site vicinity, the closest active well to the Casmalia Resources Superfund Site is situated approximately 1/3 mile northwest of the western Site boundary, along Casmalia Creek (URS Well ID #29). This well is owned by the former site operator and is currently under the control of EPA and the CSC. The well is currently used by the Site as non-potable water supply. The only other wells proximal to the Site are also located along Casmalia Creek (URS Well ID #28, 30 and 31; all inactive). The next closest wells are towards the south and southeast, located within or adjacent to the town of Casmalia. These wells lie at distances of between 1.2 to 1.7 miles from the nearest Site boundary. These wells are owned primarily by private parties and used for agricultural purposes. The rest of the identified wells, to the north, south and east, are located at least two miles away from the Site boundaries.

Based on the well survey information, the groundwater beneath and in the immediate vicinity of the Site is not currently being used for potable water. In addition, groundwater extraction for purposes of potable water will not be allowed in the future. Therefore, this exposure pathway was not considered complete and was not evaluated in the BHHRA. (

3.1.4 Receptors of Concern

The current land-use of Zone 1 is a hazardous waste management facility. Land-use surrounding the Site includes open-space, cattle grazing and oil-field development. The majority of land that adjoins the Site (Zone 2) is owned and controlled by the CSC. There are privately held land(s) that currently adjoin the Site on the southwest border of the Site. These lands are being used for cattle-grazing. Property ownership adjacent to Zone 1 is depicted in Figure T-4.

The CSC is in the process of working with EPA to place deed restrictions on both the parcels of land that the Site occupies and approximately 1,000 acres that surround the Site (i.e. in Zone 2). Future residential development is unlikely based on current zoning and the deed restrictions that will be in place. Nevertheless, a hypothetical future residential exposure scenario for Zone 2 was included in this BHHRA as the deed restriction process has not been completed. Residential exposure pathways are indicated as only potentially complete in the CSM due to the hypothetical nature of this pathway.

The following receptors may be potentially exposed to Site-related chemicals within Zone 1:

- Onsite workers maintaining the liquids treatment area, surface impoundments, and landfill covers and drainage structures;
- Trespassers; and
- Ranchers using the NTU road to access their lands.

The following receptors were also evaluated in the BHHRA since they are potentially exposed to Site-related chemicals within Zone 2:

- Ranchers working the fields along the southwest border of Zone 1;
- Consumers of beef raised in the fields near Zone 1;
- Recreational users of the drainage areas; and
- Hypothetical residents living near the Site.

Middle school- and high school-aged children (11-17 year olds) were included as part of the evaluation for the recreational scenario. Based on professional judgment, recreational use of the surrounding area of the Site, within Zone 2, is not expected. Access to this area is considered limited, no trails have been observed, and the area is used primarily for cattle grazing. Although the area surrounding the Site does not appear to be used for recreational purposes, this scenario was evaluated in this BHHRA as a conservative approach. Moreover, an assumption of once per month as the exposure frequency is considered conservative for this particular receptor given that person has a low potential for recreating within Zone 2.

The following table summarizes the receptor groups, exposure medium and exposure pathways under current and potential future land use conditions that were quantitatively evaluated in this BHHRA.

Receptor Population	Exposure Medium	Study Area	Exposure Pathways
Commercial/ Industrial Worker	Onsite Soil/Sediment SS = 0-6 inches bgs SB = 0-5 feet bgs	**Onsite Soil =** • Administration Building • Burial Trench • Central Drainage • FPP • Liquid Treatment • Maintenance Shed • RCRA Canyon • Roadways • Remaining Onsite • West Canyon Spray **Onsite Sediment =** • Pond 18 • Pond A-5	• Incidental Ingestion • Dermal Contact • Outdoor Fugitive Dust/Vapor Inhalation • Indoor Air Vapor Inhalation (Administration Building only)

Receptor Population	Exposure Medium	Study Area	Exposure Pathways
	Onsite Surface Water	• A-Series Pond • Pond 13 • RCF Pond	• Incidental Ingestion • Dermal Contact • Outdoor Inhalation
	Onsite Soil Vapor	• Onsite Soil Vapor	• Outdoor Vapor Inhalation
Trespasser	Onsite Soil/Sediment SS = 0-6 inches bgs SB = 0-5 feet bgs	**Onsite Soil =** • Administration Building • Burial Trench • Central Drainage • FPP • Liquid Treatment • Maintenance Shed • RCRA Canyon • Roadways • Remaining Onsite • West Canyon Spray **Onsite Sediment =** • Pond 18 • Pond A-5	• Incidental Ingestion • Dermal Contact • Outdoor Fugitive Dust/Vapor Inhalation
	Onsite Surface Water	• A-Series Pond • Pond 13 • RCF Pond	• Incidental Ingestion • Dermal Contact
Recreator	Offsite Soil SS = 0-6 inches bgs SB = 0-5 feet bgs Offsite Sediment SS = 0-6 inches bgs	**Offsite Soil =** • B Drainage **Offsite Sediment =** • North Drainage • A Drainage • Lower Drainage • Upper C Drainage	• Incidental Ingestion • Dermal Contact • Outdoor Fugitive Dust/Vapor Inhalation
Rancher	Offsite Soil SS = 0-6 inches bgs SB = 0-5 feet bgs Offsite Sediment SS = 0-6 inches bgs	**Offsite Soil =** • B Drainage **Offsite Sediment =** • North Drainage • A Drainage • Lower Drainage • Upper C Drainage	• Incidental Ingestion • Dermal Contact • Outdoor Fugitive Dust/Vapor Inhalation • Ingestion of Beef
	Onsite Roadway Soil SS = 0-6 inches bgs SB = 0-5 feet bgs	• Roadways	• Incidental Ingestion • Dermal Contact • Outdoor Fugitive Dust/Vapor Inhalation
Hypothetical Offsite Resident	Offsite Soil SS = 0-6 inches bgs SB = 0-5 feet bgs Offsite Sediment SS = 0-6 inches bgs	**Offsite Soil =** • B Drainage **Offsite Sediment =** • North Drainage • A Drainage • Lower Drainage • Upper C Drainage	• Incidental Ingestion • Dermal Contact • Outdoor Fugitive Dust/Vapor Inhalation
	Offsite Soil Vapor	• Offsite Soil Vapor	• Indoor Air Vapor Inhalation

Receptor Population	Exposure Medium	Study Area	Exposure Pathways
	Onsite Soil SS = 0-6 inches bgs SB = 0-5 feet bgs	**Onsite Soil =** • Administration Building • Burial Trench • Central Drainage • FPP • Liquid Treatment • Maintenance Shed • RCRA Canyon • Roadways • Remaining Onsite • West Canyon Spray	• Outdoor Fugitive Dust/Vapor Inhalation

Notes:
SS = refers to surface soil; SB = refers to shallow soil

3.2 Exposure Point Concentrations

Exposure point concentrations (EPCs) are the concentrations of chemicals in environmental media to which receptors may be exposed through defined exposure pathways. EPCs were estimated for each of the environmental media associated with complete and potentially complete pathways identified in the CSM. These media and pathways include the following:

- Surface (0 to 6 inches bgs) and shallow soil (0 to approximately 5 feet bgs; this also includes data from 5 – 5.5 feet bgs) considered for incidental ingestion, dermal contact, and inhalation of fugitive dust and vapor pathways, as well as ingestion of beef;
- Surface (0 to 6 inches bgs) and shallow sediment (0 to approximately 5 feet bgs) considered for incidental ingestion, dermal contact, and inhalation of fugitive dust and vapor pathways;
- Soil vapor considered for the vapor inhalation pathway; and
- Surface water considered for incidental ingestion, dermal contact and inhalation pathways.

Evaluating data collected from shallow soils (0 to approximately 5 feet bgs) accounts for potential future exposure to the subsurface soils if the Site and adjacent areas become reconfigured and deeper soils are brought to the surface and made available for direct contact exposures (e.g., via incidental ingestion, dermal contact) and outdoor air inhalation of fugitive dust and vapors. While individuals are unlikely to have direct contact with impacted soil at depths greater than 5 feet bgs, the potential does exist for VOCs to migrate from beneath the subsurface. Therefore, onsite and offsite soil vapor samples collected at depths of greater than 5 feet bgs were used in the BHHRA. Onsite soil vapor samples were used to evaluate the outdoor air inhalation pathway for a commercial/industrial worker and a hypothetical resident living near the Site, respectively. Offsite soil vapor samples were used to evaluate the indoor air inhalation pathway for a hypothetical resident receptors living near the Site. Additionally, VOCs detected in onsite soil samples collected from the Administration Building area were evaluated for the indoor air inhalation pathway for a commercial/industrial worker.

The soil data was used as soil vapor sampling was not conducted in the Administration Building area. The soil vapor sampling was focused on the primary source areas in the northern portion of the Site along the landfill perimeters, Burial Trench area and along the PSCT south of the

Central Drainage Area with the nearest soil vapor sample over 500 feet from the Administration Building Area. As a result the soil vapor data was not usable to evaluate the Administration Building Area. However several soil samples were collected immediately adjacent to or in close proximity to the Administration Building and analyzed for VOCs with sufficient reporting limits. These data are considered adequate for evaluating the vapor intrusion pathway for this area.

For surface water exposures, historical seep data from the four seeps, A-Series Seep, CA Seep, Caustic LF Seep, and Seep 9B, was not used in the exposure assessment. The seep data were mainly collected in 1997 and 1998 (and not as part of the RI data) and were not validated to the same level as the rest of the RI data. In addition, the seeps are no longer present at the Site and therefore do not pose a complete exposure. A qualitative discussion of the historic seep data and potential seep impacts should they return is provided in the Uncertainty Section.

EPCs were derived for the Site using two primary approaches. For all exposure areas, EPCs were derived as point estimates, represented by the 95% Upper Confidence Limit (95% UCL) or data maximum, using ProUCL version 4.0 and the methodology outlined in the USEPA guidance for calculating EPCs (USEPA 2006, 2007a, and 2007b). Where possible, the 95% UCL was selected over the data maximum per USEPA guidance. Since the EPC term represents the average exposure contacted by an individual over an exposure area during a long period of time, EPCs should be estimated by using an average value (such as an appropriate 95% UCL of the mean) and not by the maximum detected concentration. This is because it is unlikely that an individual will visit the location of the maximum detected value all of the time.

