APPENDIX H
BASELINE HUMAN HEALTH RISK ASSESSMENT

Van Stone Mine Site
Baseline Human Health Risk Assessment

Prepared for:

Hart Crowser, Inc.

1700 Westlake Avenue North, Suite 200

Seattle, Washington 98109

Prepared by:

TerraGraphics Environmental Engineering, Inc.

121 S. Jackson St.

Moscow, ID 83843

www.terragraphics.com

and

Pascoe Environmental Consulting

210 Taylor Street, Rm 15

Port Townsend, WA 98368

October 29, 2013

This page intentionally left blank

Table of Contents

Appendices

List of Figures

List of Tables

Acronyms and Abbreviations

µg/dL	micrograms per deciliter
µg/L	micrograms per liter
ACGIH	American Conference of Governmental Industrial Hygienists
ALM	Adult Lead Model
AOI	Area of Interest
ARAR	Applicable or Relevant and Appropriate Requirement
CDC	Centers for Disease Control and Prevention
CLARC	Cleanup Levels & Risk Calculations
COC	Contaminant of Concern
COPC	Chemical of Potential Concern
CSM	conceptual site model
Ecology	Washington State Department of Ecology
EPC	Exposure point concentration
FS	Feasibility Study
HEAST	Health Effects Assessment Summary Tables
HHRA	Human Health Risk Assessment
HI	hazard index
HQ	hazard quotient
IARC	International Agency for Research on Cancer
IEUBK	Integrated Exposure Uptake Biokinetic Model
IRIS	Integrated Risk Information System
LOAEL	low-observed-adverse-effects level
MCL	Maximum Contaminant Level
mg/kg	milligrams per kilogram
MTCA	Model Toxics Control Act
NHANES	U.S. National Health and Nutrition Examination Survey
NIOSH	National Institute for Occupational Safety and Health
NOAEL	no-observed-adverse-effects level
OSHA	Occupational Safety and Health Administration
ppb	parts per billion
ppm	parts per million
QA	quality assurance
RfD	Reference Dose
RI	Remedial Investigation

UCL	upper confidence limit
USEPA	U.S. Environmental Protection Agency
WAC	Washington Administrative Code

Section 1.0 Introduction

This baseline Human Health Risk Assessment (HHRA) has been prepared for the Van Stone Mine in Stevens County, Washington (or "the Site") in accordance with the Washington State Model Toxics Control Act (MTCA; Washington Administrative Code [WAC] 173-340) (Ecology 2007). The purposes of a baseline HHRA are to (1) evaluate potential risk at a site and determine the primary causes of that risk, (2) help determine whether remediation response actions are necessary, and (3) help modify cleanup levels (or support a "no-action" alternative when appropriate). The results of the baseline HHRA and the Feasibility Study (FS) are to be used by the risk manager of a site to provide information for the decision-making process.

The term "cleanup level" is used in this document as the definition provided in MTCA: "the concentration of a hazardous substance in soil, water, air, or sediment that is determined to be protective of human health and the environment under specified exposure conditions [WAC 173-340-700(1)]. These are typically initially established during the scoping of the Remedial Investigation and may be further refined during the Remedial Investigation and/or Feasibility Study [WAC 173-340-350 (9)(a)]. Cleanup levels are modified and finalized as remediation levels when final cleanup actions are submitted and accepted by the Washington State Department of Ecology (Ecology).

The HHRA involves five basic elements:

1. *Data Evaluation and Initial Screening.* Available data are reviewed to identify site-specific contaminants of potential concern (COPCs). COPCs are those contaminants that exceed background levels and the lowest human health Applicable or Relevant and Appropriate Requirement (ARAR).
2. *Exposure Assessment.* The exposure assessment defines the amount, frequency, duration, and routes of receptor exposure to site-related COPCs. The exposure assessment considers both current and likely future site uses and is based on MTCA unrestricted land use scenario. Exposure point concentrations (EPCs) representative of upper-bound exposure conditions in each affected medium are also estimated in the exposure assessment.
3. *Toxicity Assessment.* The toxicity assessment summarizes (1) the nature and degree of toxicity of each COPC, and (2) the dose-response relationship (the relationship between magnitude of exposure and magnitude of adverse health effects) for each COPC. Two kinds of effects are discussed: (1) noncarcinogenic effects, and (2) carcinogenic effects. The same chemical may exert both kinds of effects.
4. *Risk Characterization.* In risk characterization, exposure and toxicity data are combined to define site-specific cleanup criteria and estimate the nature and magnitude of potential risks to defined receptor populations. Noncarcinogenic risks to human receptors are quantified by the hazard quotient (HQ), the ratio of COPC concentration in site media to the corresponding noncancer risk-based concentration. Carcinogenic risks are quantified by estimating the excess cancer risks, expressed by the ratio of the COPC concentration to the cancer risk-based levels multiplied by the acceptable cancer risk. The risk characterization will identify contaminants of concern (COCs) based on the hazard index (HI: the sum of the HQs) and carcinogenic risk.

5. *Uncertainty Analysis*. Like any other form of modeling, risk assessment relies on a set of assumptions and estimates, each of which has some element of uncertainty. The uncertainty analysis accounts for both variability in and lack of knowledge about measured and estimated parameters, allowing decision makers to better evaluate risk estimates in the context of the assumptions and data used in the assessment.

Section 2.0 Data Evaluation and Initial Screening

Analytical results collected during the Remedial Investigation (RI) were reviewed to ensure suitability for use in the HHRA. The HHRA uses background and chemical data from samples of the following environmental media collected during the RI:

- Waste rock and tailings piles.
- Surface and subsurface soils.
- Surface water from Onion Creek and its tributaries.
- Sediment from Onion Creek and its tributaries.
- Groundwater.

Data quality was evaluated as part of the RI and qualifiers were assigned by the laboratory or the validator. Estimated values ("J") were used in the risk assessment. There were no rejected values ("R") deemed unusable for risk assessment purposes. When both an original sample and a field quality assurance (QA) sample were collected from the same sample location, the higher of the two results was used. In this HHRA, all RI sample results collected from the Site in 2011 and 2012 were used to conduct the initial screening.

To identify COPCs at the Van Stone Mine, a stepwise selection process described by WAC guidance (Ecology 2007) was used. The screening process consists of three steps: 1) determine the frequency of detection, 2) compare the maximum concentrations to background concentrations, and 3) compare maximum concentrations to appropriate and relevant risk-based criteria. If contaminants pass these three screening steps, they are assumed to present potential risks to human receptors and are further evaluated in the next steps of the risk assessment process.

Table 1 summarizes the COPCs identified through this initial screening. The following COPCs in the specified environmental media were carried forward into the exposure, toxicity, and risk characterization steps:

- Soil: antimony, arsenic, cadmium, chromium, copper, lead, mercury, thallium, and zinc
- Sediment: arsenic and cadmium
- Surface water: antimony
- Groundwater: antimony, arsenic, cadmium, chromium, lead, and nickel

Thallium is the only compound without screening criteria and was retained as a COPC for a qualitative evaluation; there is no MTCA Method B cleanup level for thallium.

Results of the initial screening, including the frequency of detection, comparisons of maximum concentrations of Site contaminants with background, and risk-based screening criteria, are presented in Appendix A.

2.1 Identification of Detected Chemicals

For a given medium, contaminants that were detected in less than five percent of the samples were eliminated from further evaluation. Appendix A summarizes the detection frequencies for each contaminant and medium. The following chemicals were eliminated from further evaluation due to low percent detection:

- Soil: none
- Sediment: none
- Surface water: arsenic, beryllium, total chromium, nickel, selenium, silver, and thallium
- Groundwater: selenium

2.2 Comparison to Site Background Concentrations

Maximum concentrations of contaminants were compared to area background concentrations established during the RI. Background samples were collected near the Van Stone Mine Site for soil, sediment, and surface water, and the 90th percentile was calculated for each contaminant (Hart Crowser 2013). Contaminants with maximum concentrations less than their respective 90th percentile background value (as recommended in WAC 173-340-709) were eliminated from further evaluation. Comparative background concentrations in groundwater were unavailable, and most surface water results were below detection limits, so it was not possible to calculate 90th percentiles; therefore, all contaminants in groundwater and surface water were carried forward to the next step of the screening process. Appendix A contains tables detailing the screening process, including the background concentrations used for comparison.

The following chemicals were eliminated from further evaluation due to maximum concentrations less than the 90th percentile background values:

- Soil: selenium
- Sediment: beryllium, total chromium, copper, nickel, selenium, silver, and thallium

2.3 Screening for Human Health

Maximum concentrations of the remaining contaminants found in soil and sediment were compared to the lower of the MTCA Method B cleanup levels for unrestricted land use soil ingestion and for the protection of groundwater. Maximum concentrations found in surface water were compared to the lower of the Method B cleanup levels and State and Federal surface and drinking water quality criteria for human health. Maximum concentrations found in groundwater were compared to the lower of Method B cleanup levels and State and Federal drinking water criteria. Contaminants with maximum concentrations exceeding these criteria were retained as COPCs for human health. The RI contains a summary of the ARARs and identifies the human health risk-based concentrations used in this step of the screening process (Hart Crowser 2013).

Additionally, Appendix A contains tables detailing the screening process, including the lowest risk-based criterion used for comparison.

Sampling results for total chromium in all media were compared to MTCA B cleanup levels for chromium VI, as recommended by Ecology with the use of the Cleanup Levels & Risk Calculations (CLARC) database (https://fortress.wa.gov/ecy/clarc/CLARCCautions.aspx; Ecology 2012). Sampling results for lead and mercury were compared to MTCA A cleanup levels, as there are no MTCA B cleanup levels for these chemicals. Contaminants eliminated from the risk assessment at this step include:

- Soil: beryllium, nickel, and silver
- Sediment: antimony, lead, mercury, and zinc
- Surface water: cadmium, copper, lead, mercury (total and dissolved), and zinc
- Groundwater: beryllium, copper, mercury, silver, thallium, and zinc

Section 3.0 Exposure Assessment

3.1 Conceptual Site Model

Exposure to site contaminants can occur when contaminants migrate from the source to an exposure point, where a receptor comes into direct contact with contaminated media. An exposure pathway is complete if a receptor can ingest, inhale, or dermally absorb contaminants at a location where site-related contaminants are present. No exposure (and therefore no risk) exists unless the exposure pathway is complete.

A preliminary conceptual site model (CSM) was developed for the *Final Work Plan for Remedial Investigation/Feasibility Study, Van Stone Mine, Stevens County, Washington* (Hart Crowser, 2011) to identify the likely and potential land use, primary and secondary release mechanisms, potential exposure routes, and potential receptors. The final CSM developed for this HHRA is based on MTCA requirements and information gathered during the RI. The final CSM is presented in Figure 1-1 and discussed below.

3.1.1 Potentially Exposed Populations

The human receptors identified in the CSM for the Site are potential future residents of and visitors (such as a hiker, tourist, or fisherman) to the Van Stone Mine Site. The unrestricted (or residential) land use scenario is required by MTCA to identify cleanup levels. Other land use scenarios, such as trespasser or recreational scenarios, shall only be considered when evaluating remedy options. As a result, the final CSM used for this HHRA evaluates an unrestricted land use scenario and targets potential future residents of the Site as the exposed receptors.

3.1.2 Exposure Pathways

Under MTCA, cleanup levels are based on the unrestricted land use scenario and reasonable maximum exposures. A reasonable maximum exposure is defined as "the highest exposure that is reasonably expected to occur at a site under current and potential future site use" (WAC 173-340-708). For groundwater cleanup levels, MTCA identifies ingestion of groundwater as the

reasonable maximum exposure for most sites. For surface water cleanup levels based on human health protection, reasonable maximum exposure is identified as ingestion of fish if surface waters have the potential to support fish or as ingestion of water if surface water is suitable for use as a domestic water supply. This HHRA assumes that receptors may catch and consume fish as a complete pathway from surface water bodies at the Site. In addition, ingestion of surface water from Onion Creek is considered a complete pathway and a reasonable maximum exposure because some residents in the vicinity of the Site use water from Onion Creek for drinking water (Hart Crowser 2011). For soil and sediment cleanup levels, residential land use represents the reasonable maximum exposure scenario.

Based on site characteristics and land use, the Site was divided into five exposure units (or Areas of Interest [AOI]1–AOI5; see Figure 3 of the RI [Hart-Crowser 2013]). It is possible that current or future use of the exposure units includes ingestion of groundwater as drinking water, ingestion of surface water as drinking water, ingestion of fish, and residential land use. Following the WAC 173-340-708 reasonable maximum exposure scenarios, the exposure pathways assessed in the HHRA include (1) incidental ingestion of surface and subsurface soil, waste rock, and tailings (AOI1-AOI4); (2) incidental ingestion of sediment in Onion Creek (AOI5); (3) ingestion of impacted groundwater as drinking water (AOI2 and AOI3); (4) ingestion of fish from impacted surface water in pit lakes, tailings ponds, and Onion Creek (AOI1, AOI2, and AOI5); and (5) ingestion of impacted surface water as drinking water (AOI1, AOI2, and AOI5). These pathways are considered complete and potentially significant sources of exposure to humans and assume the highest beneficial uses (see Figure 1-1).

3.2 Exposure Point Concentrations

EPCs for each COPC were developed to quantify exposures to humans at the Site. The EPC represents the concentrations to which a receptor may be exposed over a long period of time as the individual randomly moves over the Site. The EPCs were determined for each medium within each AOI.

When there are six or more detections in the data set, the EPC is the 95 percent upper confidence limit (UCL) of the mean concentration for each medium in each AOI, calculated with ProUCL software (version 4.1.00 [USEPA 2010]). ProUCL incorporates undetected values by assigning values based on the distribution of detected values. ProUCL uses this substituted data set to determine the overall data distribution and recommends a UCL appropriate to that distribution. The ProUCL software makes UCL recommendations based on the distribution of data and sometimes recommends a 97.5 percent or 99 percent UCL. The UCL recommended by ProUCL is used as the EPC when six or more detected values exist.

When the data set consists of fewer than six detections, the maximum concentration is used as the EPC. EPCs are presented with two significant figures in Tables 2 through 16 (discussed in Section 1.4) and Appendix A. Fewer than six detections were found for COPCs in groundwater in AOI2 and AOI3.

For purposes of this HHRA, the one soil sample in AOI5 (BG12-SS) was included in the sediment EPC calculation. It is not likely that the one sample result is representative of a potential future resident's exposure if a residence abuts Onion Creek. In addition, the sample was collected near the creek bank and all sediment and soil data for Onion Creek is assumed to represent yard soil of a potential residence along Onion Creek.

Section 4.0 Toxicity Assessment

Two general types of health effects are evaluated: cancer effects and adverse noncancer health effects. This distinction is made because it is generally assumed that a dose threshold exists for noncarcinogens and that compensatory processes prevent the expression of adverse effects if humans are exposed to chemical doses below the threshold. No such threshold is generally assumed for carcinogens. Instead, it is generally assumed that there is a finite probability of developing cancer associated with any exposure to a carcinogen. As a result, carcinogens and noncarcinogens have separate toxicity criteria discussed below.

In accordance with Ecology guidance (Ecology 2007), the U.S. Environmental Protection Agency's (USEPA) online Integrated Risk Information System (IRIS, USEPA 2012a) was used to identify toxicity criteria for the Site COPCs. If toxicity criteria were not available through IRIS, the CLARC database and Health Effects Assessment Summary Tables (HEAST) were reviewed. Reference dose (RfD) values are used to evaluate noncarcinogenic health effects, and cancer potency factors are used to evaluate carcinogenic health risks.

Appendix A (Table A8) summarizes the available oral RfD values and oral cancer potency factors used in standard Method B CLARC cleanup levels. Detailed toxicity information for each COPC is provided in Appendix B. Each COPC is summarized below for cancer or noncancer health effects.

