IEc

Risk Assessment of the Inhalation of Particulate Matter from Lake Bed Sediments in the Upper Columbia River/Lake Roosevelt

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EXECUTIVE SUMMARY

Industrial Economics, Incorporated (IEc) conducted a risk assessment of exposures to airborne particulate matter at three locations along the Upper Columbia River (UCR) in the north-central portion of Washington State. The analysis focuses on potential health risks that may result from the inhalation of re-suspended particles from contaminated sediments along the lakebed, banks and beaches of the UCR, in addition to general ambient particulate matter (PM) concentrations in the area. This report addresses potential exposures and risks to a variety of non-tribal individuals who may spend time at or near the portion of the UCR site designated as the Lake Roosevelt National Recreation Area (LRNRA) – nearby residents, park employees, and park visitors.

Over the past century, discharges of granulated slag, liquid effluents, emissions, and accidental spills and "upsets" from smelting processes and facility operations by the Teck Cominco Metals Limited facility in Trail, British Columbia have contributed to metals contamination in the sediment of the UCR (USEPA, 2009a). Concerns over re-suspension of metal-contaminated particles, particularly during high wind events (HWEs) in the spring, when the water level of the Lake Roosevelt reservoir is drawn down, led to sampling of particulates in air by the U.S. Geological Survey (USGS) between 2002 and 2006. IEc based its risk assessment on these data, which included analyses of 33 metals plus PM measured as particles 10 microns in diameter or smaller (PM_{10}). USGS collected samples at three different monitoring sites – Seven Bays Marina, Inchelium, and Marcus, from north to south. The data collected between 2002 and 2005 consisted of 30 24-hour composite samples collected regularly at each monitor between January and September. The 2006 dataset consisted of a smaller set of seven-day composite samples of particles collected during HWEs between mid-March and May when the lake levels are lowest and lakebed exposure is maximized.

IEc's risk assessment follows the conventional four steps of hazard identification, exposure assessment, concentration-response (C-R) assessment, and risk characterization. We also performed a screening analysis following the hazard identification step, in order to focus the risk analysis on those contaminants detected at levels that could potentially pose unacceptable risks to human health.

In the hazard identification step, we researched the potential carcinogenic and non-carcinogenic effects that have been associated with each of the detected contaminants in the toxicological, epidemiological, and clinical literature. We found a subset of the contaminants measured at the site to be carcinogenic. In addition, many of the metals have been associated with a variety of non-cancer effects. The majority of these metals affect the respiratory system. However, some of the metals also had effects in organ

systems remote from the respiratory tract, including cardiovascular, gastrointestinal, immunological and other effects. PM exposure has also been associated with increased risk of premature mortality. Lead has been found to adversely affect health in exposed individuals a number of ways, most notably affecting the neurobehavioral development of children.

In the screening step, IEc compared the maximum detected concentration of each metal contaminant with a human health risk-based (HHRB) screening level, reflecting either cancer or non-cancer effects, to determine the final list of contaminants of potential concern (COPC). We compared the maximum detected concentration of each metal from the 2002 to 2005 data to a long-term (i.e., chronic) HHRB screening level from EPA's Regional Screening Levels for Chemical Contaminants at Superfund Sites. We also compared the maximum HWE data, collected in 2006, for each metal to a very short-term (i.e., acute) screening level to assess whether any of the metals posed an imminent health danger to individuals exposed during a HWE that may occur within the course of a single 24-hour period. In addition, we compared PM levels to the current PM National Ambient Air Quality Standards (NAAQS). We carried 10 chemicals through to the final risk assessment as chronic COPCs: arsenic, barium, cadmium, chromium, cobalt, lead, manganese, nickel, thorium, and vanadium. No metals exceeded the acute screening levels.

IEc assessed exposure, cancer risk, and non-cancer hazard to all metals except for lead and thorium in accordance with recommended methods from EPA's *Risk Assessment Guidance for Superfund, Part F* (RAGS F; USEPA, 2009b). We assessed lead exposure and risk using EPA's recommended biokinetic models – the Integrated Exposure Uptake Biokinetic (IEUBK) model for exposures to children age 7 and under, and the Adult Lead Model (ALM) to evaluate potential lead exposures to fetuses following exposure to women of childbearing age. Lead risk assessment generally focuses on the blood lead levels (BLLs) of children at a contaminated site when they are part of the exposed population, since young children tend to have higher exposures than adults, have higher lead absorption rates, and are more susceptible to lead effects (USEPA, 2009a). We evaluated risk from exposure to thorium, a radionuclide, by first calculating a total radiation exposure for each exposed group from inhaled thorium-232 (Th-232), the most commonly occurring thorium isotope, and then multiplying this total by the cancer slope factor for Th-232 found in EPA's Health Effects Assessment Summary Tables (HEAST) - Radionuclides Table (USEPA, 2001).

For the non-lead metals, we calculated time-weighted Reasonable Maximum Exposure (RME) exposure concentrations (ECs) – the highest exposure that is reasonably expected to occur at a site – according to Superfund practice for each of the three sampling locations. The RME ECs combine high-end estimates of the parameters describing exposure to individuals, such as days per week exposed, with a conservative estimate of the mean concentration of each metal (USEPA, 1989). We derived the exposure parameters from our conceptual site model (CSM) of the UCR site, which identified three potentially exposed non-tribal groups of individuals ("receptors") – adult and child residents living near the lake who are exposed both at home and during frequent visits to

the LRNRA; adult employees working at the LRNRA, particularly archaeologists who excavate artifacts from the lake bed sediments; and adult and child visitors to the LRNRA engaged in recreational activities on the lake. Table ES-1 presents the exposure parameters for each receptor.

We assessed risks from long-term (i.e., chronic) metal exposures to residents and permanent LRNRA employees who are assumed to be exposed for several years. We also assessed risk from shorter-term (i.e., subchronic) exposures to temporary LRNRA archaeologists and child and adult park visitors whose exposures are expected to last only a few months for the workers and just two weeks for the visitors. Collectively, these three sets of receptors bracket the range of likely exposures to particles in air, in terms of both exposure frequency and duration. We combined the exposure parameters for each receptor group with the 95th percentile Upper Confidence Limit on the mean for each metal (95UCL) in each location, derived using EPA's ProUCL software, to produce location-, receptor-, and metal-specific ECs in micrograms per cubic meter.

For lead, we estimated a cumulative exposure estimate using the IEUBK and ALM models; this cumulative exposure reflects the intake of lead from multiple media at the site, including direct ingestion of lake sediments, ingestion of lead contaminated fish caught in Lake Roosevelt, and inhalation of lead particulates in air. The models then convert these exposure estimates to an overall body burden of lead expressed as a BLL in micrograms per deciliter of blood. Unlike the non-lead metals, the lead models use as a concentration input the mean lead concentration in air, which is more appropriate than the 95UCL for calculating a distribution of BLLs in a child population. We conducted the lead analysis using the highest of the three mean lead concentrations, with the intention of analyzing additional areas if lead at the highest monitor posed unacceptable risks.

For thorium, we first calculated a total radiation exposure for each exposed group in each area based on the exposure parameters in Exhibit ES-1, including the inhalation rate in cubic meters per hour, and a measure of the radioactivity of thorium per unit mass. In the absence of data on the specific radionuclides present in each sample, we assumed all inhaled thorium was Thorium-232 (Th-232), the most common thorium isotope. While it is possible that a more radioactive isotope of thorium (Th-230) might be present following decay of uranium-238-containing wastes discharged to the UCR, we found that the small amount of uranium detected at the monitors, along with its extremely long half life, suggest Th-230 would likely be present in exceptionally small amounts.

TABLE ES-1. EXPOSURE PARAMETERS SUMMARY

	OUTDOOR WORKERS			LOCAL RESIDENTS		PARK VISITORS	
	FULL-TIME ARCHAEOLOGIST	SEASONAL ARCHAEOLOGIST	FULL-TIME MAINTENANCE	ADULTS	CHILDREN	ADULTS	CHILDREN
CA = contaminant concentration in air ($\mu g/m^3$)	Contaminant-specific	Contaminant-specific	Contaminant-specific	Contaminant-specific	Contaminant-specific	Contaminant-specific	Contaminant-specific
ET = Exposure Time (hours/day)	9 [1]	9 [1]	9 [1]	24	24	8 [6]	8 [6]
EF = Exposure Frequency (days/year)	80 [2]	65 [3]	200 [4]	350 [5]	350 [5]	14 [6]	14 [6]
ED = Exposure Duration (years)	20 [1]	1 [7]	25 [1]	30 [5]	6 [5]	30 [5]	6 [5]
AT = Averaging Time – Noncancer (hours)	175,200	8,760	219,000	262,800	52,560	262,800	52,560
AT = Averaging Time – Cancer (hours)	613,200	613,200	613,200	613,200	613,200	613,200	613,200
Inhalation rate (For radionuclide exposure only) (m^3/hr) [8]	1.3 m^3/hr [9]	1.3 m^3/hr [9]	1.3 m^3/hr [9]	0.55 m^3/hr [10]	0.3 m^3/hr [11]	0.55 m^3/hr [10]	0.3 m^3/hr [11]
Lead Analysis	For analysis of lead, we use EPA's IEUBK and ALM models. We use the default parameters developed for the IEUBK model, but update concentrations in air, sediment, and fish to site-specific values. For the ALM, we use default values developed by EPA, but apply site-specific concentrations of lead in air, sediment, and fish at the UCR site. For the analysis of lead exposure through inhalation, we assume the same adult breathing rate we use for the radionuclide analysis, 13.2 m^3/day. [10] See sections 4.4 and 4.5.3 in the report for more details on the two models and parameters.						