3.2.1 EPCs Derived Using ProUCL

In early 2007, USEPA released statistical software called ProUCL Version 4.0 (ProUCL 4.0) to facilitate the calculation of 95% UCLs (USEPA, 2007a and 2007b). ProUCL 4.0 is an upgrade of ProUCL Version 3.0 and contains statistical methods to evaluate both full environmental data sets without nondetect values and data sets with nondetect values (also known as left-censored data sets).

Prior to calculating 95% UCLs for each exposure area with ProUCL, the data were screened with respect to sample size and number of detects as follows:

1. If a chemical was not detected in any sample for a given exposure area and media, it was assumed to not be present, so an EPC was not calculated;
2. If the number of samples in an exposure area was less than 8, then the maximum detected concentration was used as the EPC; and
3. If the number of detects was less than 5, then the maximum detected concentration was used as the EPC.

If a sufficient number of samples and detections were present for a COPC, ProUCL 4.0 was used to calculate the 95% UCL. ProUCL 4.0 can calculate UCLs using up to 15 different parametric and nonparametric statistical methods. Some of the methods (e.g., Kaplan-Meier method, regression on order (ROS) methods) are applicable to left-censored data sets having multiple detection limits. The optimal method(s) for a particular data are identified by the software based on USEPA's numerical experiments with hypothetical data sets with a wide range of statistical properties, such as distribution shape, sample size, percent non-detects, and skewness (USEPA, 2006b). If multiple UCLs were identified as being equally plausible, the relative percent difference (RPD) in 95% UCLs was evaluated. If the RPD was less than 5%, the EPC was determined by the method that yields the highest value. If the RPD was greater than 5%, then professional judgment was used to select the method that generally exhibits the most consistent performance according to USEPA guidance (USEPA, 2007a). EPCs were derived using the same methodology for soil, sediment, soil vapor and surface water. For soil vapor, the maximum detected concentrations were used in this BHHRA. A summary of the EPCs by exposure area and media is presented in **Tables T-2** through **T-9**. Detailed information on the EPCs is presented in **Appendix X**.

EPCs for the outdoor and indoor air exposure pathways in this BHHRA were further developed using fate and transport modeling as described below.

3.2.2 EPCs for Air Pathways

3.2.2.1 Fugitive Dust Emissions from Onsite Soil/Sediment

Chemicals detected in soil at the Site and adjacent areas may become airborne due to fugitive dust emissions. Inorganic compounds (e.g., SVOCs and metals) can adhere to soil particles then become airborne due to wind erosion, which could generate dust containing COPCs. Exposure to these chemicals may then occur via inhalation of airborne fugitive dust. Inhalation exposure to non-volatile compounds is typically minor in fugitive dust when compared to direct ingestion exposure (USEPA, 2002). Nevertheless, a relationship can be estimated between the chemical concentration in soil and the corresponding concentration in air (secondary media) attributable to fugitive dust emissions from soil.

Potential exposure to airborne dust is estimated using a particulate emission factor (PEF) that relates the concentration of a soil constituent to the concentration of dust particles in air. The PEF represents an annual average emission rate based on wind erosion. The PEF equation can be found in Section 4.2.3 (Equation 4-5: Derivation of the PEF) of the *Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites* (USEPA, 2002). The emissions part of the PEF equation is based on the "unlimited reservoir" model developed to estimate PM_{10} emissions (particulate matter less than 10 micrometers in diameter [PM_{10}]) due to wind erosion (Cowherd et al., 1985).

The PEF was derived using the following equation (USEPA, 2002):

$$PEF = \frac{(Q/C \times 3600)}{[(0.036 \times (1 - G) \times (U_m/U_t)^3 \times F_x]}$$

Where:

PEF	=	particulate emission factor cubic meters per kilogram (m^3/kg)
Q/C	=	inverse of mean concentration at center of a 10-acre square source (g/m^2-s per kg/m^3)
G	=	fraction of vegetative or other cover (0.5, unitless)
U_m	=	mean annual wind speed (3.70 m/s, average Site-specific)
U_t	=	equivalent threshold value of wind speed at 7 meters (11.32 m/s, USEPA 2002 default)

F_x	=	function dependent on U_m/U_t derived using Cowherd et al. (1985) (0.022 unitless, based on average Site-specific wind speed)
0.036	=	respirable fraction (g/m^2-hr)

The dispersion part of the PEF equation includes the dispersion coefficient (Q/C) in units of grams per square meter-second per kilogram per cubic meter (g/m^2-s per kg/m^3). The Q/C term was generated using the Industrial Source Complex model and varies depending on the source area, city, and climatic zone. This term accounts for the dispersion of particulate matter, once emitted, and was estimated using the following equation (USEPA, 2002):

$$(Q/C) = A \times \exp\left[\frac{(\ln A_{SITE} - B)^2}{C}\right]$$

Where:

A_{SITE}	=	aerial extent of soil impact (10 acres)
A	=	constant = 11.911 (USEPA, 2002)
B	=	constant = 18.4385 (USEPA, 2002)
C	=	constant = 209.7845 (USEPA, 2002)

The coefficients A, B, and C are for the Los Angeles area and are published in the *Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites* (Exhibit E-3 in USEPA, 2002). A Q/C value of 41.21 g/m^2-s per kg/m^3 was estimated as the inverse of the mean concentration at the center of a 10-acre source in Los Angeles, California (USEPA, 2002) based on a reasonable average size for the various Study Areas. The PEF was therefore estimated at $1.1 \times 10^{+10}$ m^3/kg for the commercial/industrial worker, trespasser, and rancher potentially exposed to onsite soil/sediment. For a hypothetical offsite resident potentially exposed to onsite soil/sediment, a different Q/C term was estimated using the following equation (USEPA, 2002):

$$(Q/C_{adj}) = A \times \exp\left[\frac{(\ln A_{SITE} - B)^2}{C}\right]$$

Where:

Q/C_{adj}	=	Inverse of the ratio of the geometric mean air concentration at the emission flux at the Site boundary (g/m^2-s per kg/m^3)
A_{SITE}	=	aerial extent of soil impact (10 acres)
A	=	constant = 15.7133 (USEPA, 2002)
B	=	constant = 21.8997 (USEPA, 2002)
C	=	constant = 269.8244 (USEPA, 2002)

The coefficients A, B, and C are for the Los Angeles area and are listed in Exhibit E-5 of USEPA, 2002. A Q/C_{adj} value of 65.22 g/m^2-s per kg/m^3 was estimated with the resulting PEF_{adj} estimated at $1.7 \times 10^{+10}$ m^3/kg for an adjacent resident.

The hypothetical offsite resident evaluation is overly conservative in that the modeling assumes the resident is located adjacent to the Study Area being evaluated. In reality, the resident would be located some distance from the Study Area boundary thereby resulting in lower estimates of exposure.

Using COPC soil concentrations (Cs) and the estimated PEF, outdoor air concentrations (Ca) were estimated using the following equation:

$$C_a = \frac{C_s}{PEF}$$

Where:

C_a = concentration of COPC in outdoor air (mg/m^3)
C_s = concentration of COPC in soil (mg/kg)
PEF = particulate emission factor (mg/kg per mg/m^3 or, m^3/kg)

Derivation of the PEF for the commercial/industrial worker, trespasser, rancher, and hypothetical offsite resident potentially exposed to onsite soil/sediment is presented in **Table T-10**.

3.2.2.2 Fugitive Dust Emissions from Offsite Soil/Sediment

Fugitive dust also has the potential of migrating from impacted offsite areas adjacent to the Site. This pathway was also evaluated in this BHHRA for the recreator, rancher, and hypothetical offsite resident potentially exposed to offsite soil/sediment using the same screening level model described above for onsite fugitive dust emissions, as a conservative approach. Derivation of the PEF for these receptors is also presented in **Table T-10**.

3.2.2.3 Vapor Emissions to Outdoor Air from Onsite Soil/Sediment

VOCs were detected in soil at the Site. Because these compounds are volatile, individuals could potentially be exposed to vapors migrating through the soil to the surface. Outdoor vapor concentrations are typically negligible considering the significant quantity of ambient air diluting the vapor emissions. Although this pathway is considered potentially insignificant, it was further evaluated in this BHHRA.

Potential migration of vapors from soil to outdoor air was estimated using the volatilization factor (Equation 4-8: *Derivation of the VF*), as presented in Section 4.2.3 of the *Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites* (USEPA, 2002). The VF was used in this BHHRA to estimate outdoor inhalation exposures for commercial/industrial workers, trespassers, recreators, ranchers, and hypothetical offsite residents. Default parameters for the Los Angeles Area were used (e.g., the Q/C term as discussed above for the PEF formula). The VF term incorporates the dispersion factor.

Chemical-specific VFs were derived using the following equation (USEPA, 2002):

$$VF = \frac{Q/C \times \left(10^{-4}\ \frac{m^2}{cm^2}\right) \times (3.14 \times D_A \times T)^{1/2}}{(2 \times Pb \times D_A)}$$

Where:

VF	=	volatilization factor (m^3/kg)
Q/C	=	inverse of mean concentration at a 10-acre Site*
D_A	=	chemical-specific apparent diffusivity (cm^2/s)
T	=	exposure interval (receptor-specific, sec)
Pb	=	dry soil bulk density (silty clay [SIC] default = 1.38 g/cm^3)

* The Q/C value of 41.21 g/m^2-s per kg/m^3 was used for a commercial/industrial worker, trespasser, and rancher potentially exposed to onsite soil/sediment. The Q/C_{adj} value of 65.22 g/m^2-s per kg/m^3 was used for a hypothetical offsite resident potentially exposed to onsite soil/sediment (**Table T-10**).

And where:

$$D_A = \frac{\left(\frac{D_{air}\ \theta_a^{\ 3.33}}{\theta_T^{\ 2}}\right) + \left(\frac{D_{water}\ \theta_w^{\ 3.33}}{H'\theta_T^{\ 2}}\right)}{Pb \times Koc \times foc + \theta_w + \theta_a \times H'}$$

Where:

DA	=	chemical-specific apparent diffusivity (cm^2/s)
D_{air}	=	chemical-specific vapor diffusion coefficient in air (cm^2/s)
D_{water}	=	chemical-specific molecular diffusion coefficient in water (cm^2/s)
θ_a	=	air-filled soil porosity (SIC default = 0.265 cm^3-air/cm^3-soil)
θ_w	=	water-filled soil porosity (SIC default = 0.216 cm^3-water/cm^3-soil)
θ_T	=	total soil porosity (SIC default = 0.481 cm^3-air/cm^3-soil)
H′	=	chemical-specific Henry's law coefficient (unitless)
Pb	=	dry soil bulk density (SIC default = 1.38 g/cm^3)
K_{oc}	=	chemical-specific soil organic carbon partition coefficient (cm^3/g)
f_{oc}	=	fraction organic carbon in soil (USEPA 2002 default = 0.002 g/g)

Default soil physical properties based on the assumption of silty clay (SIC) were used in the above equation. These default values are presented in the DTSC version of the J&E model spreadsheet for that soil type. This soil type was based on the soil borings logs from the Site.