- Antimony – There is no oral cancer potency factor for antimony but an RfD exists. Consequently, antimony is only evaluated for noncancer health effects.
- Arsenic – Both a cancer potency factor and oral RfD exist for arsenic, so both cancer and noncancer effects are evaluated.
- Cadmium – There is no cancer potency factor for cadmium but separate RfD values exist for water ingestion and food ingestion (applied to soil ingestion). Consequently, cadmium is only evaluated for noncancer health effects.
- Chromium – There is no cancer potency factor for total chromium but an RfD exists for chromium VI. Consequently, total chromium results available for this Site are only evaluated for noncancer health effects using toxicity information for chromium VI, as recommended by Ecology (2012).
- Copper – There is no cancer potency factor for copper but an RfD exists. Consequently, copper is only evaluated for noncancer health effects.
- Lead – There is no cancer potency factor for lead. There is no consensus RfD because of the difficulty of identifying a threshold level for adverse health effects needed to establish an RfD. Alternatively, lead risk is evaluated through biokinetic modeling predicting a biological marker (i.e., blood lead concentrations).
- Mercury – Toxicity information for mercuric chloride is used to assess risk in this HHRA because the bulk of the information regarding toxicity resulting from oral exposure to inorganic mercury comes from studies of mercuric chloride. There is no cancer potency factor for mercury. The oral RfD (based on mercuric chloride) allows mercury to be evaluated for noncancer health effects.

- Nickel – There is no cancer potency factor for nickel. The oral RfD allows nickel to be evaluated for noncancer health effects.
- Thallium – There is no cancer potency factor or RfD for thallium.
- Zinc – There is no cancer potency factor for zinc. The oral RfD allows zinc to be evaluated for noncancer health effects.

Section 5.0 Risk Characterization

Risk characterization involves estimating the magnitude of the potential adverse health effects of site COPCs and making summary judgments about the nature of the health threats to the defined receptor population. It combines the results of the toxicity assessment and exposure assessment.

5.1 Comparison to MTCA Method B Cleanup levels

WAC 173-340 mandates that site cleanups protect the state's citizens and the environment. Ecology has established cleanup standards and requirements for hazardous waste sites to implement this statutory mandate. These pre-calculated cleanup levels are available in the online CLARC database for a large number of chemicals and exposure scenarios. Ecology integrates toxicological criteria, exposure factors, soil and drinking water ingestion rates, and target risk levels in calculating cleanup levels.

The unrestricted land use (Method B) cleanup levels for soil, surface water, and groundwater are presented in the CLARC database. Cleanup levels for soil are developed assuming that ingestion is the dominant route of exposure for metals. Surface water cleanup levels consider fish consumption as the dominant exposure route, unless the surface water body may be used for domestic drinking water, as in the case of Onion Creek (AOI5). Groundwater cleanup levels are based on ingestion. Exposure factors used to develop the cleanup levels in the CLARC database are contained in Appendix A and considered representative of reasonable maximum exposures under unrestricted land use conditions. The cleanup levels are considered protective of the expected exposure scenarios at the Site.

Method B cleanup levels were used to quantitatively assess risks associated with the following pathways: (1) incidental ingestion of surface and subsurface soil, waste rock, and tailings; (2) incidental ingestion of sediment in Onion Creek; (3) ingestion of impacted groundwater using available groundwater data, as well as the soil to groundwater protection pathway; (4) ingestion of fish; and (5) ingestion of impacted surface water as drinking water (compared to Method B cleanup levels for groundwater). Method B soil/sediment cleanup levels for mercury and cadmium are not listed in the CLARC database. To quantitatively assess risks associated with incidental ingestion of mercury and cadmium in soils/sediments, Method B cleanup levels for mercuric chloride were used and cleanup levels for cadmium were calculated using cadmium toxicity information available from IRIS (USEPA 2012a).

For unrestricted land use (standard Method B), the target risk level for estimated excess cancer risk is less than or equal to one in one million (1×10^{-6}) for known or suspected carcinogens, or less than one in one hundred thousand (1×10^{-5}) for multiple carcinogenic substances or pathways. Cancer risks for Site receptors were calculated as follows:

$$Cancer\ Risk = \frac{EPC}{Method\ B\ Cleanup\ Level\ for\ Carcinogen} \times 10^{-6}$$

Noncarcinogenic risks to human receptors are quantified by the ratio of COPC concentration in site media to the corresponding noncancer risk-based concentration (known as a HQ). Cumulative noncarcinogenic risk is expressed as a HI and sums all HQs. Risk levels for noncarcinogens under standard Method B are unacceptable if a HQ exceeds 1 for individual chemicals, and a HI exceeds 1 for multiple hazardous substances or pathways. Noncancer risks for Site receptors were calculated as follows.

$$HQ = \frac{EPC}{Method\ B\ Cleanup\ Level\ for\ Noncarcinogen}$$

$$HI = \sum HQ$$

Results for noncarcinogenic and carcinogenic risks are reported in tables with one significant figure, as suggested by USEPA guidance (2004).

5.1.1 Ingestion of Soil/Sediment

Tables 2 through 5 summarize calculated carcinogenic and noncarcinogenic risks due to incidental ingestion of soils in AOIs 1–4. Standard Method B cleanup levels (noncarcinogen) are unavailable for lead and thallium. However, lead is evaluated separately in Section 1.4.2 using the Method A cleanup level. AOI2, AOI3, and AOI4 meet the noncarcinogenic target risk levels for both the HI and HQ. AOI1 does not meet the noncarcinogenic target risk level for the HI, with cadmium, arsenic, and zinc as the highest contributors. The calculated carcinogenic risks for AOI1, AOI2, AOI3, and AO14 are above the target risk for arsenic. The calculated cumulative carcinogenic risk in AOI1 exceeds the target risk for multiple COCs . Table 6 summarizes calculated carcinogenic and noncarcinogenic risks due to incidental ingestion of soil/sediment in Onion Creek (AOI5). The calculated noncarcinogenic risk meets the target risk level; however, arsenic concentrations exceed carcinogenic target risk levels.

5.1.2 Ingestion of Impacted Groundwater

Tables 7 and 8 summarize calculated carcinogenic and noncarcinogenic risks due to ingestion of groundwater in AOI2 and AOI3, respectively. Lead is evaluated separately in Section 1.4.2. No groundwater data are available for AOI1, AOI4, or AOI5. The carcinogenic and noncarcinogenic target risk levels are acceptable in AOI2. The calculated risks in AOI3 do not meet either carcinogenic target risk level (due to arsenic) or the noncarcinogenic target risk level (due to antimony, arsenic, chromium, and nickel).

Under MTCA, soil concentrations must also be evaluated for their potential to cause groundwater contamination in the future. The CLARC database contains calculated cleanup levels for soil and sediment for the protection of groundwater using MTCA Method B. These soil cleanup levels are based on a three-phase (soil, water, and air) partitioning model using default input parameters, providing a concentration that is intended to be protective of groundwater under most circumstances and conditions. Tables 9 through 13 compare the EPCs for soil and sediment concentrations found at each AOI to these cleanup levels. In AOI1,

antimony, arsenic, cadmium, and zinc values exceeded cleanup levels in soil for the protection of groundwater. Arsenic and cadmium exceeded cleanup levels in AOI4. Cadmium exceeded cleanup levels in AOI2, AOI3, and AOI5. Results of the protection of groundwater pathway are not included in the cancer risk, HQ, and HI equations presented above, but are discussed separately in the conclusions.

5.1.3 Ingestion of Fish

Table 14 summarizes calculated carcinogenic and noncarcinogenic risks due to ingestion of fish in AOI5. Although surface water data were available for AOI1 and AOI2, there were no detectable antimony results. The EPC for antimony in Onion Creek is estimated to result in no adverse effects on human health based on fish consumption.

5.1.4 Ingestion of Impacted Surface Water

Table 15 summarizes calculated carcinogenic and noncarcinogenic risks due to ingestion of surface water in Onion Creek (AOI5). Target risk levels for health effects are met in AOI5 for antimony. Lead is evaluated separately in Section 1.4.2.

5.2 Lead Risk

The approach to human health risk assessment for lead differs from that of other metals in several ways. Among the important considerations are the nature of the health effects, the behavior of lead in the body, measurements of biological effects, indices of risk, how risks are quantified, availability of data (both site-specific and in the national experience), and the relationships between absorption levels and environmental media.

The adverse health effects of lead have been related to blood lead concentration or micrograms of lead per deciliter of whole blood (µg/dL). As a result, blood lead levels have evolved as indices of health criteria. Currently, the USEPA lead health criterion with respect to children assumes no child in an identifiable population should have a greater than 5 percent probability of a blood lead level of 10 µg/dL or greater (USEPA 1998). Recently, the Centers for Disease Control and Prevention (CDC) lowered the blood lead level of concern to 5 µg/dL and agreed that value should be termed a reference value (as opposed to a level of concern) because no safe blood lead level in children has been identified (CDC 2012). The health effects observed at a blood lead level of 10 µg/dL or less are sub-clinical, meaning that, generally, these effects cannot be diagnosed in an individual child. Establishment of these sub-clinical health effects of lead was based on numerous scientific studies involving comparisons of large groups of children (NAS 1993, ATSDR 2007b, and CDC 1997).

Lead health risk assessment involves modeling blood lead levels and relating those to national criteria because there is no consensus RfD for lead. Risks to population groups are assessed by determining the expected or observed percentage of the population to exceed those criteria. Risk to individuals is often expressed as the probability that the subject's blood lead level will exceed the specified level (e.g., 10 µg/dL). Models have been developed to predict blood lead levels resulting from different lead intake scenarios. Those are the Integrated Exposure Uptake Biokinetic Model (IEUBK) for predicting childhood blood-lead levels and the Adult Lead Model (ALM) for assessing fetal blood lead levels through adult exposures (USEPA 2003). The IEUBK integrates site-specific soil and house dust lead concentrations with default water, food, and air

lead concentrations to calculate individual probabilities of blood lead exceedances for a hypothetical child population living at a site. If lead concentrations in site media exceed a screening level for soil or drinking water, associated risk may be estimated using the IEUBK and ALM, as appropriate.

It is important to understand these differences for lead relative to other contaminants and how the methodologies employed relate to MTCA. WAC 173-340 provides a Method A soil cleanup level based on preventing unacceptable childhood blood lead levels, and provides a groundwater cleanup level based on State and Federal drinking water quality, both for unrestricted land use (Ecology 2007). However, no Method B cleanup levels are calculated for lead because if Method B or C is to be applied, then the IEUBK and/or ALM would be employed.

Risks associated with exposure to lead at the Van Stone Mine Site are discussed relative to the Method A soil cleanup level of 250 milligrams per kilogram (mg/kg) and State and Federal drinking water criteria to determine if site concentrations may pose an unacceptable risk to human receptors. The Method A cleanup level targets potential future children living at the Site and is considered a reasonable maximum exposure scenario. Incidental soil ingestion by a potential future child resident in AOIs 1–4 would result in excess lead health risk as each AOI's EPC is greater than the 250 mg/kg cleanup level. AOIs 1 and 3 have the highest levels of lead, with EPCs of 7,300 mg/kg and 1,400 mg/kg, respectively (Table 16). The EPCs for AOIs 2 and 4 are 350 mg/kg and 400 mg/kg, respectively. Lead concentrations in soil and sediment from Onion Creek (AOI5) are low enough not to present a potential estimated risk from incidental ingestion. In addition to the soil ingestion pathway, groundwater lead concentrations in AOI3 exceed the State and Federal standards for lead in drinking water (Table 16). Based on the evaluation of each AOI as an exposure area, AOI3 (the Lower Tailings Pile area) poses the highest lead health risk to potential future residential child receptors. AOIs 1, 2, and 4 also pose excess lead health risk.

5.3 Qualitatively Evaluated Pathways

5.3.1 Inhalation of Fugitive Dusts from Soil

Inhalation of fugitive dusts from soil is a consideration for semivolatile organics and metals in surface soils. However, USEPA believes that fugitive dust soil screening levels need not be calculated for most metals, with the exception of chromium due to the carcinogenicity of chromium VI through the inhalation exposure pathway. The USEPA generic soil screening level for chromium VI inhalation (16 mg/kg, USEPA 2012b) is below the MTCA B soil ingestion screening level (240 mg/kg). All soil EPCs for total chromium at the Site were below 16 mg/kg, indicating that chromium VI does not significantly contribute to exposure in these AOIs for the fugitive dust inhalation exposure pathway.

5.3.2 Inhalation of Water

Contaminated water may pose risk by the inhalation route if the contaminants present are volatile; however, the COPCs identified at the Site are not. MTCA assumes that ingestion of water is the dominant pathway for groundwater, and ingestion of fish is the dominant pathway for surface water. Exposure from the water inhalation pathway is likely insignificant relative to other complete pathways evaluated in this HHRA.

5.3.3 Dermal Contact with Surface Water, Groundwater, Sediment, and Soil

The dermal exposure route is complete for surface water, sediment, and soil, and incomplete for groundwater. MTCA assumes that ingestion of water is the dominant pathway for groundwater, and ingestion of fish is the dominant pathway for surface water. Risks from dermal pathways are not assessed quantitatively.

USEPA risk assessment guidance assumes that the water dermal pathway is significant only if dermal absorption is likely to be greater than10 percent of the direct ingestion dose (USEPA 1992, 2004). USEPA provides estimated percentages to assist in determining whether the dermal pathway should be evaluated for a given chemical. Antimony (identified as a COPC in surface water) has an estimated dermal to oral percentage of 3.5 percent (USEPA 2004), indicating the surface water dermal pathway is likely not significant (less than 10 percent). Antimony, arsenic, mercury, and nickel (identified as COPCs in groundwater) also have estimated dermal to oral percentages less than 10 percent (3.5, 0.55, 7.5, and 2.63 percent, respectively, USEPA 2004), indicating the groundwater dermal pathway is likely not significant for these chemicals. The other COPCs identified in groundwater, cadmium and chromium (assumed to be chromium VI), have estimated dermal to oral percentages greater than 10 percent (10.5 and 42 percent, respectively, USEPA 2004), indicating the dermal pathway may be significant. However, dermal contact with groundwater is currently an incomplete pathway at the Site. In addition, concentrations available for this HHRA are for total chromium and should be further evaluated for chromium VI and III.

For the soil/sediment dermal pathway, USEPA guidance indicates that it is not necessary to estimate dermal absorption of inorganics that do not exceed 10 percent of the ingested dose, when the fraction absorbed from the gastrointestinal tract has been estimated or quantified (USEPA 2007). If the fraction absorbed from the gastrointestinal tract has not been estimated or quantified, the soil absorption fraction may be used to screen contaminants for this pathway. Estimated soil dermal absorption fractions for arsenic and cadmium (3 and 0.1 percent, respectively) have been provided in USEPA guidance (USEPA 2004), and these fractions suggest that dermal exposure to soils is less significant than direct ingestion. Other inorganic dermal absorption fractions are not available. However, it is assumed that the other metals of potential concern have dermal absorption fractions less than 10 percent, and therefore direct ingestion is a more significant pathway than the soil dermal pathway at the Site.

5.3.4 Ingestion of Homegrown Produce

Consumption of garden fruits and vegetables grown in contaminated residential soils can result in a risk to human health. Soil contaminants enter the body primarily by incidental ingestion, and ingestion of homegrown produce could result in ingestion of soil that adheres to the plant. The risk associated with ingestion of soil on homegrown produce would be included in risk estimates for incidental soil ingestion.

There is some potential for plant uptake of contaminants from soil. However, with the exception of arsenic, metal concentrations in soil considered toxic to plants are well below levels that may impact human health through plant uptake. This implies that phytotoxic effects may prevent completion of this pathway for most metals (USEPA 1996). Arsenic was identified as a COPC in soils in AOI1, AOI2, AOI3, and AOI4, indicating that ingestion of contaminated homegrown produce may potentially be a complete pathway that will contribute to overall exposure.

However, exposure from this pathway is likely insignificant relative to other complete pathways evaluated in this HHRA.