[1] Based on interviews with archaeologists and maintenance workers at Lake Roosevelt (IEc, 2007a & b).

[2] Assumes a 5 day work week for approximately 9 months of the year, 40 percent of which is spent in the field. Estimate based on interviews with archaeologists at Lake Roosevelt (IEC, 2007a).

[3] Assumes a 5 day work week for approximately 3 months of the year, with all time spent in the field. Estimate based on professional judgment.

[4] Assumes a 5 day work week for 12 months a year (50 weeks, vacation adjusted), 4 days per week spent outdoors. Estimate based on interviews with maintenance workers at Lake Roosevelt (IEc, 2007b).

[5] USEPA, 1991a.

[6] Assumes park visitors are at Lake Roosevelt for two weeks a year and are exposed for eight hours a day, based on professional judgment.

[7] Assumes seasonal staff is a one year commitment and most do not return, based on interviews with archaeologists at Lake Roosevelt (IEc, 2007a).

[8] Exposure to radionuclides is calculated as the radionuclide concentration in mass per m^3 times the receptor's inhalation rate, ET, EF, and ED, and the specific activity of the radionuclide (in pCi per unit mass) to obtain a total estimate of exposure in pCi.

[9] Hourly average for outdoor workers (USEPA, 1997a).

[10] Average of adult male and female rates (USEPA, 1997a).

[11] Weighted average of rates for children under the age of six (USEPA, 1997a).

In the C-R assessment step for non-lead metals, we searched for available inhalation toxicity information for cancer and non-cancer effects published by the sources identified in the hierarchy recommended by EPA's Superfund program in the directive entitled *Human Health Toxicity Values in Superfund Risk Assessment* (USEPA, 2003a) and selected the value from the highest ranking source. For non-cancer effects we identified chronic and, if available, subchronic non-cancer inhalation toxicity values, such as Reference Concentrations (RfCs); for cancer, we identified inhalation unit risks (IURs). We then conducted our risk characterization according to EPA's RAGS F for non-lead metals. For noncancer hazards, we calculated for each location receptor-and metal-specific hazard quotients (HQs), which are the ratio of the time-weighted EC for a given metal to its RfC or other appropriate toxicity value. If an HQ exceeds one, there is a possibility that some non-cancer effects may occur. Generally, larger HQs indicate greater levels of concern. If the HQ is less than one, it is believed that there is no increased risk of non-cancer health effects at a site. For each receptor in each location, the HQs are summed across all metals to yield a Hazard Index (HI), which, as a first pass, is also compared to a benchmark of one. The excess risk of cancer over a lifetime as a result of exposure to a particular contaminant is expressed in terms of a probability of developing cancer due to that exposure. EPA considers cancer risks between 1E-06 (i.e., 1 in 1,000,000) and 1E-04 (i.e., 1 in 10,000) to be generally acceptable (USEPA, 1991b).

For lead, we compared the results of the IEUBK and ALM models to established benchmarks for the distribution of BLLs in children under seven and in fetuses; specifically, no more than five percent of children or fetuses should have a BLL above 10 micrograms per deciliter. For thorium, we use the cancer slope factor for Th-232 found in EPA's HEAST - Radionuclides Table (USEPA, 2001).

We present the results of our risk assessment in Tables ES-2 (noncancer hazards) and ES-3 (cancer risks). The HI calculated for exposure to all of the non-lead COPCs was below one for all of the exposed groups in all three locations, with the highest value estimated for residents living near the Seven Bays site (with an HI of 0.91). The cancer risks due to inhalation exposures at the site were within EPA's acceptable risk range (i.e., 1E-06 to 1E-04) for each individual carcinogen as well as across all carcinogens. The highest total cancer risks are experienced by residents living in proximity to the Marcus site, with risks of 1.79E-05. These cancer risks include the results of the thorium analysis.

In addition, as noted earlier (not shown in the exhibit) none of the metals detected in 2006 had a maximum concentration that exceeded human health-risk benchmarks for acute exposures, such as those that might occur during HWEs.

TABLE ES-2. HAZARD INDICES FOR RECEPTORS AT THE UCR SITE DUE TO INHALATION EXPOSURE TO PARTICLES

RECEPTOR	INCHELIUM	MARCUS	SEVEN BAYS
RESIDENT	0.86	0.47	0.91
FULL-TIME ARCHAEOLOGIST	0.07	0.04	0.08
SEASONAL ARCHAEOLOGIST	0.01	0.01	0.01
FULL-TIME MAINTENANCE WORKER	0.18	0.10	0.20
PARK VISITOR	0.08	0.07	0.06

TABLE ES-3. TOTAL CANCER RISKS FOR RECEPTORS AT THE UCR SITE DUE TO INHALATION EXPOSURE TO PARTICLES

RECEPTOR	INCHELIUM	MARCUS	SEVEN BAYS
RESIDENT	1.75E-05	1.79E-05	1.35E-05
FULL-TIME ARCHAEOLOGIST	1.01E-06	1.03E-06	7.79E-07
SEASONAL ARCHAEOLOGIST	2.56E-08	2.87E-08	1.87E-08
FULL-TIME MAINTENANCE WORKER	3.15E-06	3.22E-06	2.44E-06
PARK VISITOR	3.13E-07	1.68E-07	2.03E-07

The lead risk assessment results were also well within acceptable levels. The results of our IEUBK model run indicate that only 0.52 percent of children exposed at the site to inhaled particles, fish ingestion and beach sediment would have BLLs above the level of concern, 10 µg/dL. This is well below the target of 5 percent. The ALM predicted that only 0.36 percent of fetuses would have BLLs greater than 10 µg/dL if their mothers lived near the site and were exposed to lead through eating fish, recreating on the UCR beaches and inhaling re-suspended sediments.

This analysis is subject to uncertainties associated with measuring ECs and determining the toxicological potency of the contaminants evaluated. In general, most of the assumptions made by IEc in conducting this analysis and those embedded in published toxicity values are more likely to lead to an overestimation of risk, rather than an underestimation. Some of the most significant limitations, however, are the small number of monitors where data were collected and the limited set of samples targeted at estimating exposures during HWEs during the drawdown period for the reservoir. The monitors provide information on exposures in only three of the six river reaches defined by EPA for the broader remedial investigation (RI), and likely do not provide sufficient information on spatial trends in particle concentrations. The lack of HWE concentration

data for multiple years limits our ability to draw broad conclusions about the acute risk from inhalation exposures during these events, as the re-suspension of particles may be sensitive to year-to-year variation in meteorological factors such as rainfall. Data from additional monitors and for additional years of HWEs would help to refine these risk estimates and allow for broader, more robust conclusions regarding the level of risk posed by metal-bearing particulates at the UCR site.