Chemical-specific VF_s were used in the risk calculations for all VOCs detected in soil. The derivation of chemical-specific VF_s is presented in **Table T-10**. Using COPC soil concentrations and their respective chemical-specific VF_s, outdoor air concentrations (C_{oa}) were estimated using the following equation:

$$C_{oa} = \frac{C_s}{VF}$$

Where:

C_{oa} = COPC concentration in ambient air (mg/m^3)
C_s = COPC concentration in soil/sediment (mg/kg)
VF = chemical-specific volatilization factor (m^3/kg)

3.2.2.4 Vapor Emissions to Outdoor Air from Offsite Soil/Sediment

Volatile chemicals also have the potential of migrating from offsite areas adjacent to the Site. This pathway was also evaluated in this BHHRA for the recreator, rancher, and hypothetical offsite resident potentially exposed to offsite soil/sediment using the same screening level model described above for onsite VOC emissions, as a conservative approach. Derivation of the chemical-specific VFs for these receptors is presented in **Table T-10**.

3.2.2.5 Vapor Emissions to Outdoor Air from Onsite Surface Water

Volatile compounds were detected in surface water at the Site. Individuals could potentially be exposed to these COPCs emanating from surface water to outdoor air. Potential COPC emission from surface water to outdoor air was estimated using the USEPA Water9 Model. This model was used to calculate volatilization rates from surface water impoundments: A-Series Pond, Pond 13, and RCF Pond. These impoundments were modeled as rectangular lagoons with a detention period of 5 years. The impoundments were conservatively considered to be approximately 5 feet deep with the approximate aerial dimensions listed below:

Pond Name	Length (m)	Width (m)
A-Series Pond	313	104
Pond 13	69	69
RCF Pond	336	112

Note: m = meters

EPCs from each pond were used to determine COPC-specific emission from each pond (J_i) in grams per second. The emission rate per each COPC was used in the evaluation of potential risk from this pathway.

Surface water EPCs for the A-Series Pond, Pond 13, and RCF Pond are listed in **Table T-6** and were used as inputs into the Water9 Model to calculate emissions from these impoundments. To estimate the ambient air concentration in the air above each impoundment (C_{a-sw}), a box-model was used, along with the COPC-specific emission derived above, using the following equation:

$$C_{a-sw} = (J_i \times CF) / (U_{air} \times W \times H)$$

Where:

CF = conversion factor (1000 mg/g)
U_{air} = ambient air velocity in mixing zone (3.70 m/s, average Site-specific)
W = width of source-zone area (meters, pond-specific)

H = mixing zone height (2 meters)

The resulting emissions rates and ambient air concentrations ($C_{a\text{-}sw}$) above each pond are listed in **Table T-11**.

3.2.2.6 Vapor Emissions to Outdoor Air from Onsite Soil Vapor

Vapor emissions to outdoor air from onsite soil vapor were estimated following the approach described in the Standard Guide for Risk-Based Corrective Action (ASTM, 1995). This pathway was evaluated for a commercial/industrial worker in this BHHRA. The estimation of outdoor air vapors assumes contaminant diffusion through the vadose zone to the surface and dispersion in outdoor air. The commercial/industrial worker was assumed to be immediately down-wind of a 0.5 acre source area.

The soil gas to ambient air volatilization factor was estimated using the following equation:

$$VF_{amb} = \frac{1}{1 + \dfrac{U_{air} \cdot \delta_{air} \cdot L}{W \cdot D_{eff}}}$$

Where:

U_{air} = average wind speed (370 cm/s, average Site-specific)
δ_{air} = air mixing height (200 cm)
L = depth of soil vapor sample point (229 cm)
W = width of source parallel to the wind (4,500 cm)
D_{eff} = effective diffusion coefficient in soils (cm^2/s)

The effective diffusion coefficient in soils was estimated using this equation:

$$D_{eff} = D_{air}\frac{\theta_a^{3.33}}{\theta_T^2} + \frac{D_{water}}{H}\frac{\theta_w^{3.33}}{\theta_T^2}$$

Where:

D_{air} = diffusivity of chemical in air (cm^2/s)
D_{water} = diffusivity of chemical in water (cm^2/s)
H = Henry's Law coefficient (dimensionless)
θ_a = air filled porosity (0.265 dimensionless, silty-clay)
θ_w = water filled porosity (0.216 dimensionless, silty-clay)
θ_T = total porosity (0.481 dimensionless, silty-clay)

Chemical-specific properties were taken from the USEPA vapor intrusion guidance (USEPA, 2003). Default soil physical properties based on the assumption of silty clay (SIC) were used in the above equation. Chemical-specific VF_{amb} were used in the risk calculations for all VOCs detected in onsite soil vapor. Derivation of the chemical-specific soil gas to ambient air volatilization factors for a commercial/industrial worker is presented in **Table T-12**.

Using COPC soil vapor concentrations and their respective chemical-specific VF_{amb}, outdoor air concentrations (C_{oa}) from soil vapor were estimated using the following equation:

$$C_{oa} = SV \times VF_{amb}$$

Where:

C_{oa} = estimated outdoor air concentration of vapors (mg/m^3)
SV = measured soil vapor concentration (mg/m^3)
VF_{amb} = soil gas to ambient air volatilization factor (unitless)

3.2.2.7 Vapor Emissions into Indoor Air

The potential exists for VOCs to volatilize from the subsurface into indoor air. This pathway was evaluated using the Johnson and Ettinger (J&E, 1991 and Cal-EPA, 2005) subsurface vapor intrusion model to estimate potential migration of subsurface vapors into indoor air for a hypothetical offsite resident. The J&E computer spreadsheet model is public domain software that is freely available at the USEPA internet website. The model accounts for the diffusion of chemicals through the subsurface, the advection of chemicals through soil and concrete slabs due to pressure differentials between the soil and buildings, and the mixing in indoor air caused by heating and ventilation systems.

The J&E vapor intrusion model may be applied using soil matrix, soil vapor or groundwater concentration data. Soil vapor data are typically the preferred medium from which to evaluate the vapor intrusion pathway. For potential offsite indoor air exposures, offsite soil vapor data were used in this model to evaluate potential exposures to hypothetical offsite residents living near the Site. For potential onsite indoor air exposures, VOCs detected in soil from areas in close proximity to the Administration Building were used in the model to evaluate potential exposures to commercial/industrial workers at the Site.

For the soil vapor-to-indoor air pathway, maximum offsite soil vapor EPCs (**Table T-9a**) were used in the J&E model. Default soil physical properties based on the assumption of silty clay (SIC) were used in the model. These default values for SIC soil type are presented in the DTSC version of the J&E model spreadsheet. A default air exchange rate of 0.5 exchanges per hour was used in the model. A Qsoil value of 5 liters per minute (l/min) was used to represent the flow rate of chemicals from directly below the building into indoor air. Due to the uncertainty associated with vapor permeability rates directly beneath a building, the use of a Qsoil value in the range of 1 to 10 l/min has been recommended by USEPA with a default assumption of 5 l/min recommended. The default building dimensions for a residential scenario were used (1000 centimeters, cm x 1000 cm), with a proposed ceiling height of 8 feet (244 cm). The depth at which the maximum VOC concentration was detected in offsite soil vapor samples was used as the sampling depth in the model.

For the soil-to-indoor air pathway, maximum concentrations detected in soil samples from the Administration Building Area (**Table T-9b**) were used in the J&E model. Default soil physical properties based on the assumption of silty-clay (SIC) were used in the model. The silty-sand soil type was selected based on the shallow soil-types from Administration Building Area boring logs. These default values for silty-clay soil type are presented in the DTSC version of the J&E model spreadsheet. A default air exchange rate of 1 exchange per hour was used in the model. A Qsoil value of 5 liters per minute (l/min) was used. The default building dimensions for a commercial scenario were used (1000 centimeters, cm x 1000 cm), with a proposed ceiling height of 12 feet (366 cm). The depth at which the maximum VOC concentration was detected in onsite soil samples was used as the sampling depth in the model.

Predicted indoor air concentrations (C_{ia}) from the model using offsite soil vapor data (for offsite hypothetical residents) and onsite soil data from buildings in close proximity to the Administration Building (for onsite commercial/industrial workers) were used as EPCs in the estimation of potential risk and hazard. The J&E model spreadsheets including the model inputs, intermediate calculations, and predicted indoor air concentrations are presented in **Attachment T-1** for soil vapor and soil. The J&E model spreadsheets were used only to estimate indoor air concentrations and not to estimate potential risk for a hypothetical offsite resident and onsite commercial/industrial worker. The approach used to estimate potential risk for the vapor intrusion pathway is described below.

3.3 Estimating Chemical Intake

The exposure assessment quantifies the magnitude, frequency, and duration of chemical intake (daily intake) by receptor populations. Estimates of exposure or chemical intake are calculated based on assumptions regarding exposure pathways and exposure parameters. Chemical intake, or ADD or “Lifetime Average Daily Dose” (LADD) of COPCs, for each exposure pathway was estimated using guidelines in the Risk Assessment Guidance for Superfund (USEPA, 1989), Exposure Factors Handbook (USEPA, 1997a), Site-specific information, and professional judgment, as appropriate.

Daily intakes are estimated as being either ADDS or LADDs, depending on whether the chemical under consideration is a carcinogen or a noncarcinogen: LADDs are estimated for carcinogens and ADDs are estimated for noncarcinogens (USEPA, 1989). They differ primarily in the length of time over which the effects of the chemical are assumed to be averaged.

The LADD is averaged over a lifetime (70 years) for carcinogens, and the ADD is averaged over the expected exposure duration for noncarcinogens. The duration of exposure is assumed to vary depending on whether exposure occurs to a working population or residential population. LADDs and ADDs are estimated from the concentration of the chemical at the exposure point, the exposure frequency (i.e., number of times during a week or year), the exposure duration (i.e., the number of days, weeks, or years the exposure persists), and the physical characteristics of the receptor (such as body weight).