5.4 Adjustment for Total Risk and Hazard Index

WAC 173-340 requires the evaluation of cumulative risk when Method B levels are used (Ecology 2007). Evaluation of cumulative cancer risk was accomplished by summing all cancer risks for each COPC and each quantified pathway for a human receptor population. Cumulative risk for noncancer effects was evaluated by summing HQs for each COPC associated with the same toxic effect endpoint. Table 17 summarizes calculated carcinogenic and noncarcinogenic risks (i.e., HIs) due to multiple pathways within each AOI.

Lead health risks and the protection of the groundwater pathway are not included in these calculations adjusting for total risk. Soil and sediment lead concentrations would need to be adjusted to the MTCA Method A cleanup level of 250 mg/kg. Further evaluation of contaminant migration from surface soils and sediment to the groundwater should be conducted prior to adjusting the protection of groundwater pathway's cleanup levels.

5.4.1 AOI1 – Mill Area

The target carcinogenic risk of 1×10^{-5} and target HI of less than 1 for multiple substances and multiple pathways is not met in AOI1 (Table 17). A slightly higher estimated carcinogenic risk due to arsenic from the soil/sediment ingestion pathway occurs (2×10^{-5}). The EPC for arsenic is 10 mg/kg (Table 2). WAC specifies that when making adjustments to cleanup levels, the concentration for individual substances should not be reduced to concentrations less than the practical quantitation limit or natural background. The carcinogenic target risk for multiple substances and pathways would be met if the natural background arsenic concentration in soil (5.04 mg/kg, Hart Crowser 2012) is used as the arsenic cleanup level. Carcinogenic risk would be estimated at 8×10^{-6} (see Table 18). However, the carcinogenic target risk of 1×10^{-6} for an individual substance would not be met at background concentrations.

The noncarcinogenic risk in AOI1 is primarily due to arsenic, cadmium, and zinc concentrations from the soil/sediment ingestion pathway. If the background arsenic concentration (5.04 mg/kg, Hart Crowser 2012) and the background cadmium concentration (1.5 mg/kg, Hart Crowser 2012) were used as the cleanup levels, the total noncarcinogenic risk for AOI1 would be 0.9, which would meet the target noncarcinogenic risk for multiple chemicals in the soil ingestion pathway and multiple pathways within AOI1 (Table 18).

5.4.2 AOI2 – Upper Tailings Pile Area

The target carcinogenic risk of 1×10^{-5} and target HI for multiple substances and multiple pathways is met in AOI2 (Table 17).

5.4.3 AOI3 – Lower Tailings Pile Area

In AOI3, the target carcinogenic risk for multiple substances and multiple pathways is not met primarily due to the soil/sediment ingestion and groundwater ingestion pathways (see Table 17). Calculated carcinogenic risk from the groundwater ingestion pathway (3×10^{-4}) does not meet the target carcinogenic risk for multiple substances and is much greater than the soil ingestion

pathway (8×10^{-6}). There are no available background levels for arsenic in groundwater for the area. However, 85 percent of groundwater samples collected near the Site from nearby residential wells was below the detection limit of 0.0038 mg/L for arsenic. If the detection limit of 0.0038 mg/L is used as the cleanup level for arsenic in groundwater, calculated carcinogenic risk would be 7×10^{-5} (Table 19). This still exceeds the target carcinogenic risk level for multiple pathways or chemicals; however, there are no other quantitative risk contributors that can be adjusted. In addition, it is not feasible to adjust a cleanup standard to less than the practical quantitation limit or background concentrations (WAC 173-340-708). Therefore, the target carcinogenic risk for multiple substances in the groundwater ingestion pathway and for multiple pathways in AOI3 cannot be met due to arsenic.

The elevated noncarcinogenic risk in AOI3 is mainly attributed to the risk from groundwater ingestion (HI of 20), and less to the risk from soil ingestion (HI of 0.5) (Table 17). Groundwater background concentrations are unavailable for the area and as previously discussed, adjusted cleanup levels should not be less than the practical quantitation limit. If cleanup levels for antimony, arsenic, cadmium, chromium, and nickel were adjusted to the method detection limits, noncarcinogenic risk of multiple substances in the ingestion of groundwater pathway would equal the target risk criterion of 1 (Table 19), with arsenic as the main contributor (HQ of 0.8). Consequently, the target noncarcinogenic risk for all COCs and pathways in AOI3 cannot be met because it is not possible to further reduce the calculated risk in the groundwater ingestion pathway and the addition of the soil ingestion pathway will result in a HI greater than 1.

5.4.4 AOI4 – Tailings Pipeline and Road Area

The target carcinogenic risk of 1×10^{-5} for multiple substances and multiple pathways is met in AOI4 (Table 17). Calculated noncarcinogenic risk is below the target HI of 1 for multiple substances and multiple pathways in AOI4.

5.4.5 AOI5 – Onion Creek

The target carcinogenic risk of 1×10^{-5} for multiple substances and multiple pathways and the HI target of 1 is met in AOI5 (Table 17).

Section 6.0 Uncertainty Summary

The purpose of this HHRA is to identify areas at the Van Stone Mine Site that pose potential risks and hazards greater than public health target goals established by Ecology. Estimating and evaluating health risks from exposure to environmental chemicals is a complex process with inherent uncertainties. Uncertainty reflects limitations in knowledge and simplifying assumptions that must be made in order to quantify health risks. The uncertainty analysis plays a key role in understanding the implications for the remedy and devising strategies to achieve a safe, effective, and efficient remedy in the FS process.

In this assessment, uncertainties relate to (1) the development of media concentrations and assumptions about exposure, (2) the assumptions about toxicity, and (3) the characterization of health risks. This section qualitatively evaluates each of these potential sources of uncertainty to determine the likely degree of uncertainty associated with the risk estimates, and whether the uncertainty is more likely to over- or under-estimate risk.

In general, when quantifying exposure and toxicity, MTCA risk assessment procedures are conservative in order to protect human health. MTCA risk assessment procedures are more likely to indicate that chemicals exceed target risk goals when health risks may actually be negligible, rather than indicate that chemicals are not a health risk when in fact they may be. This conservative approach is used to ensure that false-negative conclusions about health risk do not occur.

6.1 Available Data and Exposure Point Concentrations

6.1.1 Available Data

Measurement errors and random and/or systematic errors arise from the inability to measure variables precisely and accurately (e.g., field equipment and laboratory protocols), or because the quantity being measured varies spatially or temporally. Basic methodological (laboratory processing and equipment) errors were less of a problem for the data set in this HHRA, given the reliance on standardized protocols and other QA and quality control dictated criteria. The principal uncertainties with the data used in this HHRA lie more with spatial and temporal errors in sampling.

It is not possible to sample every square inch of potentially impacted media at a site. Instead, a limited number of samples must be obtained to represent the contaminant characteristics of a larger medium. This introduces uncertainty in the development of media concentrations. The sampling strategies were, in general, designed to prevent underestimation of media concentrations, thus avoiding an underestimation of human health risks.

Spatial and temporal errors apply to the environmental exposure measurements. The Site is large in area, and the five AOIs were used to differentiate exposure units; therefore, the data for each AOI were considered to represent environmental media exposures to potential future residents within each AOI. For example, a residential exposure scenario in AOI5 (Onion Creek) assumes that a future residence may abut the Creek. In order to assess exposure to a potential resident in AOI5, the analytical data obtained from the single soil sample and all sediment samples collected from AOI5 are assumed to represent possible future soil exposures to adults and children from their property (temporal), which may be located at any point over the length of Onion Creek (spatial).

Little to no data were available for some AOIs for certain media and exposure routes. Three surface water samples were collected in each of AOI1 and AOI2, and none were collected in AOI3 and AOI4. There were two groundwater samples in AOI2, five in AOI3, and none in AOI1, AOI4, and AOI5. Exposure from the groundwater ingestion pathway was evaluated for these AOIs using soil/sediment cleanup levels for the protection of groundwater. There was one soil sample in AOI5; it was included with the sediment data in this assessment. It is unknown if these data limitations over- or under-estimate exposures and subsequent risks.

In addition to the amount of data per AOI, samples were purposefully collected in a biased manner at the Site in order to characterize the worst-case scenario. For example, in AOI1 at the upper mill area, stained soil, waste rock piles, etc. were targeted for sampling. This type of sampling approach may increase an EPC and likely represents maximum exposures.

6.1.2 Receptor Population and Exposure Pathways

The unrestricted land use scenario assumes residential use by an adult and/or child receptor. Currently, rural residential properties exist near the Site, and it is plausible that a future residence could be located on or abutting the site without Institutional Controls in place. However, people do not currently live on site, although a small residential population (site caretakers) abuts the site to the north of AOI1. With the exception of some people using Onion Creek as a drinking water source, current land use is recreational (Hart Crowser 2011). This risk assessment likely overestimates current exposures and risk related to the current land use because exposure factors (such as duration and frequency) are greater for a residential scenario than for a recreational scenario.

Although the Northeast Fork of Onion Creek in the mine site area is categorized as a confirmed fish-bearing Type 3 Stream and bull trout are known to populate the area, it is unknown whether sufficient fish of edible size can be caught from Onion Creek in the vicinity of the mine site at levels assumed under the MTCA ingestion of fish pathway. Additionally, the surface waters of the upper and lower tailings pit lakes do not currently sustain a fishery. This risk assessment assumes there is exposure due to fish ingestion that would likely overestimate risk; however, all target risk criteria based on this pathway were met.

6.1.3 Development of Exposure Point Concentrations

In addition to the available data and the spatial and temporal error, there is inherent uncertainty in calculating an EPC. First, there are a variety of methods for determining the UCL of a population. In this HHRA, the recommended UCL calculated by ProUCL (version 4.1) was used, even if it was not a 95 percent UCL. Difficulty in estimating underlying data distributions can also occur with a large number of non-detected results. When there were fewer than six detected sample results for a specific medium in an AOI, the maximum concentration was used as the EPC. This is likely an overestimate of the exposure, but again, is conservative in protecting human health.

ProUCL recommended 97.5 percent and 99 percent UCLs for copper in soil of AOI1; antimony, chromium, copper, mercury, and zinc in soil of AOI2; chromium and zinc in soil of AOI3, and mercury in soil of AOI4. In combination with the biased sampling design, the soil EPCs for these are likely biased high. The maximum concentration was used as the EPC in groundwater for cadmium, chromium, lead, and nickel in AOI2, and for antimony, arsenic, cadmium, chromium, lead, and nickel in AOI3. The soil EPCs for these COCs are likely biased high.

6.2 Toxicity Criteria

Reference doses and cancer potency factors are quantitative toxicity criteria with inherent uncertainty that are used to assess noncarcinogenic health effects and carcinogenic risk. A chronic RfD is an estimate of a lifetime (70 years) daily chemical dose that is likely to result in no appreciable deleterious noncarcinogenic effects. To derive an RfD, a series of professional judgments are made to assess the quality and relevance of human or animal data and to identify the critical study and the most critical toxic effect. These criteria are generally developed by USEPA risk assessment work groups and are listed in USEPA risk assessment guidance documents and databases. For each factor representing a specific area of uncertainty inherent in the extrapolation from the available data, an uncertainty factor is applied. Uncertainty factors

generally consist of multiples of 10, although values less than 10 are sometimes used. Data typically used in developing the RfD are the highest "no-observable-adverse-effect" levels for the critical studies and effects of the noncarcinogen, which means that the RfD is likely to overestimate risk.

Cancer potency factors are chemical-specific values used to calculate the risk of cancer resulting from exposure to carcinogenic chemicals. A higher value implies a more potent carcinogen. USEPA develops potency factors from animal studies or, where possible, from epidemiological data. Because animal studies use much higher doses over shorter periods of time than the exposures generally expected for humans, the dose-response relationship from the dose range used in animal studies is extrapolated to the low dose range generally experienced by humans typically using a "linearized multistage" mathematical model. To ensure protectiveness, potency factors are typically derived from the 95 percent UCL of the slope. Thus, actual risks are unlikely to be higher than those predicted and may be considerably lower.

The potential toxicity-modifying interactions between COPCs at the site are numerous and complex. Some of these interactions could be expected to increase toxic effects, while some would reduce toxic effects. As a result, it is difficult to estimate if risks are over- or under-estimated due to toxicity criteria.

6.3 Characterization of Health Risks

Risks in this HHRA are quantitatively evaluated by comparing EPCs to MTCA B cleanup levels. Uncertainty in risk characterization can be attributed to EPC derivation (previously discussed in section 1.5.1), chemicals and pathways that are not quantitatively assessed, and uncertainties in the derivation of cleanup-level calculations and models. In addition, uncertainty in risk characterization arises when surrogate toxicological information is used, as is the case with chromium and mercury.

6.3.1 EPC Derivation

It is possible that the use of the maximum concentrations or UCLs as EPCs biased the resulting risk calculations high. Alternatively, some AOIs had only a few samples collected in a given medium, with no detected results for certain COPCs. For example, there were no detected antimony results for surface water in AOI1 and AOI2, but only three samples were collected in each AOI. The ProUCL software will not compute UCLs with less than five sample results. The assumption that the EPC is below the limit of detection for antimony in surface water in AOI1 and AOI2 may underestimate characterized risk for these areas and the associated pathways.

6.3.2 Total Risk

Total risk sums each COPC and each identified pathway. Risk due to multiple substances and pathways within each AOI was evaluated and summarized in Section 1.4. Total risk may be underestimated because it does not include calculated risk from lead and thallium.

MTCA Method B cleanup levels are derived for assumed dominant routes of exposure, and subsequent risks for these pathways are quantified. Risk from all potential pathways identified in the preliminary CSM is not calculated under MTCA. Risks are potentially underestimated if additional pathways are complete but not quantified (such as ingestion from homegrown

produce) because these pathways could add to total risk. Additionally, data were not available for all pathways in all AOIs. Quantitative risks from groundwater ingestion in AOI1, AOI4, and AOI5 were not available. The cumulative risks in AOI1, AOI3, AOI4, and AOI5 may therefore be underestimated due to the exclusion of chemicals and pathways.

Furthermore, the use of individual exposure areas within the site may underestimate total risk because receptors may utilize multiple exposure areas. An unrestricted land use scenario could assume that a potential future resident living in AOI2 might eat fish from AOI5; however, the total risk evaluated for AOI2 reflects only exposure within AOI2 itself, and does not contain the biota ingestion pathway evaluated for AOI5. This likely underestimates risk in AOI2 because it does not contain the fish ingestion pathway.

6.3.3 Cleanup Values

MTCA B cleanup levels are calculated using accepted exposure factors. The calculated cleanup levels make conservative assumptions about exposures, and are therefore protective of human health. In general, MTCA risk assessment procedures are more likely to indicate that chemicals exceed target risk goals when health risks may actually be negligible, rather than indicate that chemicals are not a health risk.

There is additional uncertainty associated with MTCA Method B soil cleanup levels for the protection of groundwater pathway calculated using a 3-phase model. Model uncertainty arises from the use of surrogate variables, the exclusion of variables that should be included, the assumption that factors used in a model apply to the site, abnormal conditions, and incorrect model forms. This is of special concern when evaluating soil/sediment concentrations for the protection of groundwater pathway. The default assumptions used in this model approach are not site-specific and failure to correctly specify these variables can lead to uncertainties in interpreting quantitative results. The Method B cleanup levels calculated using the 3-phase model are not site-specific, and use variables that require assumptions about site characteristics, including distribution coefficients (which are based on soil fraction of organic carbon), water-filled soil porosity, air-filled soil porosity, Henry's law constant, and dry soil bulk density. If soil at the Site differs from these assumptions, risk may be over- or underestimated.