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LIST OF ACRONYMS

AF	Absorption Factor
ALM	Adult Lead Model
AT	Averaging Time
ATSDR	Agency for Toxic Substances and Disease Registry
BKSP	Biokinetic Slope Factor
BLL	Blood Lead Level
CA	Concentration in Air
CalEPA	California Environmental Protection Agency
Ci	Curie
COI	Contaminant of Interest
COPC	Contaminant of Potential Concern
CSM	Conceptual Site Model
C-R	Concentration-Response
EC	Exposure Concentration
ED	Exposure Duration
EF	Exposure Frequency
ET	Exposure Time
GSD	Geometric Standard Deviation
HEAST	Health Effects Assessment Summary Table
HI	Hazard Index
HHRA	Human Health Risk Assessment
HHRB	Human Health Risk-Based
HQ	Hazard Quotient
HWE	High Wind Event
IEc	Industrial Economics, Incorporated
IEUBK	Integrated Exposure Uptake Biokinetic Model
IR	Intake Rate
IRIS	EPA's Integrated Risk Information System
IUR	Inhalation Unit Risk

LRAQM	Lake Roosevelt Air Quality Monitoring
LRNRA	Lake Roosevelt National Recreation Area
MRL	Minimal Risk Level
NAAQS	National Ambient Air Quality Standards
NIOSH	National Institute for Occupational Safety and Health
NPS	National Park Service
Pb	Lead Concentration
PbB	Geometric Mean Blood Lead Concentration in Exposed Population
pCi	Picocurie
PM	Particulate Matter
PPRTV	Provisional Peer Reviewed Toxicity Values
p-RfC	Provisional RfC
RAGS A	Risk Assessment Guidance for Superfund (Part A)
RAGS F	Risk Assessment Guidance for Superfund (Part F)
REL	Reference Exposure Level
RfC	Reference Concentration
RI/FS	Remedial Investigation and Feasibility Study
RM	River Mile
RME	Reasonable Maximum Exposure
SF	Slope Factor
SRC	Syracuse Research Group
STSC	EPA's Superfund Health Risk Technical Support Center
TR	Total Radioactivity Inhaled
UCL	Upper Confidence Limit
UCR	Upper Columbia River
USEPA	United States Environmental Protection Agency
USGS	United States Geological Survey
WSHD	Washington State Health Department
95UCL	95 Percent Upper Confidence Limit on the Mean

CHAPTER 1 | INTRODUCTION

1.1 SITE DESCRIPTION/HISTORY

Over the past hundred years, the Upper Columbia River (UCR) has been affected by hazardous waste contamination from multiple sources. This has included discharges of granulated slag[1], liquid effluents, emissions, and accidental spills and "upsets" from smelting processes and facility operations by Teck Cominco Metals Limited at the Trail facility located in Trail, British Columbia (USEPA, 2009a). In 1999, the Colville Confederated Tribes petitioned EPA to conduct an assessment of this contamination, citing concerns to human health and the environment. In response to this petition, a remedial investigation and feasibility study (RI/FS) is currently underway at the site. EPA is investigating the impacts of the river contamination on human health through multiple exposure pathways (e.g., air, water). To date, EPA has only collected sediment and fish tissue samples from the site as part of its 2001 Phase I sampling effort (USEPA, 2006a & b), but plans to take additional samples in other media over the next few years.

The UCR site is located in upper central portion of the State of Washington. It extends from the U.S.-Canada border to the Grand Coulee Dam, covering approximately 150 river miles of the Columbia River (USEPA, 2009a). The portion of the river directly upstream from the Grand Coulee Dam forms the Lake Roosevelt reservoir. The water level of the reservoir is managed by the U.S. Bureau of Reclamation to provide flood control, irrigation, recreation, fisheries, navigation, flow regulation, and power generation (USEPA, 2005). During the annual operating cycle, water levels are typically drawn down between January and April to accommodate increased spring flow (USEPA, 2009a; hereafter, "drawdown").

A large part of Lake Roosevelt has been designated as the Lake Roosevelt National Recreation Area (LRNRA), which is managed by the National Park Service (NPS). The LRNRA attracts more than 1.3 million visitors per year and employs approximately 54 permanent and 49 seasonal employees (USEPA, 2009a). Recreational activities at the site include boating, fishing, swimming, wading, camping and hunting (USEPA, 2009a). Other non-recreational uses of the site include municipal drinking water supply, fish hatcheries, and irrigation water for commercial agriculture.

[1] Granulated slag is a byproduct of smelting furnaces. It consists of sand-sized granules that contain various quantities of trace and major metals (USEPA, 2009a).

The Colville and Spokane Indian Reservations border the site, with a total population of approximately 10,000 people (USEPA, 2009a). Several other small towns and communities also exist along the river, such as Northport, Marcus, Kettle Falls, Coulee Dam, Grand Coulee and Inchelium, with a total local population of approximately 4,600 people (USEPA, 2009a).

1.2 PURPOSE/SCOPE

As part of the RI/FS process, EPA identified the inhalation of re-suspended contaminated sediment particles as a potential complete exposure pathway for recreational, occupational, and subsistence receptors in their conceptual site model in the draft *Work Plan for the Human Health Risk Assessment for the Upper Columbia River Site Remedial Investigation and Feasibility Study* (hereafter, "UCR HHRA Workplan"; USEPA, 2009a; Figure 5-3). As described above, the water levels in the UCR fluctuate seasonally due to reservoir operations. In the spring, the reservoir is drawn down, leading to low pool conditions, which expose large areas of sediment. During this time, fine-grained sediment particles may become airborne due to wind. A recent air monitoring effort was conducted by the United States Geological Survey (USGS) as part of an air quality study to investigate the occurrence and distribution of trace elements in air along Lake Roosevelt. We have investigated this exposure pathway using the USGS data in order to determine whether the concentration of contaminants on particles in the air is expected to adversely affect human health at the site. We assessed risks to potentially exposed non-tribal groups at the site, as a comprehensive survey is currently underway to assess tribal exposures at the site.

1.3 SOURCE OF DATA

USGS collected data on the concentration of total particulate matter with diameter of 10 µm or less (PM_{10}) as well as the concentration of trace metals on airborne dust particles at three sites along Lake Roosevelt (Seven Bays Marina, Inchelium and Marcus[2]) as part of the Lake Roosevelt Air Quality Monitoring (LRAQM) program. The concentration of PM_{10} and 33 different metals were measured at each monitoring site. Each site consisted of a PM_{10} high volume air sampler and a fully instrumented meteorological station. The PM_{10} samples were collected using 8-inch by 10-inch quartz fiber filters. The trace metal samples were analyzed at the USGS laboratory in Denver, CO using Inductively Coupled Plasma Mass Spectroscopy (USGS 2003, 2004, 2005, 2006).

During the years 2002-2005, 24-hour composite samples were collected at regular intervals between the months of January and September.[3] This dataset consists of

[2] Note that in the first sampling year (2002), a monitor was placed at Kettle Falls. However, in the following year, the monitor was moved to Marcus due to safety concerns at the Kettle Falls location (USGS, 2004).

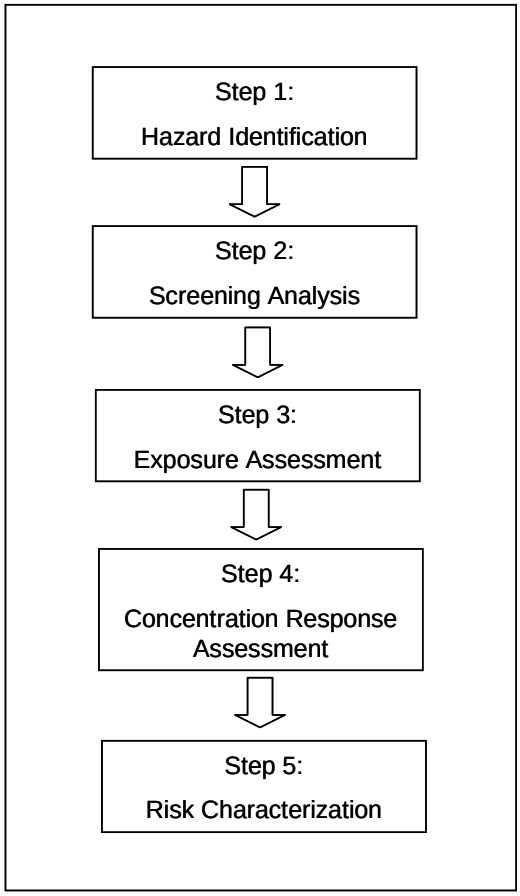
[3] For 2002 and 2003, samples were collected every six days throughout the sampling period. In order to increase the probability of measuring concentrations during high wind events, an alternate sampling plan was enacted during 2004 and 2005. The samples were taken less frequently (every 12 days) in January and February, then increased to every six days during March. During the drawdown period (April and May), sampling was increased further to every three days and then was decreased again to every six days for June through September.

roughly 30 samples per year per site. For the year 2006, seven-day composites samples were taken, consisting of 30-minute samples collected only during high wind events (HWEs) (i.e., above a pre-determined wind speed threshold) between mid-March and May when the lake levels are normally at their lowest and the bed sediment exposure is maximized (i.e., the "drawdown" period). For 2006, there are approximately 10 samples per site in the dataset. Meteorological data, including wind speed and direction, relative humidity, and precipitation were also collected simultaneously for all years' samples.