LADDs and ADDs under RME conditions are calculated by combining the upper-bound estimate of the concentration for each chemical (maximum or 95% UCL) with reasonable maximum exposure factors so that the result is the maximum exposure that is reasonably expected to occur (USEPA, 1989).

The ADD or LADD is estimated by multiplying an intake factor by the selected EPC (COPC concentration). The intake factor combines the Site-specific and receptor-specific assumptions for a given exposure pathway and is expressed as the amount of media (e.g., soil) taken into the body per unit concentration of chemical in the media, or mg/kg-day. Multiplying the intake factor by the selected EPC yields the ADD or LADD (mg/kg-day) for that receptor population and exposure pathway. The following is a generic equation used to estimate the daily dose:

$$\text{ADD/LADD (mg/kg-day)} = \text{Selected EPC x Summary Intake Factor}$$

Separate intake factors are estimated for each complete exposure pathway. The values and assumptions used to estimate each intake factor are dependent on the exposure pathway and receptor population being evaluated. A more detailed description of the values used for the intake calculations is presented below. The exposure assumptions used in this BHHRA are summarized in **Tables T-13** through **T-21**.

An important aspect of evaluating potential exposures to chemicals detected at the Site is that an individual cannot be present at each Study Area simultaneously. Nevertheless, a conservative assumption was made that hypothetical workers spend 100% of their time (e.g., 40 hours per week for 50 weeks out of the year for 25 years) within each Study Area. With this approach, intakes for exposures to onsite soils were not adjusted since workers are assumed to spend all their time in one area.

3.3.1 Incidental Soil/Sediment Ingestion

The rate of soil ingestion is based on the amount of soil/sediment an individual inadvertently swallows in a given day from all sources. Exposures to COPCs via incidental ingestion of soil/sediment were estimated using the following variables: (1) the rate of ingestion; and (2) the frequency and duration of exposure. Individuals may ingest soil/sediment through incidental contact of the mouth with hands and clothing. The following equation was used to estimate the potential daily dose (mg/kg-day) of COPCs from incidental ingestion of soil/sediment:

$$\text{ADD/LADD} = \frac{C_s \times IR_s \times ABS \times EF \times ED \times CF}{BW \times AT}$$

Where:

C_s = COPC concentration in soil/sediment (mg/kg)
IR_s = ingestion rate of soil (mg/day)
ABS = percent absorption (assumed to be 100 percent)
EF = exposure frequency (days/year)
ED = exposure duration (years)
CF = conversion factor for soil (10^{-6} kg/mg)
BW = body weight (kg)
AT = averaging time (days)
cancer effects: 70 years x 365 days = 25,550 days
noncancer effects: ED x 365 days

Details on the exposure parameters used to estimate intake of COPCs from incidental soil/sediment ingestion are provided in **Table T-13** for a commercial/industrial worker, **Tables T-15** and **T-17** for a trespasser, **Table T-18** for a recreator, **Table T-19** for a rancher, and **Table T-21** for a hypothetical offsite resident.

3.3.2 Dermal Contact with Soil/Sediment

Skin may come into contact with COPCs in soil/sediment, with subsequent absorbtion across the skin into the bloodstream. The amount of absorption into the body depends upon the amount of soil/sediment in contact with the skin, COPC concentrations in soil/sediment, the skin surface area exposed, and the potential for the chemical to be absorbed across the skin. To estimate the steady-state dose absorbed across the skin from dermal contact with soil/sediment, the following equation was used:

$$\text{ADD/LADD} = \frac{C_s \times SA \times SAF \times EF \times ED \times CF \times ABS_d}{BW \times AT}$$

Where:

- C_s = COPC concentration in soil/sediment (mg/kg)
- SA = skin surface area exposed to soil per day (cm^2/day)
- SAF = soil-skin adherence factor (mg/cm^2)
- EF = exposure frequency (days/year)
- ED = exposure duration (years)
- CF = conversion factor for soil (10^{-6} kg/mg)
- ABS_d = chemical-specific dermal absorption factor (unitless, see **Table T-22**)
- BW = body weight (kg)
- AT = averaging time (days)
  - cancer effects: 70 years x 365 days = 25,550 days
  - noncancer effects: ED x 365 days

Details on the exposure parameters used to estimate intake of COPCs from dermal contact with soil/sediment are provided in **Table T-13** for a commercial/industrial worker, **Tables T-15** and **T-17** for a trespasser, **Table T-18** for a recreator, **Table T-19** for a rancher, and **Table T-21** for a hypothetical offsite resident.

3.3.3 Inhalation of Vapors/Fugitive Dust from Soil/Sediment

The following inhalation exposure pathways were considered for soil/sediment in this BHHRA: 1) inhalation of outdoor vapors; and 2) inhalation of outdoor fugitive dust generated from wind erosion. The following equation was used to estimate the potential daily dose (mg/kg-day) from outdoor inhalation of vapors or fugitive dust:

$$\text{ADD/LADD} = \frac{C_{oa} \times IR_a \times ABS \times EF \times ED}{BW \times AT}$$

Where:

- C_{oa} = COPC concentration in ambient air (mg/m^3)
- IR_a = inhalation rate (m^3/day)
- ABS = percent absorption (assumed to be 100 percent)
- EF = exposure frequency (days/year)
- ED = exposure duration (years)

BW = body weight (kg)
AT = averaging time (days)
cancer effects: 70 years x 365 days = 25,550 days
noncancer effects: ED x 365 days

For the outdoor air pathway, COPC concentrations in soil or sediment were either divided by a particulate emission factor (PEF) for non-VOCs, or by a volatilization factor (VF) for VOCs, to arrive at an outdoor air concentration (C_{oa}) in units of mg/m^3. Use of the PEFs and VFs in the risk calculations was described earlier in this section.

The exposure parameters that were used to estimate intake of COPCs via inhalation are provided in **Table T-13** for a commercial/industrial worker, **Tables T-15** and **T-17** for a trespasser, **Table T-18** for a recreator, **Table T-19** for a rancher, and **Table T-21** for a hypothetical offsite resident.

3.3.4 Ingestion of Beef

As discussed in Section 3.1.4 there are privately held land(s) that currently adjoin the Site on the southwest border of the Site. These lands are being used for cattle-grazing, therefore cattle may potentially graze in the fields within Zone 2 (pastureland) along the southwest border of Zone 1. Field grass in this area may potentially take up COPCs from soil impacted by Zone 1, which may subsequently be eaten by grazing cattle. Cattle may also potentially ingest impacted soil present on and around grass. Therefore, consuming beef raised within the fields along the southwest border may transfer certain chemicals from the Site to the consumer.

Typically, local ranchers raise the cattle until they are sold to feed lots at 6-12 months of age. Therefore, the cattle are potentially exposed to chemicals in soil up to one year. Once at the feed lots, meat would enter the open market. Once transported to the feed lots, beef purchased on the open market is typically from multiple sources, not only from one source. In other words, most individuals consume beef raised in a location other than their immediate community; thus, consumption of beef purchased on the open market is not expected to contribute to total exposure in the general population. According to the Exposure Factors Handbook (Table 13-71 in USEPA, 1997a), 3.8% of beef eaten in the average household can be home-raised. However, a conservative assumption of 10% of beef originating from the Site was used in this BHHRA. For comparison, an assumption of 100% of beef originating from the Site was also used to estimate the potential daily dose of COPCs for ranchers from ingestion of beef with the results discussed in Section 6.0, Uncertainty Analysis.

Although chemicals taken up into plant tissue are available for ingestion by beef cattle and other herbivores (e.g., metals), most chemicals are generally present in plant tissues at concentrations often orders of magnitude less than the surrounding soil concentrations. Except for certain metals, chemicals do not tend to bioaccumulate in plant tissue. In addition, the amount of soil ingested by cattle while grazing, approximately 0.05% by weight of the pasture grasses ingested is small (Cal-EPA, 2003). Therefore, the uptake of chemicals while cattle are grazing is likely negligible.

Exposure via ingestion of beef was quantitatively evaluated pursuant to agency request. However, it is assumed that consumers are unlikely to receive a significant dose of COPCs from their cattle since COPCs may concentrate in the blood and bone and not in the tissues that individuals typically consume.

All of the equations presented below for this pathway were taken from the *Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments* (Cal-EPA, 2003). The following equation was used to estimate the potential daily dose of COPCs (mg/kg-day) for ranchers from ingestion of beef:

$$ADD/LADD = \frac{C_f \times IF \times GI \times L \times EF \times ED \times CF}{AT}$$

Where:

C_f	=	concentration in beef cattle (ug/kg, see equations below)
IF	=	beef ingestion (2.32 g/kg adult body weight-day)
GI	=	gastrointestinal absorption factor (1, unitless)
L	=	fraction of food type consumed from source (assume 10 percent and for a sensitivity analysis, 100% was also assumed and discussed in Section 6.0)
EF	=	exposure frequency (350 days/year)
ED	=	exposure duration (30 years)
CF	=	conversion factor for soil (10^{-6} from ug/kg to mg/g)
AT	=	averaging time (days) cancer effects: 70 years x 365 days = 25,550 days noncancer effects: ED x 365 days

In order to estimate human exposure to COPCs from consuming beef, the concentration of COPCs was estimated in edible beef (C_f) using the following equation:

$$C_f = [D_{inh} + D_{wi} + D_{feed} + D_{past} + D_{si}] \times T_{co}$$

Where:

D_{inh}	=	dose through inhalation (ug/day)
D_{wi}	=	dose through water ingestion (ug/day)
D_{feed}	=	dose through feed ingestion (ug/day)
D_{past}	=	dose through pasturing/grazing (ug/day)
D_{si}	=	dose through soil ingestion (ug/day)
T_{co}	=	transfer coefficient from ingested/inhaled media to meat/milk products (day/kg)

The dose via inhalation is proportional to the concentration of the COPC in air and the amount of air cattle breathe in one day. The following equation was used to estimate the potential dose (ug/day) via inhalation:

$$D_{inh} = BR \times GLC$$

Where:

BR	=	daily breathing rate for cattle (100 m^3/day)
GLC	=	ground level concentration (ug/ m^3)

$$GLC = \frac{C_s \times CF}{PEF \text{ or } VF}$$

Where:

C_s	=	concentration of COPC in soil/sediment (mg/kg)
CF	=	conversion factor (1,000 ug/mg)
PEF	=	particulate emission factor cubic meters per kilogram (m^3/kg) for non-VOCs
VF	=	volatilization factor (m^3/kg) for VOCs

Airborne COPCs depositing in surface water used as a source of drinking water for cattle can end up in the human food chain. For this assessment, it is assumed that the major source of water for the cattle is not adjacent to the Zone 1 boundary and therefore would not contribute to the dose to the cattle. Therefore, D_{wi} would be zero.