6.3.4 Chromium

In this HHRA, chromium was evaluated using toxicity data and cleanup levels for chromium VI, which is the most toxic form of chromium. This is the recommended approach discussed in the CLARC database (Ecology 2012). Using this method, chromium poses an unacceptable noncarcinogenic risk in groundwater ingestion pathway in AOI3. Sample results from the Site are for total chromium, which consists of several states of chromium (potentially including chromium VI). It is possible that risk due to chromium is an overestimate because total chromium results were compared to chromium VI levels. Further evaluation should be completed to identify the chromium species present at the Site. The maximum result for chromium in groundwater in AOI3 was used as the EPC, which also suggests that risk may be overestimated; however, due to the small sample size (5) for groundwater data in AOI3, it is not possible to determine whether chromium risk in AOI3 is over- or underestimated.

Section 7.0 Conclusions

7.1 Summary of Risk Characterization

Based on a screening evaluation, numerous metals were identified as COPCs in soils and groundwater at the Site. Arsenic and cadmium were identified as COPCs in sediment. Antimony was identified as a COPC in surface water. Exposure point concentrations were defined based on the UCLs of the mean concentrations, or maximum concentration of each COPC within each type of media in each AOI. These EPCs were compared to MTCA B carcinogenic and noncarcinogenic cleanup levels to calculate risk.

Specific pathways were evaluated quantitatively and qualitatively to evaluate carcinogenic and noncarcinogenic risks. Ingestion of soils/sediment in AOI1, AOI2, AOI3, AOI4, and AOI5 poses unacceptable carcinogenic risk due to arsenic as an individual contaminant. Soil/sediment ingestion in AOI1 poses unacceptable noncarcinogenic risk primarily due to arsenic, cadmium, and zinc. Groundwater ingestion in AOI3 poses an unacceptable carcinogenic risk due to arsenic and an unacceptable noncarcinogenic risk primarily due to arsenic, antimony, chromium, and nickel.

The groundwater ingestion pathway was further evaluated by comparing soil/sediment concentrations to MTCA B soil cleanup levels for the protection of groundwater. Antimony, arsenic, cadmium, and zinc were identified as COCs in AOI1; cadmium was identified as a COC in AOIs 2, 3, and 5; and arsenic and cadmium were identified as COCs in AOI4. Groundwater data were not collected during the RI in AOI1, AOI4, and AOI5. It is therefore unknown if these chemicals are currently of concern in groundwater in these AOIs. Alternatively, cadmium was identified as a COPC in AOI2 and AOI3 where groundwater data are available. Current data in these areas indicate acceptable risks from cadmium due to the ingestion of groundwater. However, based on the protection of groundwater pathway, it is possible that cadmium may become a concern in the future if soils remain in place.

7.1.1 AOI1 – Mill Area

Cumulative risk was calculated and evaluated to account for exposure resulting from multiple substances and multiple pathways. AOI1 does not meet the carcinogenic or noncarcinogenic target risks due to the soil ingestion pathway. Elevated carcinogenic risk is due to arsenic concentrations, and noncarcinogenic risk is due to arsenic, cadmium, and zinc concentrations. If the background arsenic concentration (5.04 mg/kg, Hart Crowser 2012) and the background cadmium concentration (1.5 mg/kg, Hart Crowser 2012) were used as the soil cleanup levels, the target carcinogenic risk of 1×10^{-5} would be met for multiple substances and multiple pathways. The noncarcinogenic target of 1 would also be met. However, lead poses unacceptable risk in AOI1, and a cleanup level different from the Method A lead value of 250 mg/kg was not quantified. Additionally, antimony, arsenic, cadmium, and zinc were identified as COCs when evaluated using the protection of groundwater pathway.

7.1.2 AOI2 – Upper Tailings Pile Area

Total risks in AOI2 meet the target carcinogenic and noncarcingenic risks. However, lead poses unacceptable risk in AOI2, and a cleanup level different from the Method A lead value of 250

mg/kg was not quantified. In addition, cadmium was identified as a COC via the protection of groundwater pathway.

7.1.3 AOI3 – Lower Tailings Pile Area

AOI3 does not meet either the target carcinogenic or noncarcinogenic risk. Elevated carcinogenic risk is mainly caused by arsenic in the groundwater ingestion pathway, with some additional risk from arsenic in the soil ingestion pathway. If the detection limit of 0.0038 mg/L is used as the cleanup level for arsenic in groundwater, calculated carcinogenic risk would still exceed the target carcinogenic risk level for multiple pathways or chemicals. Therefore, the target carcinogenic risk for multiple substances in the groundwater ingestion pathway and for multiple pathways in AOI3 cannot be met.

The elevated noncarcinogenic risk in AOI3 is mainly attributed to the risk from several chemicals in the groundwater ingestion pathway, with some additional risk from the soil ingestion pathway. If cleanup levels for antimony, arsenic, cadmium, chromium, and nickel were adjusted to the method detection limits, noncarcinogenic risk of multiple substances from the ingestion of groundwater pathway would equal the target risk of 1. Since it is not possible to further reduce the calculated risk in the groundwater ingestion pathway, and the soil ingestion pathway adds additional risk, the target noncarcinogenic risk in AOI3 cannot be met.

In addition, lead poses unacceptable risk in AOI3, as well as cadmium in the soil for protection of groundwater pathway.

7.1.4 AOI4 – Tailings Pipeline and Road Area

Total risks in AOI4 meet the target carcinogenic and noncarcingenic risks. However, lead also poses unacceptable risk in AOI4, and a cleanup level different from the Method A lead value of 250 mg/kg was not quantified. Soil arsenic and cadmium concentrations were identified as a potential threat to groundwater via the protection of groundwater pathway.

7.1.5 AOI5 – Onion Creek

Total risks in AOI5 meet the target carcinogenic and noncarcingenic risks. Lead concentrations in sediment and soil were not expected to cause excessive risk. However, cadmium was identified as a COC in the protection of groundwater pathway.

7.2 Recommendations

While the adjustment of lower cleanup standards to meet target carcinogenic and noncarcinogenic risks for multiple substances and pathways is mathematically possible, further evaluation of the data is recommended prior to adopting these revised cleanup levels. There may be areas of relatively high contamination or hot spots within an AOI. If these hot spots or areas were remedied and background concentrations or quantitation limits were assumed, resulting EPCs may be less than screening levels or may not contribute to elevated risk. Additionally, the protection of groundwater pathway identified COCs; however, due to the various levels of uncertainty previously discussed, further evaluation of this pathway and contaminant migration modeling is recommended prior to adopting cleanup levels.

Chromium was identified as a chemical that contributes to unacceptable risk through the groundwater ingestion pathway in AOI3. Samples from AOI3 should be re-evaluated to determine the oxidation state of chromium (such as chromium VI) to refine the risks attributed to this element in groundwater. Chromium VI is more toxic than chromium III and it is possible that a low percentage of the total chromium observed at the Site is chromium VI.

Section 8.0 Summary of Site Human Health Risks

The HHRA narrowed the focus to COPCs for human health by screening for frequency of detection, and comparison of site-wide maximum contaminant concentrations with site-specific background levels and risk-based criteria. The screens identified numerous metals as COPCs for human health in soils and groundwater, arsenic and cadmium in sediment, and antimony in surface water.

For this HHRA, the Site was evaluated assuming unrestricted land use, with residents having access to the entire Site. This assumption is health protective under MTCA and is considered conservative; the extent of potential residential exposures at the Site is unknown.

EPCs were identified as the 95 percent UCLs of the mean concentrations (or maximum concentrations where data were insufficient) of each COPC within each type of media in each AOI. To calculate risk, the EPCs were compared to MTCA Method B carcinogenic and noncarcinogenic cleanup levels, and lead was evaluated using the Method A cleanup level (summarized in Table 20 and described below).

8.1 AOI1 – Mill Area

Ingestion of soils/sediment in AOI1 was found to pose unacceptable carcinogenic risk due to arsenic, and unacceptable noncarcinogenic risk due to arsenic, cadmium, and zinc. If the background arsenic concentration (5.04 mg/kg, Hart Crowser 2012) and the background cadmium concentration (1.5 mg/kg, Hart Crowser 2012) were used as the soil cleanup levels, the target carcinogenic risk of 1×10^{-5} would be met for multiple substances and multiple pathways. The noncarcinogenic target of 1 would also be met. Lead poses unacceptable risk, and a cleanup level different from the Method A value of 250 mg/kg was not quantified. Additionally, antimony, arsenic, cadmium, and zinc were identified as soil COCs for the protection of groundwater pathway.

8.2 AOI2 – Upper Tailings Pile Area

Ingestion of arsenic in soils/sediment in AOI2 poses unacceptable carcinogenic risk; however, total risks for all COPCs and pathways meet the target carcinogenic and noncarcinogenic risk criteria. Lead poses unacceptable risk in AOI2, and a cleanup level different from the Method A value of 250 mg/kg was not quantified. In addition, cadmium was identified as a soil COC via the protection of groundwater pathway.

8.3 AOI3 – Lower Tailings Pile Area

Ingestion of soils/sediment in AOI3 poses unacceptable carcinogenic risk due to arsenic. Groundwater ingestion in AOI3 poses an unacceptable carcinogenic risk due to arsenic and an unacceptable noncarcinogenic risk due to antimony, arsenic, cadmium, chromium, and nickel. If the detection limit of 0.0038 mg/L is used as the cleanup level for arsenic in groundwater, calculated carcinogenic risk would still exceed the target carcinogenic risk level for multiple pathways or COCs. If cleanup levels were adjusted to method detection limits for antimony, arsenic, cadmium, chromium, and nickel, total noncarcinogenic risk from multiple pathways would still exceed the target risk.

In addition, lead poses unacceptable risk in AOI3, as well as cadmium in the soil for protection of groundwater pathway.

8.4 AOI4 – Tailings Pipeline and Road Area

The target carcinogenic risk of 1×10^{-5} and target HI of 1 are met for multiple substances and multiple pathways in AOI4; however, soil ingestion poses unacceptable carcinogenic risk for arsenic. Lead poses unacceptable risk in AOI4 and a cleanup level different from the Method A value of 250 mg/kg was not quantified. Soil arsenic and cadmium concentrations were identified as potential threats to groundwater in AOI4.

8.5 AOI5 – Onion Creek

The target carcinogenic risk of 1×10^{-5} and target HI of 1 are met for multiple substances and multiple pathways in AOI5. However, arsenic individually poses unacceptable carcinogenic risk via the ingestion of soils/sediment pathway.

Section 9.0 Summary of Further Recommendations

Recommendations for further evaluation to refine the risk estimates consist of the following:

1) Re-evaluate the adjustment of human health cleanup standards based on assumed remediation of hot spots by replacing remediated sample areas with background concentrations.
2) If after the above recommendation is accomplished and soil lead concentrations exceed 250 mg/kg, then evaluate human health risks from exposure to lead using the IEUBK and ALM.
3) Further evaluate the protection of groundwater pathway.
4) Determine the oxidation state of chromium in groundwater to refine human health risks.

Section 10.0 References

Agency for Toxic Substances and Disease Registry (ATSDR). 1992a. Toxicological Profile for Antimony and Compounds. 1992. U.S. Department of Health and Human Services, Public Health Service. September.

ATSDR. 1992b. Toxicological Profile for Thallium. 1992. U.S. Department of Health and Human Services, Public Health Service. July.

ATSDR. 1999. Toxicological Profile for Mercury. 1999. U.S. Department of Health and Human Services, Public Health Service. September.

ATSDR. 2004. Toxicological Profile for Copper. 2004. U.S. Department of Health and Human Services, Public Health Service. September.

ATSDR. 2005a. Toxicological Profile for Nickel. 2005. U.S. Department of Health and Human Services, Public Health Service. August.

ATSDR. 2005b. Toxicological Profile for Zinc. 2005. U.S. Department of Health and Human Services, Public Health Service. August.

ATSDR. 2007a. Toxicological Profile for Arsenic. 2007. U.S. Department of Health and Human Services, Public Health Service. August.

ATSDR. 2007b. Toxicological Profile for Lead. U.S. Department of Health and Human Services, Public Health Service. August.

ATSDR. 2008a. Draft Toxicological Profile for Cadmium. 2008. U.S. Department of Health and Human Services, Public Health Service. September.

ATSDR. 2008b. Draft Toxicological Profile for Chromium. 2008. U.S. Department of Health and Human Services, Public Health Service. September.

Avocet. 2003. Development of Freshwater Sediment Quality Values for Use in Washington State. Phase II Report: Development and Recommendation of SQVs for Freshwater Sediments in Washington State. Washington Department of Ecology, Publication Number 03-09-088. September.

Bechtel Jacobs. 1998. Empirical Models for the Uptake of Inorganic Chemicals from Soil by Plants. Appendix A. BJC/OR-133. Prepared for the U.S. Department of Energy Office of Environmental Management. BECHTEL JACOBS COMPANY LLC, Oak Ridge National Laboratory. September.

CDC (Centers for Disease Control and Prevention). 1991. Preventing Lead Poisoning in Young Children. A statement by the Centers for Disease Control. October.

CDC. 1997. Screening young children for lead poisoning. http://aepo-xdv-www.epo.cdc.gov/wonder/prevguid/p0000029/p0000029.htm

CDC. 2012. CDC Response to Advisory Committee on Childhood Lead Poisoning Prevention Recommendations in "*Low Level Lead Exposure Harms Children: A Renewed Call of Primary Prevention*". June 7, 2012.

Concha, G., B. Nermell, and M. Vahter. 1998a. Metabolism of Inorganic Arsenic in Children with Chronic High Arsenic Exposure in Northern Argentina. *Environ. Health Perspect.* 106: 355-359.

Concha, G. Vogler, D. Lezeano, B. Nermell, and M. Vahter. 1998b. Exposure to Inorganic Arsenic Metabolites During Early Human Development. *Toxicol. Sci.* 44: 185-190.

Ecology (Washington State Department of Ecology). 2003. Water Quality Standards for Surface Waters of the State of Washington. Washington Administrative Code (WAC) 173-201A. Washington State Department of Ecology, Olympia, WA. Amended July 1, 2003.

Ecology (Washington State Department of Ecology). 2007. Model Toxics Control Act. Washington Administrative Code (WAC) 173-340.

Ecology. 2012. CLARC database. https://fortress.wa.gov/ecy/clarc/Reporting/CLARCReporting.aspx, accessed in October 2012.

Finkel, A.M. 1990. Confronting Uncertainty in Risk Management. A guide for Decision Makers. Center for Risk Management, Washington, DC: Resources for the Future.

Hart Crowser 2011. Final Work Plan for Remedial Investigation/Feasibility Study Van Stone Mine, Stevens County, Washington. Prepared for Washington State Department of Ecology Toxics Cleanup Program, September 27, 2011.

Hart Crowser 2012. Draft Technical Memorandum: Results of Van Stone Mine Background Sampling and Natural Background Metal Concentrations. To Brendan Dowling, Ecology Eastern Division. March 16, 2012.

Hart Crowser 2013. Remedial Investigation (RI) for the Van Stone Mine Site. October. [This risk assessment is an appendix to the RI.]

Hattis, D. and D.E. Burmaster. 1994. Assessment of variability and uncertainty distributions for practical risk analysis. Risk Analysis 14:713-730.

Kurttio, P., H. Komulainen, E. Hakala, H. Kahelin, and J. Pekkanen. 1998. Urinary Excretion of Arsenic Species After Exposure to Arsenic Present in Drinking Water. *Arch. Environ. Contam. Toxicol.* 34: 297-305.

Maddaloni, M., N. Lolacono, W. Manton, C. Blum, J. Drexler, and J. Graziano. 1998. Bioavailability of soil borne lead in adults, by stable isotope dilution. *Environ Health Perspect.* 106:1-9.

National Academy of Sciences (NAS). 1993. Measuring lead exposure in infants, children, and other sensitive populations. Washington, DC: National Academy Press. ISBN 0-309-04927-X

NRC (National Research Council). 1999. Arsenic in drinking water. National Academy Press. Washington, D.C.