1.4 ORGANIZATION OF THE DOCUMENT

The remainder of this document is organized into six chapters that describe the approach and results of the five-step process we employed to assess risk from inhalation of re-suspended sediment at the UCR site, as shown in Figure 1. Chapter 2 contains the hazard identification (i.e., our assessment of the potential adverse health effects that have been linked to the contaminants measured at the site through the inhalation pathway). Chapter 3 outlines the screening analysis that we used to narrow the focus of the risk assessment to only those contaminants with the potential to pose inhalation risks to receptors at the site above de minimus levels. Chapter 4 describes our evaluation of the exposures to airborne metals that would likely be experienced by site receptors. Chapter 5 provides our concentration-response (C-R) assessment of the toxicity of each of the contaminants we evaluated. Chapter 6 explains our approach to the risk characterization step, where we calculate inhalation risk estimates and discuss the uncertainties associated with our estimates of risk.

FIGURE 1. RISK ASSESSMENT PROCESS



CHAPTER 2 | HAZARD IDENTIFICATION

In the first step in the risk assessment, we investigated the contaminants detected in air at the UCR site to identify the potential hazards, both carcinogenic and noncarcinogenic, posed by these contaminants. This involved examining the available scientific data for each monitored contaminant to assess and describe any potential links between each chemical and their adverse effects. Below we outline the methodology we employed to identify inhalation toxicity information. We then provide an overview of the major health effects (both cancer and non-cancer) that could result from exposure to inhaled sediment particulates at the site.

2.1 METHODS

2.1.1 METALS

We investigated the potential hazards from inhalation exposure to each of the 33 metals analyzed by USGS. The 33 metals are listed below in Table 1. We searched for available acute, subchronic and chronic inhalation toxicity information published by the sources identified in the hierarchy recommended by EPA's Superfund program in the directive entitled *Human Health Toxicity Values in Superfund Risk Assessment* (USEPA, 2003a). This included inhalation unit risks (IURs) and reference concentrations (RfCs) from EPA's Integrated Risk Information System (IRIS), Provisional Peer-Reviewed Toxicity Values (PPRTVs), California Environmental Protection Agency (Cal EPA) reference exposure levels (RELs), the Agency for Toxic Substances and Disease Registry (ATSDR) minimal risk levels (MRLs), and Health Effects Assessment Summary Tables (HEAST) values (HEAST, 1997).

TABLE 1. METALS DETECTED AT USGS SAMPLING SITES

DETECTED METALS			
Antimony	Cobalt	Molybdenum	Thorium
Arsenic	Copper	Nickel	Titanium
Barium	Gallium	Niobium	Uranium
Beryllium	Iron	Phosphorous	Vanadium
Bismuth	Lanthanum	Rubidium	Yttrium
Cadmium	Lead	Scandium	Zinc
Cerium	Lithium	Silver	
Cesium	Manganese	Strontium	
Chromium	Mercury [1]	Thallium	

[1] Mercury data were only collected in 2004 (30 samples) and 2005 (35 samples).

We were unable to identify any inhalation toxicity information for 18 of the metals detected at the site within the hierarchy sources.[4] In order to ascertain potential human health effects of these metals, we examined other sources of information, including *Casarette and Doull's Toxicology* (1996), the U.S. National Library of Medicine's TOXNET database[5], the Merck Index (Windholz, 1983) and the National Institute for Occupational Safety and Health (NIOSH) Recommended Exposure Limits (RELs).[6] This additional search identified hazard information for 14 of these chemicals. However, we were unable to locate any information on the inhalation toxicity of four metals (lanthanum, rubidium, niobium, and scandium).

2.1.2 PARTICULATE MATTER

We also investigated the health effects due to inhalation exposure of particulate matter (PM) (both fine and coarse size fractions). EPA's *Integrated Science Assessment for Particulate Matter* (USEPA, 2009b) provided information about the short- and long-term health effects of inhalation exposure from PM_{10} and $PM_{2.5}$.

2.2 RESULTS

Appendix A displays a summary table of the health effects that have been linked to the measured contaminants at the site through the inhalation exposure pathway. We identified 10 carcinogens from among the 33 metals measured at the site (arsenic, beryllium, bismuth, cadmium, chromium, cobalt, nickel, thorium, uranium and vanadium). These metals all produce tumors of the respiratory tract (e.g., lung, trachea, bronchus, nasal passage) with the exception of the radionuclides, thorium and uranium. We assessed risks from these radioactive metals based on an elevated total cancer risk (see Section 4.3 for further information).

The contaminants measured at the site have also been associated with a variety of non-cancer effects. The majority of these metals affect the respiratory system. However, some of the metals also had effects in organ systems remote from the respiratory tract, with one or more metals linked to adverse effects on the cardiovascular, gastrointestinal, and immune systems as well as hematological, musculoskeletal, hepatic, renal, dermal, ocular, neurological, reproductive and developmental effects. PM exposure has also been associated with increased risk of premature mortality.

Lead has been found to adversely affect health in exposed individuals a number of ways, including effects on neurobehavioral development, heme synthesis, and the cardiovascular, renal, immune, and reproductive systems (USEPA, 2006c). Lead risk assessment generally focuses on the blood lead levels (BLLs) of children at a contaminated site when they are part of the exposed population, since young children tend to have higher exposures than adults, have higher lead absorption rates, and are more

[4] This included lithium, phosphorus, scandium, titanium, iron, zinc, gallium, rubidium, strontium, niobium, molybdenum, silver, antimony, cesium, lanthanum, cerium, thallium, and bismuth.

[5] http://toxnet.nlm.nih.gov/.

[6] http://www.cdc.gov/niosh/npg/default.html.

susceptible to lead effects (USEPA, 2009a). Toxicity values per se are not available for lead and therefore the risks from lead exposure are assessed through the use of two biokinetic models that relate inhaled lead concentrations with BLLs: the Integrated Exposure Uptake Biokinetic (IEUBK) model and the Adult Lead Model (ALM), as described further in Section 4.4.

CHAPTER 3 | SCREENING ANALYSIS

The second step in our risk assessment process involved a screening analysis in order to narrow the focus of the risk assessment to only include those metals that would pose a potential risk to receptors at Lake Roosevelt through the inhalation exposure route. This involved identifying human health inhalation screening levels and comparing these to the monitoring data from Lake Roosevelt. Below we describe the methodology we employed for the screening analysis, and the resulting contaminants of potential concern (COPC) that we identified and carried through to the risk assessment. For more detail on the screening analysis methodology and results, please see Appendix B, which provides a detailed memorandum describing the screening analysis.

3.1 METHODS

Our screening analysis consisted of comparing the maximum contaminant concentrations measured by the three USGS monitors on-site between 2002 and 2006 with conservative human health risk-based (HHRB) screening values when available, or, in the case of PM concentrations, with the current PM National Ambient Air Quality Standards (NAAQS). If the maximum concentration for a contaminant at the site exceeded the screening value, then we considered it a COPC and carried the contaminant through to our risk assessment.

We assessed that receptors at Lake Roosevelt (e.g., park employees, local residents and recreators; see Chapter 4 for further details on site receptors) would likely be exposed to inhaled particles in two main ways: short periods of exposure to high concentrations of particles re-suspended during HWEs or longer periods of exposure to varying concentrations of particles.[7] For the first exposure scenario, we performed a screen using acute toxicity benchmark concentrations intended to protect against extreme exposures to high concentrations. For the second scenario, we compared maximum concentrations against chronic HHRB screening values, to protect against longer-term exposure to particles at the site. If we could not identify appropriate acute toxicity values or chronic HHRB screening values for a contaminant, we employed alternative means for determining whether the chemical was a COPC at the site, as described below.

3.1.1 CONTAMINANT CONCENTRATIONS

We first calculated summary statistics (e.g., mean, median, minimum, maximum) for each contaminant by observation site (Marcus/Kettle Falls, Inchelium or Seven Bays) and

[7] We defined a HWE as four or more consecutive hours with mean wind speed at or above 5.14 m/s. This definition is consistent with that used by USGS when designing the sampling protocol for 2006, which was targeted to collect data only during HWEs (USGS, 2006).

year using the air monitoring data provided by USGS for 2002-2006. We then calculated the maximum observed concentration across all years and sites for each contaminant. We compared this overall maximum concentration to the screening values described below. For $PM_{2.5}$, we also calculated the maximum mean annual concentration across all sites and years for comparison to the annual average $PM_{2.5}$ NAAQS.

3.1.2 SCREENING LEVELS

Non-Lead Metals

To determine whether any of the monitored contaminants could potentially pose an unacceptable acute risk, we compared the maximum concentration to the acute inhalation toxicity values we identified in our Hazard Identification (see Chapter 2).[8] If any concentration exceeded the acute toxicity value for that contaminant, we carried it through as a contaminant to assess possible risks of acute exposure.