Cattle may graze and pasture in fields growing feed where airborne COPCs may have been deposited. For this assessment, it is assumed that the major source of feed for the cattle was not locally grown in impacted soil and therefore would not contribute to the dose to the cattle. Therefore, D_{feed} would be zero.

The equations below were used to estimate their potential dose (ug/day) from pasturing and grazing (Cal-EPA, 2003).

$$D_{past} = G \times FI \times C_p$$

Where:

G	=	fraction of diet provided by grazing (assume 100 percent)
FI	=	food ingestion rate for cattle (8 kg/day; Cal-EPA, 2003)
C_p	=	concentration in pasture (ug/kg)

The average concentration of COPCs in and on vegetation/pasture (C_p) is a function of direct deposition of the COPC onto the vegetation and of root translocation or uptake from impacted soil/sediment and was estimated using the following equation:

$$C_p = (C_{depv} \times GRAF) + C_{trans}$$

Where:

C_{depv}	=	concentration due to direct deposition (ug/kg)
GRAF	=	gastrointestinal relative absorption factor (0.43 for dioxins; 1 for all other COPCs, unitless)
C_{trans}	=	concentration due to root translocation or uptake (ug/kg)

The concentration of COPCs due to direct deposition (C_{depv}) was estimated using the following equation:

$$C_{depv} = [Dep \times IF / (k \times Y)] \times (1 - e^{-kT})]$$

Where:

Dep	=	deposition on affected vegetation per day (ug/m^2/day)
IF	=	interception fraction (0.1 for exposed crops)
k	=	weathering constant (10 d^{-1})
Y	=	yield (2 kg/m^2)
e	=	base of natural log (2.718)
T	=	growth period (90 days for exposed crops)

The deposition of COPCs on affected vegetation/pasture (Dep) was estimated using the following equation:

$$Dep = GLC \times Dep_{rate} \times 86{,}400$$

Where:

GLC	=	ground level concentration (ug/m^3)
Dep_{rate}	=	vertical rate of deposition (0.05 meters/second for uncontrolled sources)
86,400	=	seconds per day conversion factor (sec/day)

Finally, the concentration due to root translocation or uptake (C_{trans}) was estimated using the following equation:

$$C_{trans} = C_{s1} \times UF_2$$

Where:

C_{s1}	=	average soil concentration over the evaluation period (ug/kg)
UF_2	=	uptake factor based on soil/sediment concentration

For inorganic compounds, UF_2 values are listed in Cal-EPA guidance (2003). For organic compounds, UF_2 was estimated using the following equation:

$$UF_2 = [(0.03 \times K_{ow}^{0.77}) + 0.82] / [(K_{oc})(F_{oc})]$$

Where:

K_{ow}	=	octanol:water partition factor (chemical-specific)
K_{oc}	=	organic carbon partition coefficient (chemical-specific)
F_{oc}	=	fraction organic carbon in soil (0.002; USEPA, 2002 default)

The average concentration of COPCs in soil is a function of the deposition, accumulation period, chemical-specific soil half-life, mixing depth, and soil bulk density and was estimated using the following equation (Cal-EPA, 2003):

$$C_{s1} = (Dep \times X) / (K_s \times SD \times BD \times T_t)$$

Where:

X	=	integral function

K_s	=	soil elimination constant
SD	=	soil mixing depth (0.15 meters for agricultural setting)
BD	=	soil bulk density (1333 kg/m^3; Cal-EPA, 2003)
T_t	=	total days of exposure period (10,950 days = 30 years)

The integral function was estimated using the following equation (Cal-EPA, 2003):

$$X = [\{e^{-Ks \times Tf} - e^{-Ks \times To}\} / K_s] + T_t$$

Where:

K_s	=	soil elimination constant
T_f	=	end of evaluation period (10,950 days)
T_o	=	beginning of evaluation period (0 days)
T_t	=	total days of exposure period (10,950 days = 30 years)

And where:

$$K_s = 0.693 / t_{1/2}$$

Where:

0.693	=	natural log of 2
$t_{1/2}$	=	chemical-specific soil half-life (days; Cal-EPA, 2003)

The dose via incidental ingestion of soil is proportional to the concentration of the COPC in soil and the amount of soil cattle ingest in one day. The following equation was used to estimate the potential dose (ug/day) via incidental ingestion of soil (D_{si}):

$$D_{si} = SI_a \times C_p$$

Where:

SI_a	=	soil ingestion rate (kg/day)
C_p	=	concentration in pasture (ug/kg)

$$SI_a = [(1\text{-}G) \times FI \times FS_f] + [G \times FI \times FS_g]$$

Where:

G	=	fraction of diet provided by grazing (assume 100 percent)
FI	=	food ingestion rate (kg/day)
FS_f	=	soil ingested as a fraction of feed ingested
FS_g	=	soil ingested as a fraction of grazing ingestion

Meat products, such as edible beef, become contaminated when cattle inhale or ingest impacted materials (e.g., soil, feed) that are transferred to the edible meat. The chemical concentration in beef can be related to the total mass of the material ingested or inhaled per day. The transfer coefficient (T_{co}) represents the ratio of the chemical concentration in beef to the mass of the chemical consumed. Transfer coefficients would ideally be obtained from animal feeding studies; however, very few types of studies are available in the literature. Some information on metals can be obtained by extrapolating work done with radionuclides and other studies on dioxins and PCBs are available.

The transfer coefficient (T_{co}) represents the ratio of the chemical concentration in beef to the mass of the chemical consumed in a "particular media" (whether it be from inhalation, from ingestion of soil or feed, from pasturing and grazing, or from ingesting water). The T_{co} does not only apply for the transfer of chemical in feed to beef, but also from soil to beef, from water to beef, etc. Because of the lack of animal feeding studies in the literature to estimate T_{co} values, the T_{co} applies to all potentially impacted media that the cattle consumes. Therefore, in the equation for estimating COPC concentration in edible beef (C_f), an assumption is made that the feed-to-beef T_{co} is also applicable to soil ingestion, water ingestion, pasturing and grazing, and inhalation. A list of transfer coefficients used to estimate COPC concentrations in edible beef (C_f) is presented in Table 5.3 (page 5-14) in the *Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk* (Cal-EPA, 2003) and is also summarized below:

COPC	Toc
Dioxin	4.0E-01
Barium	2.0E-04
Beryllium	1.0E-03
Cadmium	5.5E-04
Chromium	9.2E-03
Cobalt	1.0E-04
Copper	9.0E-03
Lead	4.0E-04
Manganese	5.0E-04
Molybdenum	1.0E-03
Nickel	2.0E-03
Thallium	4.0E-02
Tin	1.0E-02
Vanadium	2.5E-03
Zinc	1.0E-01

Details on the exposure parameters used to calculate intake of COPCs for a rancher from ingestion of beef are provided in **Table T-20**.

3.3.5 Incidental Ingestion of Surface Water

Commercial/industrial workers and trespassers may come into contact with COPCs via incidental ingestion of surface water. Exposures to surface water COPCs are estimated using the following variables: (1) the rate of ingestion and (2) the frequency and duration of exposure. The rate of ingestion is based on the amount of water an individual inadvertently swallows in a given day.

An incidental water ingestion rate of 0.02 liters/day was conservatively used in this BHHRA for a commercial/industrial worker while 0.1 liters/day was used for a trespasser. These rates were recommended by USEPA Region IV for incidental ingestion exposures (USEPA Region IV, 2007). At this particular Site, individuals are not expected to swim in surface water because it will not be allowed onsite and the offsite water depth is shallow (this depth will vary seasonally and with precipitation); however, they may splash around while working/playing with the impacted water. The ingestion rates of 0.02 and 0.1 liters/day were considered sufficiently conservative to evaluate exposure to COPCs in surface water at this Site.

The following equation is used to estimate the potential dose (mg/kg-day) from incidental ingestion of surface water:

$$\text{ADD/LADD} = \frac{C_{sw} \times IR_{sw} \times ABS \times EF \times ED}{BW \times AT}$$

Where:

C_{sw} = chemical concentration in surface water (mg/L)
IR_{sw} = incidental ingestion rate of water (L/day)
ABS = percent absorption (assumed to be 100 percent)
EF = exposure frequency (days/year)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (days)
cancer effects: 70 years x 365 days = 25,550 days
noncancer effects: ED x 365 days

The exposure parameters that were used to estimate intake of COPCs via incidental ingestion of surface water are provided in **Table T-14** for a commercial/industrial worker and **Table T-16** for a trespasser.

3.3.6 Dermal Contact with Surface Water

Commercial/industrial workers and trespassers may come into contact with COPCs via dermal contact with surface water. Their skin may come into contact with surface water COPCs, with subsequent absorption across the skin into the bloodstream. The amount of absorption into the body depends upon the amount of surface water in contact with the skin, COPC concentrations in surface water, the skin surface area exposed, and the potential for the chemical to be absorbed across the skin.

Surface area is a measure of the area of skin potentially exposed to contaminated media. The surface area used depends upon the exposure scenario and activity evaluated. The USEPA (2004) default of 3,300 cm^2 is used for workers, while it is conservatively assumed children ages 11 to 17 may trespass on the Site. For children, an age weighting approach is used to reflect the actual skin surface area exposed. The skin surface area selected for a child is 4,700 cm^2 assuming exposure to head, arms, hands and legs (USEPA, 1997a).

The estimated exposure from dermal contact with water is actually an absorbed dose and not the amount of chemical that comes into contact with the skin (i.e., intake). The amount of dose absorbed across the skin is determined using chemical-specific dermal permeability constants (Kp), expressed in units of centimeters per hour. Dermal permeability constants reflect the rate of movement at which a chemical crosses the skin to the stratum corneum (outermost skin layer that provides resistance to absorption) and into the bloodstream, and are based on an equilibrium partitioning. USEPA (2004) provides equations for calculating dermal permeability constants. USEPA recommends using estimated values for Kp rather than measured values, because replicated experiments gave measured values that varied up to two orders of magnitude. A small number of constants will be derived based on measured values if estimated values are unavailable. These Kp values are estimated using the following formula: log k_p = -2.72 + 0.71 log k_{ow} - 0.0061 * molecular weight (see Section 5.2.3, equation 5.8 in USEPA, 1992a). For inorganics without Kp values, a default value of 10^{-3} cm/hr is used as recommended by USEPA (2004).