OSHA (Occupational Safety and Health Administration). 2012. Chemical Sampling Information. http://www.osha.gov/dts/chemicalsampling/toc/toc_chemsamp.html

USEPA (U.S. Environmental Protection Agency). 1992. Dermal Exposure Assessment: Principles and Applications. Interim Report. Office of Health and Environmental Assessment, January, 1992. EPA/600/8-91/011/B.

USEPA. 1996. Soil Screening Guidance. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC. EPA 540-R-96-018.

USEPA. 1998. OSWER DIRECTIVE: Clarification to the 1994 Revised Interim Soil Lead (Pb) Guidance for CERCLA Sites and RCRA Corrective Action Facilities. EPA/540/F-98/030PB98-963244. OSWER Directive #9200.4-27P. August 1998.

USEPA. 2003. Recommendations of the Technical Review Workgroup for Lead for an Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil. EPA-540-R-03-001. January 2003.

USEPA. 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Final EPA/540/R/99/005 OSWER 9285.7-02EP PB99-963312 July 2004.

USEPA. 2007. Dermal Exposure Assessment: A Summary of EPA Approaches. EPA/600/R-07/040F. September 2007.

USEPA. 2010. *ProUCL Version 4.1 User Guide (Draft). Statistical Software for Environmental Applications for Data Sets with and without Nondetect Observations.* EPA/600/R-07/041. Prepared by A. Singh, R. Maichle, and N. Armbya, Lockheed Martin Environmental Services, Las Vegas, NV. Prepared for Technology Support Center, U.S. Environmental Protection Agency, Region 4, Atlanta, GA. May.

USEPA. 2012a. Integrated Risk Information System (IRIS) Substance File Database. Online Database. http://www.epa.gov/iris/.

USEPA. 2012b. Regional Screening Levels, Region 9, May 2012. Online Screen Level Calculator, http://www.epa.gov/region9/superfund/prg/index.html, accessed on 10/29/2012.

USEPA. 2012c. National Primary Drinking Water Regulations. http://water.epa.gov/drink/contaminants/index.cfm, accessed on 10/29/2012.

Table 1. Initial Screening COPCs Identified at the Site

COPC	Surface Soil	Sediment	Surface Water	Groundwater
Antimony	x		x	x
Arsenic	x	x		x
Beryllium				
Cadmium	x	x		x
Chromium	x			x
Copper	x			
Lead	x			x
Mercury	x			
Nickel				x
Selenium				
Silver				
Thallium	x			
Zinc	x			

Table 2. Soil EPC and Risk Calculations - AOI1

Contaminant	Average Concentration (mg/kg)	Maximum Concentration (mg/kg)	EPC (mg/kg)	MTCA B soil cleanup levels, carcinogen, unrestricted, ingestion of soil (mg/kg)	Carcinogenic Risk	MTCA B soil cleanup level, noncarcinogen, unrestricted, ingestion of soil (mg/kg)	Noncarcinogenic Risk[a]
Antimony	3.1	20	6.0	NA	NA	32	0.2
Arsenic	8.0	45	10	6.70E-01	2.E-05	24	0.4
Cadmium	19	180	41	NA	NA	80	0.5
Chromium	7.0	35	7.3	NA	NA	240	0.03
Copper	46	640	120	NA	NA	3200	0.04
Mercury	0.27	2.8	0.60	NA	NA	24	0.02
Zinc	5000	37000	10000	NA	NA	24000	0.4
Total Risk					2.E-05		2

NA = not applicable

Results for noncarcinogenic and carcinogenic risks are reported in tables with one significant figure, as suggested by USEPA guidance (2004).

[a]Each noncarcinogenic risk value = HQ; the sum of all HQs = HI

Table 3. Soil EPC and Risk Calculations - AOI2

Contaminant	Average Concentration (mg/kg)	Maximum Concentration (mg/kg)	EPC (mg/kg)	MTCA B soil cleanup levels, carcinogen, unrestricted, ingestion of soil (mg/kg)	Carcinogenic Risk	MTCA B soil cleanup level, noncarcinogen, unrestricted, ingestion of soil (mg/kg)	Noncarcinogenic Risk[a]
Antimony	0.79	3.1	1.8	NA	NA	32	0.06
Arsenic	4.1	16	5.3	6.70E-01	8.E-06	24	0.2
Cadmium	3.6	15	7.4	NA	NA	80	0.09
Chromium	4.2	8.6	4.7	NA	NA	240	0.02
Copper	17	150	45	NA	NA	3200	0.01
Mercury	0.061	0.2	0.13	NA	NA	24	0.01
Zinc	1200	6500	4100	NA	NA	24000	0.2
Total Risk					8.E-06		0.6

NA = not applicable

Results for noncarcinogenic and carcinogenic risks are reported in tables with one significant figure, as suggested by USEPA guidance (2004).

[a]Each noncarcinogenic risk value = HQ; the sum of all HQs = HI

Table 4. Soil EPC and Risk Calculations - AOI3

Contaminant	Average Concentration (mg/kg)	Maximum Concentration (mg/kg)	EPC (mg/kg)	MTCA B soil cleanup levels, carcinogen, unrestricted, ingestion of soil (mg/kg)	Carcinogenic Risk	MTCA B soil cleanup level, noncarcinogen, unrestricted, ingestion of soil (mg/kg)	Noncarcinogenic Risk[a]
Antimony	0.87	5	1.1	NA	NA	32	0.04
Arsenic	4.4	14	5.2	6.70E-01	8.E-06	24	0.2
Cadmium	3.6	35	8.1	NA	NA	80	0.1
Chromium	5.4	9.3	5.4	NA	NA	240	0.02
Copper	27	180	47	NA	NA	3200	0.01
Mercury	0.043	0.21	0.069	NA	NA	24	0.003
Zinc	1100	11000	3000	NA	NA	24000	0.1
Total Risk					8.E-06		0.5

NA = not applicable

Results for noncarcinogenic and carcinogenic risks are reported in tables with one significant figure, as suggested by USEPA guidance (2004).

[a]Each noncarcinogenic risk value = HQ; the sum of all HQs = HI

Table 5. Soil EPC and Risk Calculations - AOI4

Contaminant	Average Concentration (mg/kg)	Maximum Concentration (mg/kg)	EPC (mg/kg)	MTCA B soil cleanup levels, carcinogen, unrestricted, ingestion of soil (mg/kg)	Carcinogenic Risk	MTCA B soil cleanup level, noncarcinogen, unrestricted, ingestion of soil (mg/kg)	Noncarcinogenic Risk[a]
Antimony	0.94	5	1.8	NA	NA	32	0.06
Arsenic	5.5	21	9.7	6.7E-01	1.E-05	24	0.4
Cadmium	4.2	25	9.4	NA	NA	80	0.1
Chromium	3.8	10	4.6	NA	NA	240	0.02
Copper	18	81	31	NA	NA	3200	0.0
Mercury	0.068	0.45	0.16	NA	NA	24	0.007
Zinc	1100	7700	2500	NA	NA	24000	0.1
Total Risk					1.E-05		0.7

NA = not applicable

Results for noncarcinogenic and carcinogenic risks are reported in tables with one significant figure, as suggested by USEPA guidance (2004).

[a]Each noncarcinogenic risk value = HQ; the sum of all HQs = HI

Table 6. Sediment EPC and Risk Calculations - AOI5

Contaminant	Average Concentration (mg/kg)	Maximum Concentration (mg/kg)	EPC (mg/kg)	MTCA B soil cleanup levels, carcinogen, unrestricted, ingestion of soil (mg/kg)	Carcinogenic Risk	MTCA B soil cleanup level, noncarcinogen, unrestricted, ingestion of soil (mg/kg)	Noncarcinogenic Risk[a]
Arsenic	2.0	7.8	2.6	0.67	4.E-06	24	0.1
Cadmium	1.1	4.5	2.1	NA	NA	25	0.08
Total					4.E-06		0.2

NA = not applicable

Results for noncarcinogenic and carcinogenic risks are reported in tables with one significant figure, as suggested by USEPA guidance (2004).

[a]Each noncarcinogenic risk value = HQ; the sum of all HQs = HI

Table 7. Groundwater EPC and Risk Calculations - AOI2

Contaminant	EPC (mg/L)	MTCA B groundwater cleanup levels, carcinogen, unrestricted (mg/L)	Carcinogenic Risk	MTCA B groundwater cleanup levels, noncarcinogen, unrestricted (mg/L)	Noncarcinogenic Risk[a]
Antimony	ND	NA	-	0.0064	-
Arsenic	ND	5.8E-05	-	0.0048	-
Cadmium	0.0014	NA	NA	0.016	0.09
Chromium	0.016	NA	NA	0.048	0.3
Nickel	0.014	NA	NA	0.32	0.04
Total			NA		0.5

NA = not applicable

Results for noncarcinogenic and carcinogenic risks are reported in tables with one significant figure, as suggested by USEPA guidance (2004).

[a]Each noncarcinogenic risk value = HQ; the sum of all HQs = HI

Table 8. Groundwater EPC and Risk Calculations - AOI3

Contaminant	EPC (mg/L)	MTCA B groundwater cleanup levels, carcinogen, unrestricted (mg/L)	Carcinogenic Risk	MTCA B groundwater cleanup levels, noncarcinogen, unrestricted (mg/L)	Noncarcinogenic Risk[a]
Antimony	0.028	NA	NA	0.0064	4
Arsenic	0.015	5.8E-05	3.E-04	0.0048	3
Cadmium	0.0095	NA	NA	0.016	0.6
Chromium	0.47	NA	NA	0.048	10
Nickel	0.31	NA	NA	0.32	1
Total			3.E-04		20

NA = not applicable

Results for noncarcinogenic and carcinogenic risks are reported in tables with one significant figure, as suggested by USEPA guidance (2004).

[a]Each noncarcinogenic risk value = HQ; the sum of all HQs = HI

Table 9. AOI1 Soil Cleanup Levels for Groundwater Protection

Contaminant	Average Concentration (mg/kg)	Maximum Concentration (mg/kg)	EPC (mg/kg)	MTCA Method B Soil Cleanup Level (for groundwater protection)	Above MTCA B Cleanup Levels
Antimony	3.1	20	6.0	5.42	Y
Arsenic	8.0	45	10	5.84	Y
Cadmium	19	180	41	0.69	Y
Chromium	7.0	35	7.3	19.2	N
Copper	46	640	120	577	N
Lead	2400	26000	7300	-	N
Mercury	0.27	2.8	0.60	2.09	N
Zinc	5000	37000	10000	6220	Y

Table 10. AOI2 Soil Cleanup Levels for Groundwater Protection

Contaminant	Average Concentration (mg/kg)	Maximum Concentration (mg/kg)	EPC (mg/kg)	MTCA Method B Soil Cleanup Level (for groundwater protection)	Above MTCA B Cleanup Levels
Antimony	0.79	3.1	1.8	5.42	N
Arsenic	4.1	16	5.3	5.84	N
Cadmium	3.6	15	7.4	0.69	Y
Chromium	4.2	8.6	4.7	19.2	N
Copper	17	150	45	577	N
Lead	150	1200	350	-	N
Mercury	0.061	0.2	0.13	2.09	N
Zinc	1200	6500	4100	6220	N

Table 11. AOI3 Soil Cleanup Levels for Groundwater Protection

Contaminant	Average Concentration (mg/kg)	Maximum Concentration (mg/kg)	EPC (mg/kg)	MTCA Method B Soil Cleanup Level (for groundwater protection)	Above MTCA B Cleanup Levels
Antimony	0.87	5	1.1	5.42	N
Arsenic	4.4	14	5.2	5.84	N
Cadmium	3.6	35	8.1	0.69	Y
Chromium	5.4	9.3	5.4	19.2	N
Copper	27	180	47	577	N
Lead	390	9500	1400	-	N
Mercury	0.043	0.21	0.069	2.09	N
Zinc	1100	11000	3000	6220	N

Table 12. AOI4 Soil Cleanup Levels for Groundwater Protection

Contaminant	Average Concentration (mg/kg)	Maximum Concentration (mg/kg)	EPC (mg/kg)	MTCA Method B Soil Cleanup Level (for groundwater protection)	Above MTCA B Cleanup Levels
Antimony	0.94	5	1.8	5.42	N
Arsenic	5.5	21	9.7	5.84	Y
Cadmium	4.2	25	9.4	0.69	Y
Chromium	3.8	10	4.6	19.2	N
Copper	18	81	31	577	N
Lead	180	1000	340	-	N
Mercury	0.068	0.45	0.16	2.09	N
Zinc	1100	7700	2500	6220	N

Table 13. AOI5 Soil Cleanup Levels for Groundwater Protection

Contaminant	Average Concentration (mg/kg)	Maximum Concentration (mg/kg)	EPC (mg/kg)	MTCA Method B Soil Cleanup Level (for groundwater protection)	Above MTCA B Cleanup Levels
Arsenic	2.0	7.8	2.6	5.84	N
Cadmium	1.1	4.5	2.1	0.69	Y

Table 14. Surface Water EPC and Risk Calculations - Fish Ingestion

Contaminant	AOI1 EPC (mg/L)	AOI2 EPC (mg/L)	AOI5 Average Concentration (mg/L)	AOI5 Maximum Concentration (mg/L)	AOI5 EPC (mg/L)	MTCA B surface water cleanup levels, carcinogen, unrestricted, ingestion of fish (mg/L)	AOI5 Carcinogenic Risk	MTCA B surface water cleanup levels, noncarcinogen, unrestricted, ingestion of fish (mg/L)	AOI5 Noncarcinogenic Risk
Antimony	ND	ND	0.0036	0.013	0.0066	NA	NA	1	0.007

ND = all results below detection limits

NA = not applicable

Results for noncarcinogenic and carcinogenic risks are reported in tables with one significant figure, as suggested by USEPA guidance (2004).

Table 15. Surface Water EPC and Risk Calculations - Surface Water as Drinking Water

Contaminant	AOI1 EPC (mg/L)	AOI2 EPC (mg/L)	AOI5 Average Concentration (mg/L)	AOI5 Maximum Concentration (mg/L)	AOI5 EPC (mg/L)	MTCA B surface water cleanup levels, carcinogen, unrestricted, ingestion of fish (mg/L)	AOI5 Carcinogenic Risk	MTCA B ground water cleanup levels, noncarcinogen, unrestricted, ingestion of groundwater (mg/L)	AOI5 Noncarcinogenic Risk
Antimony	ND	ND	0.0036	0.013	0.0066	NA	NA	0.0064	1

ND = all results below detection limits

NA = not applicable

Results for noncarcinogenic and carcinogenic risks are reported in tables with one significant figure, as suggested by USEPA guidance (2004).

Table 16. Lead EPC

Medium	Lead	Average Concentration	Maximum Concentration	EPC	Comparison Criterion	Criterion Source
Soil (mg/kg)	AOI1	2400	26000	7300	250	MTCA A
	AOI2	150	1200	350		
	AOI3	400	9500	1400		
	AOI4	180	1000	400		
Groundwater (mg/L)	AOI2	0.0035	0.0042	0.0042	0.015	State MCL
	AOI3	0.053	0.22	0.22		

Table 17. Carcinogenic and Noncarcinogenic Risk

AOI	Risk	Soil/Sediment ingestion	Surface Water (fish ingestion)	Surface Water (ingestion)	Groundwater (ingestion)	Total	Above Target Risk
1	Carcinogenic	2.E-05	NA	NA	ND	2.E-05	Y
	Noncarcinogenic	2	NA	NA	ND	2	Y
2	Carcinogenic	8.E-06	NA	NA	NA	8.E-06	N
	Noncarcinogenic	0.6	NA	NA	0.5	1	N
3	Carcinogenic	8.E-06	ND	ND	3.E-04	3.E-04	Y
	Noncarcinogenic	0.5	ND	ND	19	20	Y
4	Carcinogenic	1.E-05	ND	ND	ND	1.E-05	N
	Noncarcinogenic	0.7	ND	ND	ND	0.7	N
5	Carcinogenic	4.E-06	NA	NA	ND	4.E-06	N
	Noncarcinogenic	0.2	0.007	1.0	ND	1	N

NA = not applicable

ND = no available data

Results for noncarcinogenic and carcinogenic risks are reported in tables with one significant figure, as suggested by USEPA guidance (2004).