To determine which contaminants could pose risks through chronic exposure, we compared each maximum contaminant concentration to its corresponding chronic HHRB screening level for residential ambient air from EPA's Regional Screening Levels for Chemical Contaminants at Superfund Sites (hereafter, "Regional Screening Table") (http://www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/index.htm).[9] These screening values are based on chronic toxicity values that were selected using the same hierarchy as we employed in our Hazard Identification (USEPA, 2003a). In addition, they are based on conservative assumptions, such as a hazard quotient of 0.1 for non-cancer and a one-in-one million cancer risk as well as conservative exposure assumptions (e.g., receptors are exposed 350 days per year, 24 hours per day for 30 years).

For contaminants lacking toxicity data or inhalation-based HHRB screening levels, we considered alternative means of screening using other toxicity information. We first assessed exposure through ingestion using oral toxicity data (assuming some fraction of particles inhaled by an exposed individual would be trapped by the mucous lining the respiratory tract and ultimately ingested). If oral toxicity data were also absent, we attempted to perform a comparison to background levels of the contaminant in sediments.[10]

Radionuclides

Thorium and uranium were the two metals detected for which naturally occurring isotopes are radioactive. We compared the maximum detected uranium concentration to a chronic screening level in air from the Regional Screening Tables. The Regional Screening Tables do not include a screening level for thorium, however. To assess the carcinogenic potential of radiation exposure from inhaled thorium, we calculated a

[8] If multiple acute toxicity values from different sources were identified for a contaminant, we compared to the lowest value.

[9] Note that comparing a one-time maximum concentration to a chronic screening value is a conservative approach, in that longer-term average exposures are likely to be significantly lower than this maximum value.

[10] More detail on alternative means of screening is included in Appendix B.

screening level for the most common thorium isotope, Th-232, based on a cancer slope factor for that radionuclide from EPA's HEAST - Radionuclides Table (USEPA, 2001). We then compared this to a total radiation exposure estimate (in pCi) using conservative assumptions about exposure patterns.[11]

Particulate Matter

To determine whether receptors may be at risk from exposure to PM_{10} and $PM_{2.5}$ at the site, we performed a screen using the NAAQS for these particle size fractions. We compared measured PM_{10} concentrations to the current acute 24-hour NAAQS for PM_{10}, and compared our estimated $PM_{2.5}$ concentrations to both the 24-hour and annual average NAAQS for $PM_{2.5}$.[12] In order to convert the measured PM_{10} concentrations to an equivalent $PM_{2.5}$ concentration, we calculated a ratio of $PM_{2.5}/PM_{10}$ using EPA monitor values from a nearby site in Colville, WA.[13]

3.2 RESULTS AND CONCLUSIONS

Below is a summary of the results and implications of our screening assessment. We also present the results in Table 2 below.

3.2.1 NON-LEAD METALS

None of the maximum measured concentrations exceeded available acute toxicity values. We also screened maximum concentrations based on sub-chronic toxicity values when available. None of the metals exceeded sub-chronic values. Therefore, our risk assessment focused on chemicals that did not pass the screening analysis based on chronic screening values. However, our risk assessment includes two subchronic exposure scenarios. We assessed risk for these receptors using sub-chronic toxicity values in order to best match their exposure patterns.

Eight contaminants (chromium, manganese, cobalt, nickel, arsenic, cadmium, barium and vanadium) exceeded chronic HHRB screening values, and were carried through the risk assessment as COPCs. These metals are listed below in Table 2.

We were unable to quantitatively estimate acute or chronic inhalation risks from exposure to 18 contaminants because they lack inhalation toxicity data. Of the contaminants without inhalation toxicity data, eight (lithium, iron, zinc, strontium, molybdenum, silver, antimony, and thallium) had available oral toxicity data, but none exceeded the oral

[11] A Curie (Ci) is a measure of the number of disintegrations per second of a radionuclide (One Ci = 3.7E10 disintegrations per second). One picocurie = 1E-12 Ci.

[12] Note that there is not currently an annual average PM_{10} standard (it was revoked in 2006).

[13] We obtained monitoring data from EPA's AirData website (http://www.epa.gov/oar/data/). Monitoring values were available for these two pollutants at the Colville site for the years 2001 and 2002. We calculated the ratio of $PM_{2.5}/PM_{10}$ at the monitor for both of these years individually. We then calculated a mean ratio, which we applied in our analysis (25 percent).

screening values for risk from chronic exposure via incidental ingestion of inhaled particles. We were unable to locate suitable background data and therefore were unable to exclude any contaminants based on a comparison with background levels. However, we determined that without toxicity data we would not be able to assess risk, and thus did not carry any of these contaminants through to the risk assessment.

TABLE 2. CONTAMINANTS OF POTENTIAL CONCERN THROUGH THE INHALATION EXPOSURE ROUTE

CONTAMINANTS THAT EXCEED SCREENING LEVELS
Arsenic
Barium
Cadmium
Chromium
Cobalt
Lead[1]
Manganese
Nickel
Thorium[2]
Vanadium

[1] Due to a lack of screening values for lead, we could not determine that concentrations exceed human health-based screening levels. However, due to the sensitivity of children to lead exposure, we included it in the risk assessment.
[2] We assessed Thorium on the basis of excess cancer risk due to radiation.

3.2.2 RADIONUCLIDES

The maximum measured concentration for uranium did not exceed the HHRB screening value, and we did not carry it through to the risk assessment. Our analysis of thorium indicated that, based on conservative assumptions about exposure, continual exposure to the maximum measured concentration of thorium may produce enough radiation to raise the incremental lifetime cancer risk above one-in-one million. We therefore carried thorium through to the risk assessment.

3.2.3 PARTICULATE MATTER

The PM_{10} concentrations did not exceed the 24-hour PM_{10} NAAQS. In addition, the estimated $PM_{2.5}$ concentrations did not exceed the 24-hour or annual average $PM_{2.5}$ NAAQS levels. Therefore, we did not evaluate PM in our risk assessment.

3.2.4 LEAD

We did not identify any inhalation toxicity data or inhalation-based screening levels, and could not screen lead from further evaluation based on these grounds. We did compare the maximum measured concentration to the three-month rolling lead NAAQS, which was not exceeded. We decided to carry lead through the risk assessment however, given that children are a sensitive subgroup for the health effects of lead and are also exposed to

lead through other media at the site.[14] Therefore, cumulative levels of lead exposure across multiple exposure pathways could be of concern.

[14] Lead has been detected in other media at the UCR site, such as sediment and fish tissue (USEPA, 2006a & b).

CHAPTER 4 | EXPOSURE ASSESSMENT

This section describes our assessment of the exposures that may be expected at the site for non-tribal receptors such as park visitors and employees, based on their expected activities and patterns of use. We first describe our conceptual site model (CSM), which identifies the sources and distribution of contamination at the site pertinent to the inhalation exposure route as well as potentially exposed receptors. We then explain our methods for developing specific exposure estimates for each type of contaminant. Finally, we provide a detailed description of the activity patterns that we assumed for each site receptor.

4.1 CONCEPTUAL SITE MODEL

4.1.1 SOURCES OF CONTAMINATION

There are several potential sources of contamination at the UCR site that could affect inhalation of metals in re-suspended sediments, which is the pathway of concern in this analysis. The main source of sediment contamination was caused by historical smelting activities, mainly at the Teck Cominco facility in Trail, British Columbia. This facility is located approximately 10 miles upstream from the U.S.-Canada border, and has been in operation since 1896 (USEPA, 2009a). Discharges from this facility due to smelting activities have included granulated slag[15], wastewater effluent, stack emissions, and groundwater discharge (USEPA, 2009a). Other sources of contamination to the UCR from the Trail facility include storage of solid materials such as slag and arsenic and production of phosphate fertilizer.[16] Smelting activities have also historically occurred at the Le Roi/Northport Smelter, located approximately seven miles downstream of the U.S-Canadian border (USEPA, 2009a).[17]

In addition to those mentioned above, the UCR HHRA Workplan has identified the following additional primary sources of contamination to the site: ambient atmospheric chemicals transported to and deposited at the site from global or regional atmospheric sources; industrial operations; municipal point and nonpoint sources (e.g., wastewater

[15] Granulated slag is a byproduct of smelting furnaces. It consists of sand-sized granules that contain various quantities of trace and major metals (USEPA, 2009a).

[16] According to EPA's UCR HHRA Workplan, uranium was likely present in phosphate ores used in Trail fertilizer production and historically released into the river as phosphogypsum waste (USEPA, 2009a).

[17] This gold and copper smelter was in operation between 1896 and 1921. Emissions from this facility include slag discharges to the UCR, seepage and surface runoff of stored materials, stack emissions as well as effluent discharges and accidental spills (USEPA, 2009a).

treatment plants or storm water runoff from local communities); and agricultural nonpoint sources (USEPA, 2009a).