The length of time skin is exposed to water determines the amount of chemical absorbed through the skin from impacted water. An assumption is made that workers could potentially be exposed to surface water for one hour per work week, 50 days per year for 25 years, while children ages 11 to 17 years old could potentially be exposed for one hour per day, once a month, and 12 days per year for 7 years.

The following equation is used to estimate the steady-state dose absorbed across the skin from dermal contact with surface water:

$$\mathrm{ADD/LADD} = \frac{C_{sw} \times SA \times K_p \times EF \times ED \times ET \times CF}{BW \times AT}$$

Where:

C_{sw}	=	chemical concentration in surface water (mg/L)
SA	=	surface area of exposed skin (cm^2)
K_p	=	permeability constant (cm/hr, see **Table T-22**)
EF	=	exposure frequency (days/year)
ED	=	exposure duration (years)
ET	=	exposure time (hours/day)
CF	=	conversion factor (10^{-3} L/cm^3)
BW	=	body weight (kg)
AT	=	averaging time (days) cancer effects: 70 years x 365 days = 25,550 days noncancer effects: ED x 365 days

The exposure parameters that were used to estimate COPC intake via dermal contact with surface water are provided in **Tables T-14** and **T-16** for a commercial/industrial worker and trespasser, respectively. The chemical-specific water permeability constants, K_p, are presented in **Table T-22**.

3.3.7 Inhalation of Vapors from Onsite Surface Water

The following equation was used to estimate the potential daily dose (mg/kg-day) for a commercial/industrial worker from outdoor inhalation of surface water vapors:

$$\mathrm{ADD/LADD} = \frac{C_{a\text{-}sw} \times IR_a \times ABS \times EF \times ED}{BW \times AT}$$

Where:

$C_{a\text{-}sw}$ = outdoor air COPC concentration from surface water (mg/m^3, **Section 3.2.2.5**)
IR_a = inhalation rate (m^3/day)
ABS = percent absorption (assumed to be 100 percent)
EF = exposure frequency (days/year)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (days)
cancer effects: 70 years x 365 days = 25,550 days
noncancer effects: ED x 365 days

The $C_{a\text{-}sw}$ was estimated using the USEPA Model Water9 as described above in **Section 8.3.2.4** of the RI Report. The exposure parameters that were used to estimate intake of COPCs for a commercial/industrial worker via outdoor inhalation of surface water vapors are provided in **Table T-14**.

3.3.8 Inhalation of Outdoor and Indoor Vapors

Inhalation of outdoor air vapors is a consideration for soil vapor exposures for a commercial/industrial worker and a hypothetical offsite resident. Inhalation of indoor air vapors is a consideration for soil vapor exposures for a hypothetical offsite resident. Additionally, inhalation of indoor air vapors is a consideration for soil matrix exposures for a commercial/industrial worker within the area of the Administration building. For these pathways, model-predicted indoor air concentrations (C_{ia}) from soil vapor or soil matrix, or outdoor air concentrations (C_{oa}) from soil vapor were used in the following intake equation:

$$\text{ADD/LADD} = \frac{(C_{ia} \text{ or } C_{oa}) \times IR_a \times EF \times ED}{BW \times AT}$$

Where:

C_{ia} = indoor air COPC concentration from soil vapor or soil matrix (mg/m^3, **Section 3.2.2.7**)
C_{oa} = outdoor air COPC concentration from soil vapor (mg/m^3, **Section 3.2.2.6**)
IR_a = inhalation rate (m^3/day)
EF = exposure frequency (days/year)
ED = exposure duration (years)
BW = body weight (kg)
AT = averaging time (days)
cancer effects: 70 years x 365 days = 25,550 days
noncancer effects: ED x 365 days

The exposure parameters that were used to estimate COPC intake for a commercial/industrial worker and a hypothetical offsite resident via inhalation from soil vapor or soil matrix are provided in **Tables T-13** and **T-21**, respectively.

4.0 TOXICITY ASSESSMENT

The toxicity assessment characterizes the relationship between the magnitude of exposure to a COPC and the nature and magnitude of adverse health effects that may result from such exposure. For purposes of calculating exposure criteria to be used in risk assessments, adverse health effects are classified into two broad categories: noncarcinogens and carcinogens. Toxicity criteria are generally developed based on the threshold approach for noncancer effects and the non-threshold approach for cancer effects.

For carcinogens, it is assumed that there is no level of exposure that does not have a finite possibility of causing cancer (i.e., there is no threshold dose for cancer effects). That is, a single exposure of a carcinogen may, at any level, result in an increased probability of developing cancer. For chemicals exhibiting noncancer effects, it is believed that organisms have protective mechanisms that must be overcome before the toxic endpoint results (i.e., there is a threshold dose for these effects). For example, if a large number of cells perform the same or similar functions, it would be necessary for significant damage or depletion of these cells to occur before a toxic effect could be seen. As a result, a range of exposures exists from zero to some finite value that can be tolerated by the organism with essentially no chance of expression of adverse effects (USEPA, 1989). Some chemicals may elicit both cancer and noncancer effects.

In this BHHRA, chronic toxicity criteria were selected (in order of preference) from the following sources: 1) Cal-EPA OEHHA Toxicity Criteria Database, online (2007a); 2) USEPA's (2007) Integrated Risk Information System (IRIS) as referenced in USEPA Region IX Preliminary Remedial Goals (PRG) table (USEPA, 2004a); 3) USEPA (1997b) Health Effects Assessment Summary Tables (HEAST), as referenced in the Region IX PRG table (USEPA, 2004a); or 4) USEPA NCEA Superfund Health Risk Technical Support Center, as referenced in the USEPA PRG table (2004a).

4.1 Toxicity Criteria for Potential Carcinogens

Potential cancer effects resulting from human exposure to chemicals are generally estimated quantitatively using oral cancer slope factors (CSFs) or inhalation unit risk factors (URFs). Oral CSFs are expressed in units of $(mg/kg\text{-}day)^{-1}$. To characterize potential cancer risks from inhalation, URFs were converted when needed from units of $(\mu g/m^3)^{-1}$ to units of $(mg/kg\text{-}day)^{-1}$ by assuming an individual inhales at a rate of 20 cubic meters per day, and has an average body weight of 70 kg and absorption is equivalent by either route (USEPA, 1989).

Oral and inhalation CSFs are derived by Cal-EPA and USEPA from the results of chronic animal bioassays, human epidemiological studies, or both. Animal bioassays are usually conducted at dose levels that are much higher than those likely to be produced by human exposure to environmental media. These high dose levels are used to detect possible adverse effects in the relatively small test populations used in the studies.

Because humans are generally exposed at lower doses, the data are extrapolated using mathematical models. Most commonly, the linearized multistage model is used to estimate the largest possible linear slope (95UCL) at low extrapolated doses that is consistent with the data. The 95UCL slope of the dose-response curve is subjected to various adjustments, and an interspecies scaling factor is usually applied to derive a CSF for humans. Dose-response data derived from human epidemiological studies are fitted to dose-time-response curves on an ad hoc basis.

Conservative (i.e., health protective) assumptions are generally applied, and the models are believed to provide rough estimates of the upper limits on potential carcinogenic potency. The actual risks associated with exposure to a potential carcinogen and quantitatively evaluated on the basis of its CSF are not likely to exceed the risks estimated and may be much lower or even zero.

CSFs that are available for the COPCs that are classified as carcinogens were presented in the toxicity criteria table. When available, Cal-EPA CSFs were also identified. At the present time, Cal-EPA and USEPA have only developed CSFs for the oral and inhalation routes of exposure. In the absence of values specific to the dermal route, the oral factors were used for the dermal toxicity factors.

The CSFs used in this BHHRA are presented in **Table T-22**.

4.2 Toxicity Criteria for Potential Noncarcinogens

Potential noncancer effects resulting from human exposure to chemicals are estimated quantitatively using chronic reference doses (RfDs) for ingested chemicals and reference concentrations (RfCs) for inhaled chemicals. As was the case for the CSFs, RfDs and RfCs are only available for oral and inhalation exposures. In the absence of criteria specific to the dermal exposure pathway, the oral RfDs were used to evaluate the dermal route of exposure.

These toxicity values are developed by the USEPA RfD/RfC workgroup on the basis of a wide array of noncancer health effects. The RfD, expressed in units of milligrams of chemical intake per kilogram of body weight per day (mg/kg-day), is an estimate of the maximum human exposure level that can be present without an appreciable risk of deleterious effects during a designated time. The RfC is expressed in units of milligrams of chemical per cubic meter of air (mg/m^3) and is an estimate of the maximum air concentration that can be present without an appreciable risk of deleterious effects. RfCs assume a human body weight of 70 kilograms and an inhalation rate of 20 m^3/day.

RfDs and RfCs are usually derived from either human studies involving workplace exposures or from animal studies, and are adjusted using generic uncertainty factors. The RfD and RfC provide benchmarks against which human intakes of chemicals resulting from exposure to impacted environmental media are compared. Chronic Reference Exposure Levels (RELs) for inhalation exposure have been developed by Cal-EPA for the Air Toxics Hot Spots program, which were used if they were available and more conservative than the USEPA RfCs.

The RfDs used in this BHHRA are presented in **Table T-22**.

4.3 Dermal Toxicity Criteria

As indicated previously, USEPA has developed CSFs and RfDs only for inhalation and ingestion (intake) exposures. There are no available toxicity values for evaluating dermal (uptake) exposures. In the absence of dermal criteria, oral CSFs and RfDs were used to evaluate dermal exposures.

4.4 Health Effects from Lead

The traditional RfD approach to the evaluation of chemicals is not applied to lead because most human health effects data are based on blood lead concentrations, rather than external dose (Cal-EPA, 1992). The Centers for Disease Control has identified the Lowest Observed Affect Level for lead of 10 micrograms of lead per deciliter (μg/dl) and 30 μg/dl for children and adults, respectively. Consistent with the Centers for Disease Control, Cal-EPA DTSC previously considered exceedances over 10 μg/dl of whole blood (μg/dl) as levels that could indicate potential adverse effects. However, more recently, the Cal-EPA OEHHA has developed a 1 μg/dL benchmark for source-specific incremental change in blood lead levels for protection of school children and fetuses (OEHHA, 2007). This value is now being used by Cal-EPA for evaluating potential lead exposures.