Table 18. Adjusted Soil EPC and Risk Calculations - AOI1

Contaminant	EPC (mg/kg)	MTCA B soil cleanup levels, carcinogen, unrestricted, ingestion of soil (mg/kg)	Carcinogenic Risk	MTCA B soil cleanup level, noncarcinogen, unrestricted, ingestion of soil (mg/kg)	Noncarcinogenic Risk[a]
Antimony	6.0	NA	NA	32	0.2
Arsenic	5.04	6.70E-01	8.E-06	24	0.2
Cadmium	1.5	NA	NA	80	0.02
Chromium	7.3	NA	NA	240	0.03
Copper	120	NA	NA	3200	0.04
Mercury	0.60	NA	NA	24	0.02
Zinc	10000	NA	NA	24000	0.4
Total Risk			8.E-06		0.9

NA = not applicable

Results for noncarcinogenic and carcinogenic risks are reported in tables with one significant figure, as suggested by USEPA guidance (2004).

[a]Each noncarcinogenic risk value = HQ; the sum of all HQs = HI

Highlighted cell indicates adjusted EPC

Table 19. Adjusted Groundwater Cleanup Levels - AOI3

Contaminant	EPC (mg/L)	MTCA B groundwater cleanup levels, carcinogen, unrestricted (mg/L)	Carcinogenic Risk	MTCA B groundwater cleanup levels, noncarcinogen, unrestricted (mg/L)	Noncarcinogenic Risk[a]
Antimony	0.0004	NA	NA	0.0064	0.06
Arsenic	0.0038	0.0000583	7.E-05	0.0048	0.8
Cadmium	0.0014	NA	NA	0.016	0.09
Chromium	0.0014	NA	NA	0.048	0.03
Nickel	0.002	NA	NA	0.32	0.006
Total			7.E-05		1

NA = not applicable

Results for noncarcinogenic and carcinogenic risks are reported in tables with one significant figure, as suggested by USEPA guidance (2004).

[a]Each noncarcinogenic risk value = HQ; the sum of all HQs = HI

Highlighted cell indicates adjusted EPC

Table 20. Human Health COCs Identified at the Site

COPC	AOI1	AOI2	AOI3	AOI4	AOI5
Antimony	GW		x		
Arsenic	x	x	x	x	
Beryllium					
Cadmium	x	GW	x	GW	
Chromium			x		
Copper					
Lead	x	x	x	x	
Mercury					
Nickel			x		
Selenium					
Silver					
Thallium					
Zinc	x				

GW = Identification as a COC based on protection of groundwater pathway only

Figure 1. HHRA Conceptual Site Model

Impacted Medium	Exposure Route	Unrestricted: Adult & Child Resident	Unrestricted: Quantified Pathways	Trespasser
Soil	Inhalation	●		●
	Dermal Contact	◐		◐
	Ingestion	●	✓	●
Groundwater	Inhalation	◐		◐
	Dermal Contact	◐		◐
	Ingestion	●	✓	◐
Surface Water	Inhalation	◐		◐
	Dermal Contact	◐		◐
	Ingestion	●	✓	●
Sediment	Inhalation	◐		◐
	Dermal Contact	◐		◐
	Ingestion	●	✓	●
Biota[a]	Ingestion	●	✓[b]	●

Legend

- ● Complete and potentially siginificant pathway
- ◐ Incomplete and/or insignificant pathway
- ✓ Exposure pathway and medium quantified in the HHRA

a) Biota (e.g., homegrown vegetables, insects, fish, plants) may by considered a secondary source that accumulate contaminants of concern from impacted soil, sediment, surface water, and groundwater and may be consumed by humans or ecological receptors. Independent of being a secondary source, terrestrial and aquatic biota are also receptors.

b) Fish ingestion is the only medium and route quantified in the HHRA, based on the highest beneficial use of a surface water body, determined in accordance with WAC 173-201A (Ecology 2003).

Appendix A - Human Health Risk Assessment Initial Screening

A1 HHRA Results of Soil Screening

A2 HHRA Results of Sediment Screening

A3 HHRA Results of Surface Water Screening

A4 HHRA Results of Groundwater Screening

A5 Exposure Point Concentrations – Soil and Sediment

A6 Exposure Point Concentrations – Surface Water

A7 Exposure Point Concentrations – Groundwater

A8 Toxicity Assessment

A9 Exposure Factors

Table A1. HHRA Results of Screening Soil

Contaminant	Detection Frequency	More than 5% Detected	Background Concentration (mg/kg)	Maximum Detected Concentration (mg/kg)	Qualifier	Above Background	MTCA Method B Soil Cleanup Level (a)	COPC
Antimony	99%	Y	0.857	20		Y	5.42	Y
Arsenic	100%	Y	5.04	45		Y	0.67	Y
Beryllium	64%	Y	0.719	1.9		Y	63	N
Cadmium	100%	Y	1.596	180	J	Y	0.69	Y
Chromium	81%	Y	15.84	35		Y	19.2	Y
Copper	83%	Y	12.65	640	J	Y	577	Y
Lead	100%	Y	44.87	26000	J	Y	250 (b)	Y
Mercury	95%	Y	0.134	2.8	J	Y	2 (b)	Y
Nickel	85%	Y	13.05	45		Y	130	N
Selenium	80%	Y	1.645	1.2	J	N	5.2	N
Silver	98%	Y	0.122	3.6		Y	13.6	N
Thallium	43%	Y	0.203	1.3	J	Y	--	Y
Zinc	96%	Y	206	37000		Y	6220	Y

(a) The lowest cleanup level of soil ingestion or protection of groundwater pathway

(b) No MTCA Method B soil cleanup levels available. MTCA Method A soil cleanup level is used.

J = qualified as an estimate

Table A2. HHRA Results of Screening Sediment

Contaminant	Detection Frequency	More than 5% Detected	Background Concentration (mg/kg)	Maximum Detected Concentration (mg/kg)	Qualifier	Above Background	MTCA Method B Soil Cleanup Level (a)	COPC
Antimony	100%	Y	0.587	1		Y	5.42	N
Arsenic	100%	Y	6.662	7.8		Y	0.67	Y
Beryllium	86%	Y	0.741	0.44		N	63	N
Cadmium	95%	Y	0.427	4.5		Y	0.69	Y
Chromium	100%	Y	14.33	7.3		N	19.2	N
Copper	95%	Y	12.2	8		N	577	N
Lead	100%	Y	22.8	110		Y	250 (b)	N
Mercury	95%	Y	0.0284	0.13		Y	2 (b)	N
Nickel	100%	Y	10.95	6.4		N	130	N
Selenium	73%	Y	2.029	0.82	T	N	5.2	N
Silver	95%	Y	0.088	0.068	T	N	13.6	N
Thallium	18%	Y	0.406	0.26	U	N	--	N
Zinc	100%	Y	120.4	970		Y	6220	N

(a) The lowest cleanup level of soil ingestion or protection of groundwater pathway

(b) No MTCA Method B soil cleanup levels available. MTCA Method A soil cleanup level is used.

T = below method reporting limit

Table A3. HHRA Results of Screening Surface Water

Contaminant	Detection Frequency	More than 5% Detected	Lowest Potential Human Health Surface Water ARAR (a) (mg/L)	Maximum Detected Concentration (mg/L)	Qualifier	COPC
Antimony	58%	Y	5.6E-03	0.013	J	Y
Arsenic	0%	N	1.8E-05	0.0038	U	N
Beryllium	0%	N	4.0E-03	0.00051	U	N
Cadmium	23%	Y	5.0E-03	0.0014	T	N
Chromium	4%	N	4.8E-02	0.0015	T	N
Copper	19%	Y	6.4E-01	0.01		N
Lead	27%	Y	1.5E-02	0.0094		N
Dissolved Mercury (ug/L)	30%	Y	2.0E-03	0.000916		N
Total Mercury (ug/L)	86%	Y	2.0E-03	0.00115		N
Nickel	4%	N	1.0E-01	0.0039	T	N
Selenium	0%	N	5.0E-02	0.0036	U	N
Silver	0%	N	8.0E-02	0.00015	U	N
Thallium	0%	N	2.4E-04	0.0014	U	N
Zinc	85%	Y	7.4E+00	0.72		N

(a) The lowest value of MTCA Method B cleanup levels for protection of human health based on fish ingestion and drinking water ingestion (groundwater pathway), National recommended water quality criteria for consumption of organisms and consumption of water and organisms, National Toxics Rule Criteria for consumtion of organisms and consumption of water and organisms, State and Federal drinking water MCLs, and Federal MCLGs.

U= below detection limit

T = below method reporting limit

J = qualified as an estimate

Table A4. HHRA Results of Screening Groundwater

Contaminant	Detection Frequency	More than 5% Detected	Lowest Potential Groundwater ARAR (a) (mg/L)	Maximum Detected Concentration (mg/L)	Qualifier	COPC
Antimony	57%	Y	0.006	0.028		Y
Arsenic	14%	Y	0.000058	0.015		Y
Beryllium	43%	Y	0.004	0.0018	T	N
Cadmium	100%	Y	0.005	0.0095		Y
Chromium	100%	Y	0.048	0.47		Y
Copper	100%	Y	0.64	0.048		N
Lead	100%	Y	0.015	0.22		Y
Mercury - Total	43%	Y	0.002	0.00056		N
Nickel	86%	Y	0.1	0.31		Y
Selenium	0%	N	0.05	0.0036	U	N
Silver	71%	Y	0.08	0.042		N
Thallium	14%	Y	0.0005	0.0014	T	N
Zinc	100%	Y	4.8	0.39		N

(a) The lowest value of MTCA Method A and B cleanup levels, State and Federal drinking water MCLs, and Federal MCLGs.

U= below detection limit

T = below method reporting limit

Table A5. Exposure Point Concentrations - Soil and Sediment

COPC	AOI1 95% UCL (mg/kg)	AOI2 95% UCL (mg/kg)	AOI3 95% UCL (mg/kg)	AOI4 95% UCL (mg/kg)	AOI5 95% UCL (mg/kg)
Antimony	6.0	1.8 (a)	1.1	1.8	NA
Arsenic	10	5.3	5.2	9.7	2.6
Cadmium	41	7.4	8.1	9.4	2.1
Chromium	7.3	4.7 (c)	5.4 (c)	4.6	NA
Copper	120 (a)	45 (a)	47	31	NA
Lead	7300	350	1400	400	NA
Mercury	0.60	0.13 (a)	0.069	0.16 (a)	NA
Zinc	10000	4100 (b)	3000 (a)	2500	NA

(a) ProUCL recommended the use of 97.5% UCL

(b) ProUCL recommended the use of 99% UCL.

(c) ProUCL recommended two 95% UCLs. The higher of the two was selected.

NA = not applicable; contaminant not identified as a COPC during sediment screening.

Table A6. Exposure Point Concentrations - Surface Water

COPC	AOI1 95% UCL (mg/L)	AOI2 95% UCL (mg/L)	AOI3 95% UCL (mg/L)	AOI4 95% UCL (mg/L)	AOI5 95% UCL (mg/L)
Antimony	ND	ND	NA	NA	0.0066

ND = All sample results were below the detection limit.

NA = No data available in this exposure area

Table A7. Exposure Point Concentrations - Groundwater

	AOI1	AOI2			AOI3		AOI4	AOI5
COPC	EPC (mg/L)	Number of Detected Samples	EPC (mg/L) (a)	Qualifier	Number of Detected Samples	EPC (mg/L) (a)	EPC (mg/L)	EPC (mg/L)
Antimony	NA	0	ND		4	0.028	NA	NA
Arsenic	NA	0	ND		1	0.015	NA	NA
Cadmium	NA	2	0.0014 (b)	T	5	0.0095	NA	NA
Chromium	NA	2	0.016		5	0.47	NA	NA
Lead	NA	2	0.0042		5	0.22	NA	NA
Nickel	NA	2	0.014 (b)	T	4	0.31	NA	NA

NA = No data available in this exposure area

ND = All sample results were below the detection limit.

(a) = Maximum concentration was used as EPC because there are less than 6 detected results

(b) = Maximum concentration is less than the reporting limit.

Table A8. Toxicity Assessment

COPC	Oral Cancer Potency Factor mg/kg-day	Oral Reference Dose mg/kg-day
Antimony	NA	0.0004
Arsenic	1.5	0.0003
Cadmium in water	NA	0.0005
Cadmium in soil	NA	0.001
Chromium (a)	NA	0.003
Copper	NA	0.04 (b)
Lead	NA	NA
Mercury (c)	NA	0.0003
Nickel, soluble salts	NA	0.02
Thallium	NA	NA
Zinc	NA	0.3

NA = not available

(a) Chromium VI

(b) value is from CLARC database. All other values are from IRIS (USEPA 2012a).

(c) Mercuric chloride

Table A9. Exposure Factors

Pathway	Parameter	NonCarcinogen	Carcinogen
Soil Ingestion	RfD = Reference Dose	defined in WAC	-
	ABW = Average body weight over the exposure duration	16 kg	16 kg
	SIR = soil ingestion rate	200 mg/day	200 mg/day
	AB1= Gastrointestinal absorption fraction	1	1
	EF = exposure frequency	1	1
	HQ = Hazard Quotient	1	-
	AT = Averaging time	6 yrs	75 yrs
	ED = Exposure duration	6 yrs	6 yrs
	Risk = acceptable cancer risk level	-	1 x 10^-6
	CPF = carcinogenic potency factor	-	defined in WAC
Surface Water (fish ingestion)	RfD = Reference Dose	defined in WAC	
	ABW = Average body weight over the exposure duration	70 kg	70 kg
	BCF= bioconcentration factor	defined in WAC	defined in WAC
	FCR = fish consumption rate	54 g/day	54 g/day
	FDF = fish diet fraction	0.5	0.5
	HQ = Hazard Quotient	1	
	AT = Averaging time	30 yrs	75 yrs
	ED = Exposure duration	30 yrs	30 yrs
	Risk = acceptable cancer risk level		1 x 10^-6
	CPF = carcinogenic potency factor		defined in WAC
Groundwater Ingestion	RfD = Reference Dose	defined in WAC	
	ABW = Average body weight over the exposure duration	16 kg	70 kg
	BCF= bioconcentration factor	defined in WAC	defined in WAC
	DWIR = drinking water ingestion rate	1 L/day	2 L/day
	DWF = Drinking water fraction	1	1
	HQ = Hazard Quotient	1	
	AT = Averaging time	6 yrs	75 yrs
	ED = Exposure duration	6 yrs	30 yrs
	Risk = acceptable cancer risk level		1 x 10^-6
	CPF = carcinogenic potency factor		defined in WAC

Appendix B - Detailed Human Health Toxicity Assessment

B.1 Antimony

Antimony is a naturally occurring metal that is mined and used in alloys, textiles, and plastics. Antimony has been used to treat two parasitic diseases in medicine. When combined with oxygen, antimony forms antimony oxide. Small amounts of antimony are released by incinerators or coal burning plants. Antimony strongly attaches to small particles and ends up in the soil or sediment. Antimony often exists, at least in part, as a poorly soluble salt and may also occur in particles of inert or insoluble material. These factors all tend to reduce the bioavailability of antimony (ATSDR 1992a).

Exposure to antimony can occur through ingestion or inhalation. Antimony is poorly absorbed from the gastrointestinal tract. Absorbed antimony is principally excreted in the urine and feces. Acute exposure by ingestion is irritating to the gastrointestinal tract. Long term inhalation of antimony may affect the lungs (pneumoconiosis), and can cause heart problems, stomach pain, diarrhea, vomiting, and stomach ulcers. There is inconclusive evidence of a relationship between the inhalation of antimony trioxide and excess risk of lung cancer and reproductive disorders (ATSDR 1992a). Cancer evidence from studies in human populations is limited, and carcinogenicity studies in laboratory animals provide conflicting results; USEPA has not classified antimony for carcinogenicity (USEPA 2012a).