4.1.2 TRANSPORT AND FATE MECHANISMS FOR INHALATION EXPOSURES

Contamination emitted into the UCR from the sources outlined above could have led to contamination of sediments or soils through dissolution in surface water, adsorption to particles and weathering of slag concentration (USEPA, 2009a). The main mechanism for inhalation exposures of interest in this analysis is through re-suspension of these chemicals adsorbed to sediments or soils. This is most likely to occur in large exposed areas of contaminated sediment that could be subjected to HWEs, especially during the drawdown period at the reservoir.

4.1.3 AREAS OF INTEREST

For the purposes of this analysis, we focused on the three sampling sites along the UCR utilized by USGS. The sites were selected based on their proximity to Lake Roosevelt, the availability of AC power, their relative lack of local source contamination and the likelihood of security (USGS, 2003). The sampling sites are located at:

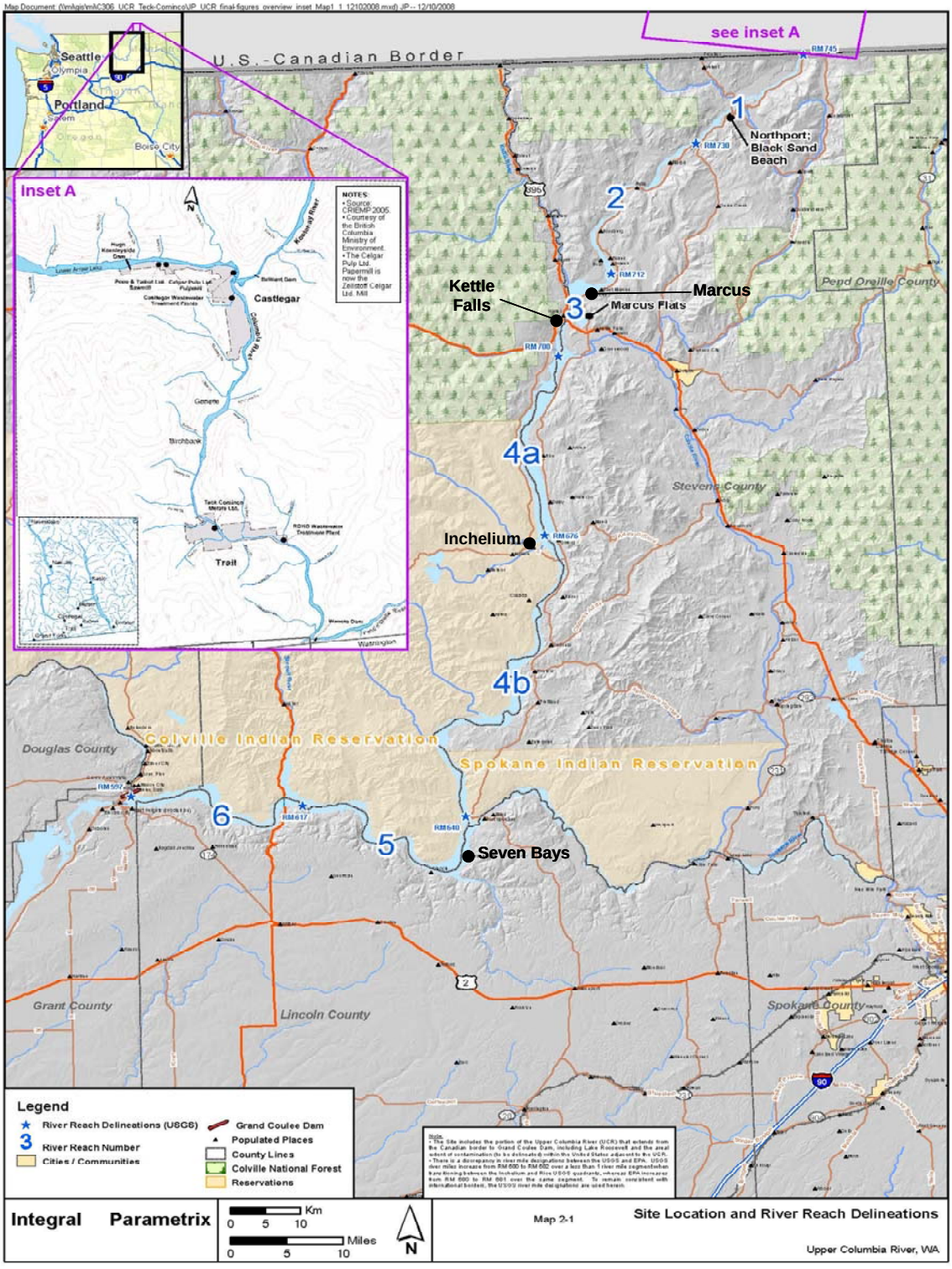
1. **Seven Bays Marina** (River Mile (RM) 635) – at a campground overlooking Lake Roosevelt;
2. **Inchelium** (RM 675) – on the roof of a one-story recreational facilities building;
3. **(a) Kettle Falls** (RM 698) – in the back yard of private residence overlooking Lake Roosevelt; and

   (b) Marcus (RM 710) – on the edge of a large open field in the town's central park.

The third site (referred to generally as Marcus) is actually a combination of the Kettle Falls and Marcus sites. In 2002 the monitor was located at Kettle Falls. The monitor was moved to Marcus at the end of the 2002 sampling season because of safety concerns (USGS, 2004). Due to the close proximity of these monitoring locations, we grouped Kettle Falls and Marcus monitoring data together and report only one set of results.

We report risk estimates for each of the three monitoring sites individually, as they are separated by large distances (40 river miles separate the Seven Bays Marina from the Inchelium site and 35 river miles separate the Inchelium and Marcus sites). Since we do not posses information on the specific time-activity patterns of receptors at the site, these risk estimates assume that a receptor would be exposed exclusively to the concentrations found at that monitoring location.

FIGURE 2. MAP OF UCR SITE



Source: HHRA Workplan, Map 2-1. Sampling sites labeled with black dots (added by IEc).

4.1.4 EXPOSED POPULATION

There are several different populations potentially exposed to PM concentrations at or near the monitoring locations at the UCR site. We did not include tribal receptors in our assessment, as a comprehensive survey is currently underway to characterize tribal exposures at the site. Therefore, we identified three types of non-tribal receptors based on the site uses outlined in Section 1.1: local residents, park employees, and park visitors. From these population groups we identified seven specific receptors: adult and child residents living in close proximity to the site, full-time archaeologists, seasonal (temporary) archaeologists, full-time maintenance workers, visiting adult recreators, and visiting children recreators.

Residents include all adults and children living in homes in close proximity to the UCR site. These residents may be exposed to wind-blown sediments at their homes, and in addition may frequent the shores of the UCR for recreational activities. The occupational receptors are individuals who travel to the site for their daily work and therefore could be exposed while performing tasks on the shores of the lake. Finally, the numerous campgrounds, beaches, boat launches and picnic areas draw recreational visitors to the UCR site, where they may be exposed to re-suspended sediments. We describe activity patterns for each receptor in more detail in section 4.5, including parameters for exposure assessment.

4.2 EVALUATING EXPOSURE TO NON-LEAD METALS

We assessed inhalation exposure to non-lead metals using the inhalation dosimetry methodology presented in EPA's *Risk Assessment Guidance for Superfund (RAGS) Part F: Inhalation Risk Assessment* (hereafter, "RAGS F") (USEPA, 2009c). We present below the methods we used to determine the concentration of contaminants to which receptors would be exposed at the site.

4.2.1 APPROACH FOR CALCULATING EXPOSURE CONCENTRATIONS

According to RAGS F, the approach for calculating inhalation exposures involves the estimation of exposure concentrations (ECs) for each receptor exposed to contaminants through the inhalation pathway. ECs are time-weighted average concentrations derived from the measured contaminant concentrations in air, adjusted based on the characteristics of the exposure scenario being evaluated. The EC is then combined with toxicity data for the particular contaminant to assess the cancer risk or non-cancer hazard to receptors at the site (see Chapter 6).

We calculated ECs for each receptor using Equation 4-1 below. The same general equation is used to calculate the ECs for carcinogens and to evaluate non-cancer effects (both chronic and sub-chronic). However, the inputs to this equation differ depending on the specific exposure scenario and type of health effect, as described below.

Equation 4-1: EC = (CA x ET x EF x ED) / AT

Where:

EC = Exposure Concentration ($\mu g/m^3$). The time-weighted average concentration based on the characteristics of the exposure scenario.

CA = Concentration of the metal in the air ($\mu g/m^3$) to which the person is exposed.