The health effects of lead were evaluated by using the DTSC LeadSpread version 7.0 model to predict 95th as well as 99th percentile blood lead levels. The LeadSpread model spreadsheets are presented at the end of **Attachment T-1**.

5.0 RISK CHARACTERIZATION

Risk characterization integrates the results of the toxicity assessment (Section 4.0) and the exposure assessment (Section 3.0) to estimate potential cancer risks and adverse noncancer health effects associated with exposure to chemicals detected at the Site. This integration provides quantitative estimates of potential risk and noncancer hazard that are then compared to acceptable standards.

The process of risk assessment is an iterative process where Site, receptor, and chemical-specific data are used when available. When Site-specific data are not available, conservative (i.e., health protective) assumptions are utilized. The use of repeated, conservative assumptions can lead to overly conservative estimations of risk but certainly provides an upper-bound estimate of the actual risk. Thus, for any site, the estimated risk level reflects an upper-bound estimate of the most probable risk. The most probable risk is likely to be much less, perhaps as low as zero, and probably not measurable in the potentially exposed population.

5.1 Introduction to Risk Characterization

Various demarcations of acceptable risk have been established by regulatory agencies. For example, the USEPA has established an acceptable risk range for Superfund sites. The National Contingency Plan (NCP; 40 CFR 300) indicates that lifetime incremental cancer risks posed by a site should not exceed a range of one in one million (1×10^{-6}) to one hundred in one million (1×10^{-4}) and noncarcinogenic chemicals should not be present at levels expected to cause adverse health effects (i.e., a hazard index [HI] greater than 1). Other relevant guidance (USEPA, 1991b) additionally states that sites posing a cumulative cancer risk of less than 10^{-4} and hazard indices less than unity (1) for noncancer endpoints are generally not considered to pose a significant risk warranting remediation. The California Hazardous Substances Account Act (HSAA) incorporates the NCP by reference, and thus also incorporates the acceptable risk range set forth in the NCP. The Resource Conservation and Recovery Act (RCRA) Corrective Action program incorporates this same range of potential health risks as the “acceptable risk range” for determining whether corrective action is warranted at RCRA facilities and for closure purposes. Finally, The Safe Drinking Water and Toxic Enforcement Act of 1986 (California Proposition 65) regulates chemical exposures to the general population and is based on an acceptable risk level of $1x10^{-5}$.

The maximum acceptable risk level for a site is between 10^{-4} and 10^{-6}, and is selected on a case-by-case basis by USEPA. The risk range between 10^{-4} and 10^{-6} is commonly called the “discretionary risk range”. These ranges of acceptable risk are in contrast to the background risk of Americans in the general population developing cancer. The background risk is approximately one chance in three (0.33 or $3.3x10^{-1}$) for every American of eventually developing some form of cancer (ACS, 2006).

For the purposes of this section, a cumulative risk of 1×10^{-5} and noncancer hazard index of 1 is used to compare industrial/commercial worker risk estimates. For all other potential exposures a risk level of 1×10^{-6} and noncancer hazard index of 1 is used. These risk levels are used to provide context to the risk results and to support the following discussion which focuses on those pathways and chemicals that contribute the majority to the risk estimates. It is acknowledged that additional risk management considerations such as technical feasibility, economic, social, political, and legal factors may be part of the final risk management decision.

The results of the risk characterization are really the starting point for risk management considerations for a site (USEPA, 1995).

5.2 Risk Characterization for Potential Cancer Effects

Excess cancer risks are expressed as the upper-bound, increased likelihood of an individual developing cancer as a result of exposure to a particular chemical. For example, a cancer risk of 1 x 10^{-4} refers to an upper-bound increased chance of one in ten thousand of developing cancer over a lifetime.

In the risk characterization step of the BHHRA, excess cancer risk was estimated by multiplying the LADD by the chemical-specific cancer slope factor (CSF). The following equation was used to estimate the excess cancer risk per each COPC:

$$\text{Excess Cancer Risk} = \text{LADD x CSF}$$

The chemical-specific excess cancer risks were then summed to yield a cumulative cancer risk, which is typically compared to the USEPA acceptable risk range of 10^{-6} to 10^{-4}.

5.3 Risk Characterization for Potential Noncancer Effects

The potential for noncancer effects due to exposure to a particular chemical is expressed as the hazard quotient (HQ). Chemical-specific hazard quotients were estimated by calculating the ratio of the ADD to the corresponding chronic reference dose (RfD) for noncancer effects. The following equation was used to estimate the noncancer hazard quotient:

$$\text{Hazard Quotient} = \frac{\text{ADD}}{\text{RfD}}$$

The chemical-specific hazard quotients were then summed to form a cumulative hazard index (HI), which was compared to an acceptable hazard level of one (1). For multiple chemical exposures, the total HI might exceed 1 even if no single chemical intake exceeds its RfD. If the cumulative HI is less than the benchmark level of one (1), cumulative exposures to the COPCs at the Site are judged unlikely to result in adverse noncancer health effects. If the sum is greater than 1, a more detailed and critical evaluation of potential noncancer health hazards may be warranted. Such additional evaluation considers the specific target organ(s) affected and mechanism(s) of action of the COPCs.

5.4 Results of the Risk Characterization

The chemical-specific potential cancer risk and hazard index estimates are presented in **Tables T-23** to **T-28** for each of the receptor groups, media, and exposure pathways discussed above. The J&E vapor intrusion model spreadsheets are presented in **Attachment T-1** for the commercial/industrial worker and hypothetical offsite residential exposure scenarios. The LeadSpread worksheets which evaluate lead in soil are presented at the end of **Attachment T-1**. The detailed risk calculations for the direct contact, outdoor and indoor inhalation pathways are presented in **Attachment T-2**. The table on the following page summarizes the receptor groups, exposure medium and exposure pathways quantitatively evaluated in this BHHRA.

For potential exposures to onsite soils and sediments via direct contact (ingestion and dermal contact) and outdoor inhalation, only the FPP and Liquid Treatment Study Areas exhibited elevated risk for commercial/industrial worker exposures with a cumulative risk of 5 x 10^{-5} and a noncancer HI of 2, respectively. PCE in shallow soil was the primary risk driver for the FPP Study Area and MCPP was the primary risk driver for both surface and shallow soils at the Liquid Treatment Study Area. In addition, risk estimates for trespasser exposures to FPP soils were slightly elevated (2 x 10^{-6}) due to the presence of PCE in subsurface soils. The sample locations that contributed the majority to these risk estimates were RISBON-37, RISBON-41 and RISBON-63 in the FPP Study Area just south of the PSCT and RISBLT-02 in the Liquid Treatment Study Area.

For onsite surface water, Ponds A-Series, 13 and RCF cancer risk estimates were elevated for commercial/industrial worker exposures (maximum cumulative risk of 8 x 10^{-5}) and trespassers (maximum cumulative risk of 3 x 10^{-6}) with arsenic being the primary risk driver. All noncancer HIs were below 1.

For offsite soils/sediments cancer risk and noncancer hazard estimates for recreational and rancher exposures were below a cancer risk level of 1 x 10^{-6} and a noncancer hazard of 1.

For the hypothetical offsite resident, the Burial Trench, Central Drainage, and FPP Study Areas exhibited elevated risk due to exposures from the transport of onsite vapors to offsite locations with a maximum cumulative risk estimate for the Burial Trench Study Area of 1 x 10^{-5}. The primary risk drivers were tetrachloroethene and trichloroethene. The sample locations that contributed the majority to these risk estimates were RISBON-37, RISBON-41 and RISBON-63 in the FPP Study Area just south of the PSCT, RISBCD-07 in the Central Drainage Study Area and RISSBC-05 in the Burial Trench Study Area. It should be noted that the hypothetical offsite resident evaluation is overly conservative in that the modeling assumes the resident is located adjacent to the Study Area being evaluated. In reality, the resident would be located some distance from the Study Area boundary thereby resulting in lower estimates of exposure.

For other hypothetical offsite residential exposures, only the vapor intrusion pathway resulted in a marginally elevated risk estimate with a cumulative risk estimate of 2 x 10^{-6}. The primary risk driver for this pathway was 1,3-butadiene. When considering more recent soil vapor sampling, this risk estimate would be even lower and similar to the target risk level of 1 x 10^{-6}.

Adverse health effects associated with exposure to lead have been correlated with concentrations of lead in whole blood and not with intake of lead by an individual. Because lead was selected as a COPC, the health effects of lead were evaluated by using the DTSC LeadSpread version 7.0 model to predict the percentile blood lead level for adults and children with an age range of 11 to 17 years old. Because this model provides blood lead predictions for adults and 1-2 year old children, the default exposure parameters (e.g., skin surface area, soil adherence factor, soil ingestion rate, etc.) were revised to reflect age-specific exposure assumptions for 11 to 17 year-old children.

Receptor Population	Exposure Medium	Corresponding Table
Commercial/Industrial Worker	Onsite Soil SS = 0-6 inches bgs SB = 0-5 feet bgs	Table T-23
	Onsite Sediment SS = 0-6 inches bgs SB = 0-5 feet bgs	Table T-25
	Onsite Surface Water	Table T-26
	Onsite Soil Vapor	Table T-27
	Administration Building Soil	Table T-28
Trespasser	Onsite Soil SS = 0-6 inches bgs SB = 0-5 feet bgs	Table T-23
	Onsite Sediment SS = 0-6 inches bgs SB = 0-5 feet bgs	Table T-25
	Onsite Surface Water	Table T-26
Recreator	Offsite Soil SS = 0-6 inches bgs SB = 0-5 feet bgs	Table T-24
	Offsite Sediment SS = 0-6 inches bgs	
Rancher	Offsite Soil SS = 0-6 inches bgs SB = 0-5 feet bgs	Table T-24
	Offsite Sediment SS = 0-6 inches bgs	
	Onsite Roadway Soil SS = 0-6 inches bgs SB = 0-5 feet bgs	Table T-23
Hypothetical Offsite Resident	Offsite Soil SS = 0-6 inches bgs SB = 0-5 feet bgs	Table T-29
	Offsite Sediment SS = 0-6 inches bgs	
	Offsite Soil Vapor	
	Onsite Soil SS = 0-6 inches bgs SB = 0-5 feet bgs	

Notes:
SS = refers to surface soil; SB = refers to shallow soil

The DTSC previously considered exceedances over 10 μg/dl of whole blood as levels that could indicate potential adverse effects consistent with the Centers for Disease Control Lowest Observed Affect Level for lead of 10 μg/dl and 30 μg/dl for children and adults, respectively. More recently, the Cal-EPA OEHHA has developed a 1 µg/dL benchmark for source-specific incremental change in blood lead levels for protection of school children and fetuses (Cal_EPA, 2007b). This value is now being used by Cal-EPA for evaluating potential lead exposures. Based on the revised benchmark lead level of 1 µg/dL OEHHA derived California Human Health Screening Levels (CHHSLs) of 80 mg/kg and 320 mg/kg for residential and occupational exposures, respectively (Cal-EPA, 2009).