USEPA allows 0.006 parts per million (ppm) in drinking water (USEPA 2012c). The U.S. Occupational Safety and Health Administration (OSHA) has set an occupational exposure limit of 0.5 mg/m³ for an 8-hour workday, 40-hour workweek (OSHA 2012). The American Conference of Governmental Industrial Hygienists (ACGIH) and the National Institute for Occupational Safety and Health (NIOSH) make the same recommendations as OSHA (ATSDR 1992a).

There is no oral cancer potency factor for antimony. The oral RfD value from IRIS is 0.0004 mg/kg-day (USEPA 2012a). Consequently, antimony is only evaluated for noncancer health effects.

B.2 Arsenic

Arsenic occurs in soil and rock along with other minerals such as copper, lead, iron, and nickel. It is typically found in soil in the form of an insoluble sulfide. Naturally occurring arsenic concentrations in soil range from 1 to 40 mg/kg, with a mean concentration of approximately 5 mg/kg. Naturally occurring arsenic concentrations in groundwater average around 1 to 2 micrograms per liter (µg/L), except for some western states with geological features that have naturally elevated concentrations of arsenic. Concentrations in groundwater in these areas range from 5 to more than 500 µg/L. Arsenic was used as a wood preservative and in pesticide. It is also associated with mining and smelting ore (ATSDR 2007a).

Arsenic has been shown to be toxic to human populations in areas of the world where it is present at naturally elevated concentrations in groundwater, and to occupationally exposed workers in copper smelters and chemical plants. There is strong evidence that arsenic is carcinogenic in humans by both oral and inhalation routes (ATSDR 2007a, USEPA 2012a).

Inorganic arsenic (the form typically found in soil or water) is often in a form that is readily absorbed by either ingestion or inhalation. Following absorption, it is distributed throughout the body. Studies with laboratory animals suggest that the bioavailability of arsenic in soil may be lower than that of arsenic ingested in solution. The issue of arsenic bioavailability is especially important at mining, milling, and smelting sites because the arsenic at these sites often exists, at least in part, as a poorly soluble sulfide and may also occur in particles of inert or insoluble material. These factors all tend to reduce the bioavailability of arsenic (ATSDR 2007a).

Arsenic is partly metabolized in the liver by methylation (the metabolic addition of methyl groups to inorganic arsenic ions), converting inorganic arsenic into methyl- and dimethylarsenic compounds. Absorbed organic and inorganic arsenic compounds are principally excreted in the urine (ATSDR 2007a).

Several organic arsenicals have been found to accumulate in fish and shellfish. These derivatives (mainly arsenobetaine and arsenocholine, also referred to as "fish arsenic") have been studied by several researchers and have been found to be essentially nontoxic.

Arsenic at high levels of exposure is irritating to the gastrointestinal tract. Common symptoms in humans after acute high-dose ingestion of inorganic arsenic compounds are nausea, vomiting, and diarrhea. Signs of peripheral neuropathy have been noted in individuals who have ingested inorganic arsenic. The neuropathy is detected as numbness in the hands and feet, progressing to a painful "pins and needles" sensation. Death from acute arsenic ingestion is usually attributed to cardiopulmonary collapse (ATSDR 2007a).

Evidence of reproductive or developmental toxicity in humans is limited. Chronic exposure to arsenic in drinking water has been associated with an increased incidence of miscarriages, stillbirths, preterm births, and infants with low birth weights. Animal studies have shown changes to reproductive organs in both sexes (ATSDR 2007a).

The distinguishing adverse effects associated with chronic ingestion of arsenic are skin lesions (hyperkeratoses and hyperpigmentation) and skin cancer. Other adverse effects due to ingestion exposure include cancer of the internal organs (prostate, liver, bladder, and kidney) and a vascular disease known as "blackfoot disease" (Blackfoot disease has been observed in areas where there are naturally elevated arsenic concentrations in drinking water). Occupational exposure (principally copper smelter workers) has been associated with an increased incidence of lung cancer (ATSDR 2007a). USEPA has given arsenic a carcinogenicity weight-of-evidence classification of Group A (human carcinogen) based on sufficient evidence of cancer mortality from both ingestion and inhalation exposures in human populations(USEPA 2012a). The International Agency for Research on Cancer (IARC) classifies arsenic as a proven human carcinogen (ATSDR 2007a).

Some populations may be sensitive to arsenic. Individuals with impaired liver function or poor nutritional status may not detoxify arsenic efficiently and may be at greater risk of adverse

effects from arsenic exposure. Studies have shown that children do not biomethylate arsenic as well as adults, which may put them at a higher risk for noncancer effects and to some extent cancer effects from the higher net fraction of inorganic arsenic (Kurttio et al. 1998; Concha et al. 1998a). Pregnant women have also been identified as a sensitive population. It has been shown that arsenic crosses the placental barrier (Concha et al. 1998b, NRC 1999), and in pregnant women exposed to arsenic, blood arsenic levels in the newborns are almost as high as the level in cord blood.

USEPA published a final rule lowering the Maximum Contaminant Level (MCL) from 50 µg/L to 10 µg/L (USEPA 2012c). OSHA has established a permissible exposure limit, 8-hour time-weighted average, of 10 µg/m3 for airborne arsenic in various workplaces that use inorganic arsenic (OSHA 2012). The cancer potency factor for arsenic is 1.5 mg/kg-day. The oral reference dose value is 0.0003 mg/kg-day (USEPA 2012c). Both cancer and noncancer effects are evaluated.

B.3 Cadmium

Cadmium is obtained mainly as a by-product during the processing of zinc-bearing ores and also from the refining of lead and copper from sulfide ores. Cadmium is used primarily for the production of nickel-cadmium batteries, in metal plating, and for the production of pigments, plastics, synthetics, and metallic alloys. Cadmium has been shown to be toxic to human populations from occupational inhalation exposure and accidental ingestion of cadmium-contaminated food. Inhalation of cadmium dust in certain occupational settings may be associated with an increased incidence of lung cancer. Ingestion of elevated levels of cadmium has resulted in toxicity to the kidney and skeletal system and may be associated with an elevated incidence of hypertension and cardiovascular disease (ATSDR 2008a).

Cadmium is poorly absorbed from the lung, gastrointestinal tract, and skin. Individuals with dietary deficiencies of iron, calcium, or protein exhibit higher absorption of ingested cadmium. The issue of cadmium bioavailability is especially important at mining, milling, and smelting sites because the cadmium at these sites often exists, at least in part, as a poorly soluble sulfide and may also occur in particles of inert or insoluble material. These factors all tend to reduce the bioavailability of cadmium in soil. Cadmium in the body binds readily to certain sulfur-containing proteins, such as metallothionein. Binding to metallothionein is thought to reduce the toxicity of cadmium. Following ingestion, fecal excretion is high due to poor gastrointestinal absorption. Most cadmium that has been absorbed, however, is excreted very slowly, with fecal and urinary excretion being about equal (ATSDR 2008a).

Ingested cadmium is not known to be carcinogenic in humans. Studies in laboratory animals generally do not indicate that cadmium is carcinogenic by ingestion. USEPA has classified cadmium as a probable human carcinogen by inhalation (Group B1) based on limited evidence in humans and sufficient evidence in laboratory animals (USEPA 2012a).

Populations potentially sensitive to cadmium have not been studied systematically; however, it is possible to infer potential sensitivities based on the available data. Individuals with poor nutritional status, particularly in terms of iron and calcium, may absorb more cadmium from the

gastrointestinal tract. Individuals with preexisting kidney damage may experience kidney toxicity at cadmium doses lower than the dose that would be toxic for normal individuals.

The MCL for cadmium in drinking water is 0.005 mg/L (USEPA 2012c). There is no cancer potency factor for cadmium. Separate RfD values exist for water ingestion (0.0005 mg/kg-day) and for food (soil) ingestion (0.001 mg/kg-day, USEPA 2012a). Consequently, cadmium is only evaluated for noncancer health effects.

B.4 Chromium

Chromium is a naturally occurring element in rocks, animals, plants, and soils that is naturally and anthropogenically released and deposited in the soil and water, where it can change from one form to another. Chromium is used in manufacturing and other industries, including tanneries, in chrome pigments, in stainless steel, and in wood treatments. Chromium has three main states: chromium(0), chromium(III), and chromium(VI). Chromium VI is more toxic than other forms of chromium. Chromium III is an essential nutrient. However, its biological target is unknown, and its classification as an essential nutrient is controversial. Documented problems from chromium III deficiency range from no ill effects to hyperglycemia, weight gain, and fertility problems (ATSDR 2008b).

Less than 10 percent of ingested chromium is absorbed in the gastrointestinal tract. Soluble forms of chromium have greater rates of absorption than insoluble forms, and chromium VI has higher rates of absorption compared to chromium III. Absorption is dependent on nutritional status. Chromium VI is reduced in the stomach to chromium III, producing reactive intermediaries. Chromium can also penetrate skin (ATSDR 2008b).

Absorbed chromium goes to most tissues within the body, specifically the kidney and liver, and is retained long-term in bone. It can be transferred to a fetus through the placenta and to infants via breast milk. Chromium is predominantly excreted through the urine, and also through hair and nail formation (ATSDR 2008b).

The general population may be exposed to chromium through ingestion of food and water, and to a lesser extent, inhalation and dermal contact. Occupational exposure to chromium is mainly via inhalation. Chromium VI is much more toxic than chromium III. Its effects depend on the route of exposure. Inhalation of chromium VI can cause respiratory problems, including irritation and reduced pulmonary function. Ingestion of chromium VI affects the gastrointestinal system, and includes irritation of the stomach and intestines, ulceration, and lesions. Evidence suggests that additional health impacts from chromium VI exposure are hematological (anemia), reproductive (affecting the male reproductive organs), and developmental, as well as dermal and ocular irritation when there is direct contact. Individuals can develop an allergic sensitization to chromium VI, which may cause asthma, dermatitis, ulcers, sores, and tonsillitis, depending on the route of exposure. Chromium III exposure may cause respiratory effects when inhaled and immunological effects with dermal contact. There is some evidence of adverse effects on developing and adult reproductive systems (ATSDR 2008b, USEPA 2012a).

Susceptible populations include individuals who exhibit different or enhanced responses to chromium exposure, or those who have reduced detoxification or excretion. People with pre-

existing respiratory, gastrointestinal, immunicological, or hematological conditions may also be susceptible. Smokers may have an increased risk of lung cancer when exposed to chromium via inhalation (ATSDR 2008b).

There is strong evidence of carcinogenicity for chromium VI, with documentation of increased lung cancers in workers who have been exposed, and stomach tumors in people drinking water with higher levels of chromium VI. As noted with health effects, the carcinogenic effect depends on the route of exposure (lung cancer via inhalation and stomach cancers via ingestion). USEPA has classified chromium VI in Group A (known human carcinogen by the inhalation route of exposure). Trivalent chromium is classified in Group D (not classified as a human carcinogen due to inadequate data) (USEPA 2012a).

USEPA has determined that exposure to chromium in drinking water at concentrations of 1 mg/L for 1 day or 10 days is not expected to cause any adverse effects in a child (USEPA 2012c). The U.S. Food and Drug Administration has determined that the chromium concentration in bottled drinking water should not exceed 0.1 mg/L. OSHA set a legal limit for chromium(VI) of 0.0005 mg/m3, for chromium(III) of 0.5 mg/m3, and for chromium(0) of 1.0 mg/m3 averaged over an 8-hour work day (ATSDR 2008b).

There is no cancer potency factor for total chromium. The oral RfD value for Chromium VI is 0.003 mg/kg-day (USEPA 2012a). Consequently, total chromium is only evaluated for noncancer health effects.

B.5 Copper

Copper is a naturally occurring metal in the earth's crust and is an essential element at low levels for all living organisms, including humans. Its most common form is copper sulfate. Copper is used in many industries, most notably for use in the penny, electric wiring, pipes, metal alloy mixes, certain agricultural products such as fungicides, and wood preservatives.

In the environment, copper strongly attaches to particles in soil or suspended in water, but it can also dissolve and enter into groundwater. Copper exposure mainly occurs via inhalation or ingestion, and copper is readily absorbed in the small intestine and stomach. Excess copper is removed from the body via homeostatic mechanisms, which synthesize and allow the binding of copper to metallothionein in the intestine and the liver. Copper bound to metallothionein is eventually excreted through feces (ATSDR 2004).

Acute exposure to copper can cause liver and kidney damage, anemia, immunotoxicity, and developmental effects. The most common acute effects are gastrointestinal distress from ingestion, which is typically not persistent, and respiratory tract irritation from inhalation, usually in an occupational setting (ATSDR 2004).

Carcinogenicity of copper is not well studied. Several studies found higher rates of lung and stomach cancers in occupational exposures, but all of them have confounding factors. USEPA classified copper in Group D (not classified as a human carcinogen, USEPA 2012a).

Sensitive populations include children with genetic defects that impair copper homeostatic mechanisms, such as Wilson's disease, Indian childhood cirrhosis, and idiopathic copper toxicosis. Children with this disease or these syndromes may exhibit hepatic damage due to their

inability to effectively metabolize copper, potentially even with normal dietary intake (ATSDR 2004).

USEPA has determined that drinking water should not contain more than 1.3 mg /L copper (USEPA 2012a). OSHA has set a limit of 0.1 mg/m^3 for copper fumes and 1.0 mg/m^3 for copper dusts for an 8-hour work shift and 40-hour workweek (OSHA 2012). The Food and Nutrition Board of the Institute of Medicine has developed recommended dietary allowances of 340 µg of copper per day for children aged 1–3 years, 440 µg/day for children aged 4–8 years, 700 µg/day for children aged 9–13 years, 890 µg/day for children aged 14–18 years, and 900 µg/day for adults (ATSDR 2004).

There is no oral slope factor for copper. The oral RfD of 0.04 mg/kg-day, identified in CLARC (Ecology 2012), allows its evaluation for noncancer effects.

B.6 Lead

Lead is a soft, bluish-gray metal. Lead acetate and lead nitrate are soluble in water; lead chloride is slightly soluble; and lead sulfide, lead phosphate, and lead oxides are not soluble in water. Some primary uses of lead in the United States are in lead-acid batteries, ammunition, bearing metals, brass, bronze, cable covering, extruded products, sheet lead, solder, ceramics, type metal, ballast or weights, tubes or containers, oxides, and gasoline additives.

Substantial quantities of both human and animal data are available regarding the toxicity of lead. This toxicity profile relies primarily on human data. Adverse effects of lead in humans are most often related to the blood lead level as an indicator of internal lead dose. Whenever possible, this text relates adverse effects to blood lead levels rather than to external exposure. CDC has based policy on primary and secondary childhood lead prevention activities on the association of certain adverse health effects with different blood lead levels.

Lead absorption is influenced by the route of exposure, the exposure medium, speciation and physiochemical characteristics of lead, and the age and physiological state of the exposed individual. Approximately 30 to 50 percent of airborne particulate lead is absorbed. Children 2 weeks to 8 years of age absorb about 40 to 50 percent of ingested lead. A study using Bunker Hill Superfund Site soils found that nonfasted adults absorbed 2.5 percent of lead ingested in soil and fasted adults absorbed 26.2 percent of lead ingested in soil (Maddaloni et al. 1998). The amount of lead absorbed from the skin in humans is unknown.

Lead is absorbed into blood, where about 99 percent is located in red blood cells. Lead in blood rapidly exchanges with other soft tissues. Bone contains about 94 percent and 73 percent of the total lead body burden in adults and children, respectively. The average half-life for lead is 28 to 36 days in blood, about 40 days in soft tissues, and about 27 years in bone. Lead in bone can be mobilized into maternal blood during pregnancy and lactation. Lead in maternal blood is efficiently transported to the fetus, and breast milk can be a significant source of lead for breast-feeding infants (ATSDR 2007b).