ET = Exposure time (hours/day). Describes how long a person is likely to be exposed over the course of a typical day.

EF = Exposure frequency (days/year). Describes how often a person is likely to be exposed over the course of a typical year. If the exposure period is less than one year, exposure frequency is expressed in days/week.

ED = Exposure duration (years). Describes how long a person is likely to be exposed during their lifetime. If the exposure period is less than one year, exposure duration is expressed in weeks.

AT = Averaging time (hours). Specifies the length of time over which the time-weighted average concentration is calculated. For carcinogens, exposure is averaged over the course of a lifetime; when evaluating non-cancer effects, the concentration is averaged over the ED (i.e., AT = ED).

We provide our assumed values each of these parameters for the specific receptors in section 4.5.

4.2.2 ESTIMATING THE CONTAMINANT CONCENTRATIONS IN AIR

We calculated the concentration in air (CA) for each contaminant for use in the above equation. At Superfund sites, risk assessment is generally based on an estimate of reasonable maximum exposure (RME) at the site (USEPA, 1989). This is the highest exposure that is reasonably expected to occur at a site. RME estimates combine upper-end estimates of exposure parameters with a conservative estimate of the mean concentration to which the receptors will be exposed. Due to the uncertainty associated with any estimate of exposure concentration, EPA recommends the use of the 95 percent upper confidence limit (UCL) on the mean (hereafter, "95UCL") as the concentration estimate (USEPA, 1989).

We calculated the 95UCL for each non-lead contaminant of concern using EPA's ProUCL software.[18] Each 95UCL was calculated using the statistical method recommended by ProUCL for that particular contaminant, based on the properties of the underlying data. We calculated a value for each contaminant and sampling location, and use this as the CA term in Equation 4-2. The 95UCLs used for each site and exposure period are summarized below in Table 3.[19]

[18] Available at: http://www.epa.gov/esd/tsc/TSC_form.htm.

[19] See Appendix C for detailed information on the 95UCL concentrations and calculation method.

TABLE 3. CONTAMINANT CONCENTRATIONS IN AIR SUMMARY (ALL VALUES IN µg/m^3)

	ALL DATA (CHRONIC EXPOSURE SCENARIO)			SUMMER DATA (SUB-CHRONIC EXPOSURE SCENARIO)			DRAWDOWN DATA (SUB-CHRONIC EXPOSURE SCENARIO)		
	INCHELIUM	MARCUS	SEVEN BAYS	INCHELIUM	MARCUS	SEVEN BAYS	INCHELIUM	MARCUS	SEVEN BAYS
Arsenic	0.0005	0.00075	0.0003	0.0008	0.00074	0.00037	0.00046	0.00077	0.00028
Barium	0.15	0.016	0.18	0.025	0.025	0.019	0.022	0.015	0.095
Cadmium	0.00014	0.00034	0.000067	0.00012	0.00036	0.000068	0.00015	0.00035	0.00007
Chromium	0.0019	0.0024	0.0013	0.0027	0.0015	0.0016	0.0021	0.0029	0.0014
Cobalt	0.00025	0.00019	0.00026	0.00028	0.00015	0.00027	0.00025	0.00021	0.00027
Lead	0.0022	0.0052	0.0015	0.0019	0.0033	0.0012	0.0024	0.0058	0.0014
Manganese	0.012	0.0088	0.014	0.015	0.0086	0.014	0.0098	0.007	0.015
Nickel	0.00054	0.00044	0.00043	0.00058	0.00031	0.00046	0.00075	0.00043	0.00044
Thorium	0.00012	0.000061	0.00012	0.00013	0.000063	0.00021	0.00012	0.000057	0.00011
Vanadium	0.0018	0.0011	0.0016	0.0022	0.00088	0.0016	0.0016	0.00096	0.0013
Note: Concentrations are rounded to two significant figures.									

For the chronic exposures (residents and full-time employees), we assumed that the receptor would be exposed to concentrations at the site year-round. We thus calculated the 95UCL using all of the samples taken at each monitoring site from 2002 to 2005. We excluded data from 2006 monitoring because these data are most representative of acute events (the samplers only operated when wind speeds exceeded a specific threshold level). Since maximum concentrations of the COPCs did not exceed any acute toxicity values, as described in Chapter 3, we did not evaluate acute exposures further. For sub-chronic exposures (seasonal employees and park visitors), we included only the data representative of the period in which the sub-chronic exposure occurred (e.g., summer or drawdown). For these periods, we used ProUCL to calculate 95UCLs based on observations only in the specific months included in that exposure period at each site from 2002 to 2005. [20]

4.3 EVALUATING EXPOSURE TO RADIONUCLIDES

We assess elevated cancer risk due to exposure of radionuclides differently than other non-lead metals. The approach for estimating radiation exposure due to inhalation is described below.

4.3.1 APPROACH FOR ESTIMATING TOTAL DOSE

Exposure to radionuclides is based on the total lifetime cumulative dose that is inhaled, expressed in pCi. Exposure is thus a measure of total intake (TI) in pCi. We estimated total intake using the following equation:

Equation 4-2: $TR = CA \times ET \times EF \times [(IR_C \times ED_C) + (IR_A \times ED_A)]$ [21]

Where:

TR	=	Total radioactivity inhaled (pCi).
CA	=	Concentration of the radionuclide in air (pCi/m^3).
ET	=	Exposure time (hours/day).
EF	=	Exposure frequency (days/year).
IR_C	=	Inhalation rate for children (m^3/hour).
ED_C	=	Exposure duration for children (years).
IR_A	=	Inhalation rate for adults (m^3/hour).
ED_A	=	Exposure duration for adults (years).

We provide our assumed values for each of these parameters in section 4.5.

[20] We use all observations for May through September for summer, and all observations for March through June for drawdown.

[21] Note that inhalation rate and exposure duration differs between adults and children and therefore we include terms for both in this equation for exposure scenarios that include children (residents and park visitors). For the occupational scenarios, we included a single adult inhalation rate and exposure duration.

4.3.2 CONTAMINANT CONCENTRATIONS IN AIR

Thorium is the only radionuclide that was identified as a COPC in our screening analysis. Therefore, we calculated the thorium CA (pCi/m^3) by first taking the 95UCL calculated using ProUCL, as described in Section 4.2. We then multiplied the 95UCL by the specific activity for Th-232, the amount of radioactivity per unit mass, to convert the $\mu g/m^3$ concentration to pCi/m^3.[22] We repeated this process for each monitoring location. We also calculated separate concentrations for drawdown and summer periods as we did for the other non-lead metals analyses, and used these for the radionuclide exposure assessment for seasonal archaeologists and park visitors, respectively.

4.4 EVALUATING EXPOSURE TO LEAD

Exposure to lead is evaluated differently than the other metals in our analysis. Lead is widespread in the environment and exposure can occur by multiple pathways. Thus, lead exposure generally includes all pathways rather than just the analysis-specific exposure pathway. Health effects in humans related to lead have traditionally been described in terms of BLLs, and consequently lead exposure is typically assessed using an uptake-biokinetic model in order to relate environmental levels to BLLs. We use two models to assess exposure to lead. For children aged 0-7, we used the IEUBK model. We also use the ALM to assess BLLs of women of childbearing age and the resulting predicted BLLs for fetuses in utero. For both models, we use an estimate of the mean concentration rather than the 95UCL, since the models use central tendency inputs to calculate an estimate of the geometric mean of the BLLs in the population of children exposed at the site. Assuming the distribution of BLLs is lognormal, the models use an assumption of the variability among children (specified by the geometric standard deviation (GSD)) to estimate an expected distribution of BLLs. The probability of exceeding the target BLL of 10 µg/dL can then be calculated based on this distribution.

4.4.1 IEUBK

The IEUBK model predicts the likely range of BLLs in children exposed to a set of environmental lead levels. The model combines data on lead levels in soil, water, air and diet and intake levels to estimate the central tendency of the distribution of blood lead values that might occur in a population of children exposed to the specified conditions. To date, sampling data has been collected in air, beach sediment and fish tissue. Therefore, we assessed BLLs in children exposed to these media at the site along with default assumptions about lead exposure in other media.

[22] We used the activity for Th-232 because over 99 percent of all naturally occurring Thorium is Th-232. According to the UCR HHRA Workplan, fertilizer production at the Trail facility could have resulted in uranium-containing waste products at the site. Therefore, we examined the decay chain of Uranium (U)-238 to see if any other radioactive isotopes of Thorium (e.g., Th-230) were likely to be present, and found that the amount of decayed U-238 needed to produce risk of concern due to Thorium was not plausible.