Lead EPCs in onsite soils for the Study Areas ranged in concentrations from 9.8 mg/kg to 498 mg/kg with a Sitewide 95UCL of 17.3 mg/kg. The highest lead EPC (498 mg/kg) was from surface soil at the Maintenance Shed Area, with the next highest lead EPC (295 mg/kg) from the same Study Area but in shallow soil. All other lead EPCs were less than or equal to 61 mg/kg. The results of the lead evaluation indicated that no predicted blood lead levels (neither 95^{th} nor 99^{th} percentiles) exceeded the previous DTSC threshold of 10 μg/dl. In addition, all Study Area lead EPCs, with the exception of the Maintenance Shed Area, as well as the Sitewide lead EPC were below the most conservative lead CHSSL of 80 mg/kg. For the Maintenance Shed Area, the surface soil EPC was above the industrial CHHSL of 320 mg/kg; however the EPC is being driven by one sample, RISBMS-11 in which the lead concentration is 970 mg/kg. The next highest concentration is 160 mg/kg well below the occupational CHHSL. The LeadSpread worksheets are presented at the end of **Attachment T-1**.

6.0 UNCERTAINTY ANALYSIS

The methodology used in the BHHRA is consistent with USEPA and Cal-EPA risk assessment guidance. However, the procedures used in any quantitative RA are conditional estimates given the many assumptions that must be made about exposure and toxicity. Major sources of uncertainty in risk assessment include (1) natural variability (e.g., differences in body weight or sensitivity in a group of people); (2) incomplete knowledge of basic physical, chemical and biological processes (e.g., the affinity of a chemical for soil, degradation rates); (3) model assumptions used to estimate key inputs (e.g., exposure, dose response models, fate and transport models); and (4) measurement error primarily with respect to sampling and laboratory analysis.

Site-specific factors, which this BHHRA incorporated, decrease uncertainty, although uncertainty may persist in even the most site-specific RAs due to the inherent uncertainty in the process. However, because the assumptions used tend to be health-protective and conservative in nature, the estimated risks are likely to exceed the most probable risk posed to potential receptors at the Site and actual risks would be much lower.

Some of the most significant elements affecting uncertainty for this BHHRA include:

- It was assumed that chemical concentrations remain constant over the duration of exposure. No abiotic or biotic degradation mechanisms, which reduce the concentrations of COPCs over time, are assumed to occur. This general assumption of steady-state conditions also applies to sources and chemical release mechanisms and may result in a conservative estimation of long-term exposure concentrations.
- The exposure assumptions used for the RME approach are considered conservative and likely lead to overstating the most probable estimate of potential risk. For example, the RME exposure scenario assumes a hypothetical offsite resident will remain at the same location from birth through age 30 years for 350 days per year, or a commercial worker will work at the Site for 25 years.
- Intake parameters for the various exposure pathways (soil ingestion, dermal contact, inhalation) were conservatively assumed to be upper bound estimates (e.g., 3300 cm2 of exposed skin exposed every day–regardless of the weather conditions–or ingestion of 100 mg of soil each day over the exposure period for adults, etc.) for the RME approach.
- For exposures via outdoor air inhalation, the outdoor air flux model assumes that the VOC is present at the surface and that the receptors will come into contact via outdoor air inhalation. When chemicals are present at depths below 6 inches, the flux would be lower resulting in lower estimates of potential risk.
- The risk assessment focused on soil from surface to approximately 5 feet bgs as this is considered the most likely depth interval that may be contacted. If significant concentrations were present at depths below that interval and the soil was brought to the surface then exposures may have been underestimated. To evaluate this issue, the data from greater than 5 feet to 10 feet bgs were reviewed. There was one area where deeper concentrations were significantly higher in RISBON-37. However, this location has already been identified in the risk assessment as posing a potential health risk and will likely be targeted for remediation.
- Soil samples were collected as part of the Phase III RI where step-out borings were completed in the RISBON-59 area (located along NTU road, south west of the west end of RCF pond). However data from this round of sampling were not included in the risk

evaluation. The Phase III data relevant for exposure from 0 to 5 feet bgs (samples collected at 0.5 feet bgs and 6 feet bgs) were compared to metals background UTLs and/or human health screening levels. This screening indicated marginal potential risks from the N-nitroso compounds in two samples. These samples represent a small potential exposure given that they represent a localized area of primarily subsurface impacts and Site-workers would not be in the area on a frequent basis. While there is some uncertainty in not including these samples, due to the localized nature of impact and infrequent exposure potential, the results and conclusions reached in this BHHRA are not significantly impacted.

- This BHHRA assumed that the PCB Landfill has been capped as discussed in the conceptual site model earlier in the report. The PCB Landfill has an interim soil (claystone) cover of unknown thickness placed in the 1980's with the northern part of the landfill currently used as a temporary storage area for the CSC's investigation-derived waste. According to existing information (RCRA Part B Permit Application, Modernization Plan Final EIR), the interim cover soil generally came from the area in which the landfill was constructed and was placed at a minimum 1-foot thickness. The presence of a 1-foot minimum thickness of cover does provide a barrier for human contact. In addition, due to the nature of the area being a landfill, worker exposure would not be expected due to intrusive activities beneath the cover. As a result potential human health risk to PCB Landfill contents is considered insignificant.
- Revising the assumption that ranchers consume only 10% of beef from their own lots to assuming that they consume 100% of their own beef (Section 3.3.4) resulted in risk and hazard estimates that were also significantly less than the target risk levels of 1×10^{-6} and 1, respectively. Assuming 10%, the risk and hazard were 1×10^{-10} and 2×10^{-7}, respectively, whereas, assuming 100%, the risk and hazard were 2×10^{-9} and 3×10^{-6}, respectively.
- Default soil physical properties were used for the soil type, silty clay (SIC). Lack of Site-specific values may introduce some uncertainty into the vapor modeling and may result in an over-prediction or under-prediction of vapor inhalation exposures from beneath the surface.
- A hypothetical resident was evaluated assuming locations near the Site (offsite) would be developed as residential land use. This land use is highly unlikely given the nature of the Site and the planned use of institutional controls such as deed restriction to preclude this type of land use.
- For modeling of onsite impacts to offsite locations via the windblown particulate and vapor pathways, it was assumed that the hypothetical resident was located adjacent to the Study Area being evaluated. This is a conservative assumption as the actual offsite location would likely be much farther from the Study Area boundary resulting in decreased exposures.

7.0 SUMMARY AND CONCLUSIONS

This BHHRA was prepared to evaluate potential baseline health risks associated with chemicals present in soil, sediment, soil vapor, and surface water at the Site. The results of the BHHRA can be used to identify chemicals and exposure media that may pose an unacceptable risk to current and/or future receptors at the Site and to provide information for remedial planning. This risk assessment was prepared as part of the RI to evaluate potential exposures and “define risks to public health and the environment” related to soil, sediment, soil vapor, and surface water, and to subsequently provide information for the FS.

The chemicals of potential concern evaluated include inorganics, PCBs, dioxins, herbicides/pesticides, PAHs, SVOCs, and VOCs. Potential exposure scenarios that were considered include inhalation of indoor air and outdoor air vapors, inhalation of particulates, dermal contact with surface water, and exposure via direct contact to soils and sediment.

The results of the BHHRA indicate that the following COPCs are primary risk drivers: MCPP, tetrachloroethene, trichloroethene in onsite soils and arsenic in onsite surface water.

For onsite soils, only the FPP and Liquid Treatment Study Areas exhibited elevated risk estimates for commercial/industrial worker exposures. PCE in shallow soil was the primary risk driver for the FPP Study Area and MCPP was the primary risk driver for both surface and shallow soils at the Liquid Treatment Study Area. Both of these chemicals are present at elevated concentrations in localized areas within the Study Areas as shown in the figures for these chemicals presented in **Section 5** of the RI Report.

For the hypothetical offsite resident, only the Burial Trench, Central Drainage, and FPP Study Areas exhibited elevated risk estimates from potential exposures from the transport of onsite soil contamination via windborne vapors. The primary risk drivers were tetrachloroethene and trichloroethene which are both present at elevated concentrations in localized areas within the Study Areas. It should be noted that the hypothetical offsite resident evaluation is overly conservative in that the modeling assumes the resident is located adjacent to the Study Area being evaluated. In reality, the resident would be located some distance from the Study Area boundary thereby resulting in lower estimates of exposure. For hypothetical offsite residential exposures to offsite soil, sediment and soil vapor, only the vapor intrusion pathway results in a marginally elevated risk estimate. The primary risk driver for this pathway was 1,3-butadiene. When considering more recent soil vapor sampling, this risk estimate would be even lower and similar to the target risk level of 1×10^{-6}

For offsite soils/sediments, cancer risk and noncancer hazard estimates for recreational and rancher exposures were below a cancer risk level of 1×10^{-6} and a noncancer hazard of 1.

Potential cumulative cancer risk and noncancer hazard estimates exceeded target health levels due to a few locations within a few Study Areas. The sample locations that contributed the majority to the risk estimates were RISBON-37, RISBON-41 and RISBON-63 in the FPP Study Area just south of the PSCT, RISBLT-02 in the Liquid Treatment Study Area, RISBCD-07 in the Central Drainage Study Area and RISSBC-05 in the Burial Trench Study Area. The results indicate that Site cleanup, engineering controls and/or institutional controls may be necessary to mitigate potential risks associated with these localized areas.

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