Lead in the gastrointestinal tract that is not absorbed is eliminated in the feces. Absorbed lead that is not retained is eliminated in the urine or excreted in the feces following biliary secretion into the gastrointestinal tract.

Death from encephalopathy has been reported in children and adults with very high blood lead levels (e.g., 80–100 µg/dL). There is conflicting evidence in occupational mortality studies of

chronic lead exposure. IQ decrements, fine-motor dysfunction, altered behavior, peripheral neuropathy, and reduced motor nerve conduction have been reported in children. A threshold below which lead does not affect IQ in children has not been identified. Decreased hearing thresholds and alterations in the electrical activity of the brain have also been observed in children exposed to lead. Lead can induce neurotoxicity in adults, including encephalopathy, overt neurological signs, decreased scores on neurobehavioral tests, and decreased motor nerve conduction (ATSDR 2007b).

Lead interferes with heme synthesis. Anemia can result from decreased hemoglobin production and increased red blood cell destruction. Lead-induced inhibition of heme synthesis can interfere with the conversion of vitamin D to its hormonal form. There is no apparent threshold for indicators of decreased heme synthesis.

Acute, generally reversible, nephropathy can occur during the early stages of high exposure to lead. Chronic (irreversible) nephropathy can also occur. Acute exposures to high levels of lead can produce cardiac lesions, electrocardiographic abnormalities, and hemolytic anemia in children and adults. There is conflicting evidence regarding the potential effects of blood lead levels on blood pressure in adults. Colic is a relatively late symptom of severe or clinical lead poisoning generally observed at blood lead levels greater than 50 µg/dL (ATSDR 2007b).

Women with occupational exposures to lead during pregnancy have an increased rate of miscarriages and stillbirths. There is no evidence of teratogenic effects in humans or animals due to exposure to low levels of lead. There is conflicting information regarding the potential effects of lead on birth weight, gestational age, and growth in children. There is conflicting evidence regarding the potential effects of lead on human chromosomes. In men with occupational exposures, some reproductive effects (e.g., decreased sperm count, abnormal sperm morphology, decreased sperm mobility, and hormonal changes) can occur at blood lead levels of 40 µg/dL or greater (ATSDR 2007b).

Although lead is considered to be carcinogenic in animals with the endpoint being renal cancer, evidence of its carcinogenicity in humans is generally considered to be inadequate. USEPA's IRIS database classifies lead as a probable human carcinogen (Group B2, USEPA 2012a), based on sufficient evidence in animals, but inadequate evidence in humans. Lead carcinogenicity was not evaluated quantitatively in this risk assessment.

Sensitive members of the population can include developing embryos/fetuses/neonates, young children, women, and individuals with chronic neurological dysfunction or kidney disease. Older adults are at risk for lead-associated hypertension (NAS 1993). The embryo/fetus/neonate may be at increased risk due to the effects of lead because of a developing nervous system that is more sensitive to the effects of lead, and the transfer of maternal lead during pregnancy and lactation. Young children may be especially at risk compared to adults because they absorb more lead from the gastrointestinal tract; retain more absorbed lead; have a greater prevalence of nutritional deficiencies (e.g., calcium, iron, and zinc), which can increase both the absorption and the toxic effects of lead; have an incompletely developed blood-brain barrier; have a developing nervous system that is more sensitive to the effects of lead; ingest much more soil/dust per kg body weight; ingest more water per kg body weight; and inhale more air per kg body weight. Women who are pregnant, are lactating, or have osteoporosis may be at greater risk due to lead because these conditions may intensify the mobilization of lead from bone.

Blood lead level is the easiest and most widely used index of lead exposure and toxicity. Blood lead primarily reflects recent exposure for lead but can also reflect, to a lesser extent, the body burden of lead, which is more related to long-term exposure. For children and fetuses, 10 μg/dL was generally considered a blood lead level of concern (CDC 1997; CDC 1991) until recently. The CDC now considers 5 μg/dL the reference level, above which the CDC recommends initiating public health actions (CDC 2012). There is less agreement on a single blood lead level of concern for male adults and nonpregnant female adults, but estimates fall within the range of 25 to 40 μg/dL. However, analysis of U.S. National Health and Nutrition Examination Survey (NHANES) II epidemiological data (NAS 1993) shows hypertensive effects in the form of elevated systolic and diastolic blood pressure in older adults at blood lead values well below this range.

The toxic effects of lead are generally considered to be similar regardless of the route of entry. Most adverse effects of lead have been related to lead in blood and (to a lesser extent) tooth dentin. There are relatively few data relating human health effects to exposure-route-specific exposure (e.g., mg/kg-day or m^3/day).

Ingestion is the primary route of exposure for children and other non-occupationally exposed individuals. However, dose-response data based on external ingestion dose (mg/kg/day) in humans were limited. Hematological effects were observed in adult humans who ingested 0.02 to 0.03 mg lead acetate/kg/day for 14 days or 0.01 to 0.02 mg lead acetate/kg/day for 3 to 7 weeks (ATSDR 2007b).

Inhalation is an important route of occupational exposure for adults. However, very little dose-response data in workers using lead air concentrations (mg/m^3) were located. A 47 percent decrease in ALAD enzyme activity was observed in men inhaling lead at a concentration of 0.011 mg/m^3 for 18 weeks.

ATSDR (2007b) reported that no studies were located regarding toxicity of lead in humans or animals specifically from dermal exposure. Dermally-applied lead nitrate is rapidly absorbed by the skin, but the toxicology significance is unknown.

There is no cancer potency factor for lead. There is no consensus RfD because of the difficulty of identifying a threshold level for adverse health effects needed to establish an RfD. Alternatively, lead risk is evaluated through biokinetic modeling predicting a biological marker (i.e., blood lead concentrations).

B.7 Mercury

Elemental mercury is a silvery metallic liquid that is volatile at room temperature. Mercury is found in soil and rocks typically as an ore known as cinnabar, consisting of insoluble mercuric sulfide. Much of the mercury produced in the United States comes from secondary sources, such as recycling. The largest use of mercury is in the electrolytic production of chlorine and caustic soda. Other uses include electrical devices, switches and batteries, measuring and control instruments, medical and dental applications, and electric lighting.

Mercury has been shown to be toxic to human populations as a result of occupational exposure and accidental ingestion of mercury-contaminated food. The nature of mercury toxicity depends on its chemical form. Accidental ingestion exposure to high levels of organic mercury compounds has produced developmental toxicity in humans (ATSDR 1999).

Ingestion of inorganic mercury, the form most likely to be found in soil, has been associated with kidney toxicity in laboratory animals. The adverse effect of concern associated with soil exposure scenarios, therefore, is likely to be kidney toxicity. Ingestion studies with inorganic mercury suggest cancer effects in laboratory animals (ATSDR 1999).

The issue of mercury bioavailability is especially important at mining, milling, and smelting sites because the mercury at these sites often exists, at least in part, as a poorly soluble sulfide and may also occur in particles of inert or insoluble material. These factors all tend to reduce the bioavailability of mercury from soil (ATSDR 1999).

Occupational inhalation exposure to metallic mercury vapor or organic mercury vapor has resulted in neurological effects and kidney toxicity. Toxicity due to inhalation of inorganic mercury salts, the form most likely to be found in soil, has not been studied (ATSDR 1999, USEPA 2012a).

Children are considered a sensitive population for exposure to mercury. Potential differences in sensitivity between children and adults are primarily due to differences in routes of exposure and rates of intake (for example exposure of infants via ingestion of breast milk), greater permeability of the blood-brain barrier in fetuses and infants, and the importance of developmental milestones during childhood exposure periods (such as language or cognitive development). Children also appear to have patterns of tissue distribution of mercury and methylmercury (i.e., biokinetic patterns) that are different to those of adults (ATSDR 1999).

More recently, USEPA has developed the Mercury Research Strategy to address key scientific questions in order to reduce uncertainties currently limiting its ability to assess and manage mercury and methylmercury risks. This strategy will include evaluations to link toxicity to exposure using a biokinetic model, assessment of sensitive populations, evaluation of recent epidemiological studies, and evaluation of immunological effects.

USEPA has published chronic oral RfDs for mercuric chloride and methyl mercury on its IRIS database (USEPA 2012a). The most sensitive adverse effect for mercuric chloride is reported to be the formation of mercury-induced autoimmune glomerulonephritis. Based on weight of evidence from three subchronic feeding and/or subcutaneous studies in rats, the oral RfD for mercuric chloride is 0.0003 mg/kg-day. All treatment groups exhibited a toxic effect; therefore, a no-observed-adverse-effects level (NOAEL) was not reported. An uncertainty factor of 1,000 was applied for extrapolations from LOAEL to NOAEL endpoints, subchronic to chronic exposures, and animal to human populations. USEPA reported a high confidence in the oral RfD for mercuric chloride. USEPA's chronic oral RfD for methyl mercury of 0.0001 mg/kg-day was used to evaluate exposures to mercury in fish (USEPA 2012a). Methyl mercury can be more toxic than mercuric chloride and is likely to be present in fish tissue. Exposures to mercury in all other media were evaluated using the oral RfD for mercuric chloride. Methyl mercury's oral RfD is based on developmental neurologic abnormalities in human infants as determined by epidemiologic studies. An uncertainty factor of 10 has been assigned to this RfD and USEPA's confidence in this RfD is high.

USEPA has classified both mercuric chloride and methylmercury as possible human carcinogens (Group C), based on the absence of data in humans and limited evidence of carcinogenicity in

animals, whereas elemental mercury is in Group D (not classifiable due to inadequate data) (USEPA 2012a). However, there is no cancer potency factor or reference dose for mercury.

B.8 Nickel

Nickel is a naturally occurring metal used in alloys for coins, jewelry, and stainless steel. Nickel compounds are also used for electroplating, ceramics, and battery production. Nickel is released into the environment by volcanoes, combustion of fuel oil, municipal incineration, and industries. People may be exposed to nickel via dermal contact, ingestion, or inhalation.

Contact dermatitis is the most widespread health effect and is an allergic reaction caused by dermal nickel exposure affecting 10–20 percent of the general population. Inhaled nickel particles are deposited in the upper and lower respiratory tract and approximately 20–35 percent of inhaled nickel is absorbed into the blood. Approximately 29–40 percent of ingested nickel is absorbed via oral exposure. Absorbed nickel is excreted in urine and feces. Inhalation exposure, mainly in occupational settings, causes respiratory irritation including chronic bronchitis, emphysema, pulmonary fibrosis, and impaired lung function (ATSDR 2005a).

Individuals sensitized to nickel may be unusually susceptible to nickel because exposure by any route may trigger an allergic response. In addition, people with kidney dysfunction are likely to be more sensitive to nickel because nickel is primarily excreted in urine (ATSDR 2005a).

Carcinogenic effects have been well documented based on occupational inhalation exposures. However, carcinogenic risk from low level environmental exposures to the general population is unclear. Metallic nickel is classified by IARC as 2B (possibly carcinogenic to humans), and nickel compounds are classified as group 1 (carcinogenic to humans, ATSDR 2005a). Nickel refinery dust and nickel sulfide are classified by USEPA as Group A (human carcinogen, USEPA 2012a). Reproductive effects from nickel are unclear. Animal studies suggest that there are deleterious effects; however, human evidence is lacking.

OSHA has set an enforceable limit of 1.0 mg nickel/m³ for metallic nickel and nickel in indoor air for an 8-hour shift over a 40-hour work week (OSHA 2012). USEPA has not published a standard for nickel in drinking water. There is no oral cancer potency factor for nickel. The oral RfD, identified in the CLARC database as 0.02 mg/kg-day (Ecology 2012), allows nickel to be evaluated for noncancer health effects.

B.9 Thallium

Thallium is a metal found in the earth's crust. It has two main chemical states: thallic and thallous; thallous is more common and stable. Thallium is used mostly in the manufacture of electronic devices, switches, and closures. It was previously used as a rat poison, but is no longer used due to its potential harm to human health. Thallium is produced or used in power plants, cement factories, and smelters. People may be exposed to thallium via ingestion and inhalation.

There are limited data indicating Thallium's effects on health due to inhalation. Some occupational data indicate cardiovascular and gastrointestinal systems are not affected by inhalation. Limited data show that thallium is absorbed through the gastrointestinal tract and excreted through urine. Acute exposure to thallium through ingestion can have multiple health effects, including respiratory damage, cardiovascular damage, gastrointestinal irritation, hepatic

changes, neurologic changes (paresthesia of the hands and feet, weakness, tremors, coma, and convulsions), renal impairment, and hair loss. Developmental, genotoxic, and reproductive effects in humans have not been confirmed due to the scarcity of human and animal studies; however, some studies indicate thallium affects reproductive function and may produce developmental toxicity (ATSDR 1992b, USEPA 2012a). Carcinogenicity of thallium in humans has also not been assessed (USEPA 2012a).

USEPA has determined a water quality criteria level of 13 parts per billion (ppb) and an MCL of 0.002mg/L (USEPA 2012a). OSHA has established an occupational limit of 0.1 mg of soluble thallium compounds per cubic meter of workplace air (mg thallium/m^3/skin) for an 8-hour workday over a 40-hour workweek (OSHA 2012). There is no cancer potency factor or reference dose for thallium.

B.10 Zinc

Zinc is used in a wide variety of industrial, agricultural, and consumer products. It is found in all human tissues and all body fluids and is essential for growth, development, and reproduction. The Recommended Daily Allowance for zinc is 11 mg/day for men and 8 mg/day for women, with a slightly higher requirement for pregnant women. Individuals with adequate nutritional levels of zinc absorb approximately 20 to 30 percent of all ingested zinc (ATSDR 2005b).

Inhalation and ingestion are the main routes of exposure. Inhalation exposure to high concentrations of some zinc compounds (zinc oxide fume) has been associated with metal fume fever in occupational settings. Attacks of metal fume fever are characterized by chills and fever, weakness, and sweating. Recovery usually occurs within 24 to 48 hours. Zinc chloride, a corrosive inorganic salt, is an irritant to the mucous membranes in the respiratory tract. Zinc chloride also may cause skin irritation and inflammatory changes with dermal exposure (ATSDR 2005b).

Gastrointestinal distress is a common symptom following acute oral exposure to zinc compounds. Symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Anemia also may occur in severe cases of acute exposure or in high-dose exposures of longer duration due to decreased absorption of copper. Long-term exposure may also decrease levels of high-density lipoprotein (HDL) cholesterol (ATSDR 2005b).

Developmental or reproductive toxicity has been reported in laboratory animals with relatively high levels of exposure to zinc. Human studies have not identified adverse reproductive or developmental effects from exposure to zinc. Available epidemiological studies of human populations and toxicity studies in laboratory animals do not indicate that zinc is carcinogenic. USEPA has given zinc a carcinogenicity weight-of-evidence classification of D (not classifiable as to human carcinogenicity), based on inadequate evidence in humans and laboratory animals (USEPA 2012a).

Zinc interacts with other trace metals and has a protective effect against toxicity from exposure to lead and cadmium. Excessive dietary zinc produces a copper deficiency in laboratory animals. Similar findings have been observed in humans receiving long-term treatment with zinc. No specific data regarding human populations that are unusually susceptible to the toxic effects of

zinc have been identified; however, individuals who are malnourished or have a marginal copper status may be more susceptible to the effects of excessive zinc exposure (ATSDR 2005b).

OSHA has set an average legal limit of 1 mg/m^3 for zinc chloride fumes and 5 mg/m^3 for zinc oxide (dusts and fumes) in workplace air during an 8-hour workday, 40-hour work week (OSHA 2012). NIOSH has made similar recommendations. There is no cancer potency factor for zinc. The oral reference dose, identified in the CLARC database as 0.3 mg/kg-day (Ecology 2012), allows zinc to be evaluated for noncancer health effects.