4.4.2 ALM

The ALM predicts the blood lead level in an adult by adding the increment in blood lead that is expected to result from site-related exposures to an estimate of "baseline" BLL (that which would occur in the absence of any site-related exposures). We assessed the total BLL expected based on exposures to levels of lead at Lake Roosevelt in all of the contaminated media for which we have sampling data - air, beach sediment, and fish tissue. The basic equation for adult exposure to lead is:

Equation 4-3: $PbB = PbB_0 + [\ BKSF \times \sum (Pb_i \times IR_i \times AF_i \times EF_i / 365\ days)\ ]$

Where:

PbB	=	Geometric mean blood lead concentration in exposed population due to all contaminated media at the site (µg/dL).
PbB_0	=	Baseline geometric mean blood lead concentration (µg/dL).
BKSF	=	Biokinetic slope factor (µg/dL blood lead increase per µg/day lead absorbed).
Pb_i	=	Lead concentration in medium 'i' (µg/g for fish and sediment, µg/m^3 for air).
IR_i	=	Intake rate of medium 'i' (g/day for fish and sediment, m^3/day for air).
AF_i	=	Absolute absorption fraction for lead in medium 'i' (unitless).
EF_i	=	Exposure frequency for medium 'i' (days/year).

We estimated fetal blood lead levels using a fetal to maternal blood lead level ratio of 0.9, EPA's default recommended value for use with the ALM (USEPA, 2003b). We assumed that the resulting fetal BLLs would be lognormally distributed, with a GSD of 1.8 µg/dL.[23]

4.4.3 CONTAMINANT CONCENTRATIONS

For both models, we used an estimate of the arithmetic mean concentration rather than the 95UCL, since the models use central tendency inputs to calculate an estimate of the geometric mean of the BLLs in the population of children exposed at the site (USEPA, 2003b, USEPA, 2007). When calculating the arithmetic mean concentrations, we assumed that non-detect samples were equal to one-half the reported detection limit.

4.5 EXPOSURE PARAMETERS FOR KEY RECEPTORS

Table 4 below presents specific exposure parameters for each of the key receptors that we identified at the site. Below we provide a description of the receptors and the parameters used for each type of contaminant in our analysis.

[23] This value is consistent with the UCR HHRA Workplan, which cites a document providing updated default baseline parameters for the ALM (SRC, 2006).

TABLE 4. EXPOSURE PARAMETERS SUMMARY

	OUTDOOR WORKERS			LOCAL RESIDENTS		PARK VISITORS	
	FULL-TIME ARCHAEOLOGIST	SEASONAL ARCHAEOLOGIST	FULL-TIME MAINTENANCE	ADULTS	CHILDREN	ADULTS	CHILDREN
CA = contaminant concentration in air ($\mu g/m^3$)	Contaminant-specific	Contaminant-specific	Contaminant-specific	Contaminant-specific	Contaminant-specific	Contaminant-specific	Contaminant-specific
ET = Exposure Time (hours/day)	9 [1]	9 [1]	9 [1]	24	24	8 [6]	8 [6]
EF = Exposure Frequency (days/year)	80 [2]	65 [3]	200 [4]	350 [5]	350 [5]	14 [6]	14 [6]
ED = Exposure Duration (years)	20 [1]	1 [7]	25 [1]	30 [5]	6 [5]	30 [5]	6 [5]
AT = Averaging Time – Noncancer (hours)	175,200	8,760	219,000	262,800	52,560	262,800	52,560
AT = Averaging Time – Cancer (hours)	613,200	613,200	613,200	613,200	613,200	613,200	613,200
Inhalation rate (For radionuclide exposure only) (m^3/hr)[8]	1.3 m^3/hr [9]	1.3 m^3/hr [9]	1.3 m^3/hr [9]	0.55 m^3/hr [10]	0.3 m^3/hr [11]	0.55 m^3/hr [10]	0.3 m^3/hr [11]
Lead Analysis	For analysis of lead, we use the IEUBK and ALM models. We use the default parameters developed for the IEUBK model, but update concentrations in air, sediment, and fish to site-specific values. For the ALM, we use default values developed by EPA, but use site-specific concentrations of lead in air, sediment, and fish. For the analysis lead exposure through inhalation, we assume the same adult breathing rate we use for the radionuclide analysis, 13.2 m^3/day. [10] See sections 4.4 and 4.5.3 for more details on the two models and parameters.						

[1] Based on interviews with archaeologists and maintenance workers at Lake Roosevelt (IEc, 2007a & b).

[2] Assumes a 5 day work week for approximately 9 months of the year, 40 percent of which is spent in the field. Estimate based on interviews with archaeologists at Lake Roosevelt (IEc, 2007a).

[3] Assumes a 5 day work week for approximately 3 months of the year, with all time spent in the field. Estimate based on professional judgment.

[4] Assumes a 5 day work week for 12 months a year (50 weeks, vacation adjusted), 4 days per week spent outdoors. Estimate based on interviews with maintenance workers at Lake Roosevelt (IEc, 2007b).

[5] USEPA, 1991a.

[6] Assumes park visitors are at Lake Roosevelt for two weeks a year and are exposed for eight hours a day, based on professional judgment.

[7] Assumes seasonal staff is a one year commitment and most do not return, based on interviews with archaeologists at Lake Roosevelt (IEc, 2007a).

[8] Exposure to radionuclides is calculated as the radionuclide concentration in mass per m^3 times the receptor's inhalation rate, ET, EF, and ED, and the specific activity of the radionuclide (in pCi per unit mass) to obtain a total estimate of exposure in pCi.

[9] Hourly average for outdoor workers (USEPA, 1997a).

[10] Average of adult male and female rates (USEPA, 1997a).

[11] Weighted average of rates for children under the age of six (USEPA, 1997a).

4.5.1 NON-LEAD METALS

Local Residents

As described in Section 1.1, there is a population of individuals that live in close proximity to Lake Roosevelt. These local residents live in dwellings that have the potential to be exposed to wind-blown sediments from the UCR. Therefore, these individuals could be exposed during their daily activities at home. Since they live near the site, it is also likely that they frequent the shores of Lake Roosevelt for recreational purposes as well. Therefore, we assumed that these receptors would be exposed to site contamination 24 hours per day, seven days per week, with the exception of a two week vacation taken each year away from home. The risk estimates for residents are based on period of time as a child (6 years) and as an adult (24 years), for a total exposure duration of 30 years.[24]

This exposure scenario follows a chronic duration and pattern according to Figure 2 of RAGS F, in that the duration consists of several years and the frequency is generally as frequent as a chronic toxicity test or occupational study. Therefore, we used Equation 4-1 above to calculate ECs for each carcinogen and non-carcinogen along with IURs to assess cancer risk and chronic toxicity values to assess non-cancer hazard at the site for this receptor.

Park Empolyees

The LRNRA employs several individuals, as described in Section 1.1. These receptors could potentially be exposed to airborne contaminants at the site based on their occupational activities. We focused on two receptors with the highest potential exposure levels: archaeologists and maintenance workers. These worker's spend a large proportion of their time working outside at the site and therefore could frequently be exposed to wind-blown sediments. We conducted interviews with a handful of archaeologists and maintenance workers as part of a preliminary risk assessment of sediment exposures for the National Park Service (IEc, 2007a, b & c). We based our description of the activities undertaken by these employees as well as the amount of time spent at the site on these interviews.

Full-Time Archaeologists: The Park employs a number of full-time archaeologists who work at the site year round. These individuals work to ensure that park activities do not infringe on cultural artifacts. In some cases, this involves digging in the sediment to test for the presence of artifacts and may lead to full excavation activities.

Full-time archaeologists at the site typically work five days per week. However, there are only nine months out of the year during which they work outside at the site (March through November). Furthermore, we assumed that these workers spend 40 percent of their time in the field during this nine-month period. Therefore, we assumed that full-time archeologists are exposed to windblown sediment at the site for a total of 80 days

[24] This is the 90th percentile estimate of the time spent in one residence (USEPA, 1991a).

per year.[25] We assumed that archaeologists spend 9 hours per day outside during these days. The average tenure for a full-time park archaeologist is approximately 20 years. Therefore, we used this as the exposure duration.

The exposure pattern of a full-time archaeologist follows that of a chronic exposure in that the duration consists of several years and the frequency is generally as frequent as a chronic toxicity test or occupational study. Therefore, we assessed this receptor as a chronic exposure, using Equation 4-1 along with IURs to assess cancer risks and with chronic toxicity values to assess non-cancer hazard.

Seasonal Archaeologists: During the drawdown period, large amounts of sediment are exposed at the lake. The Park hires a group of seasonal archaeologists during this time to help with artifact excavations. Because these workers are only hired for a short period of time specifically to perform field work, we assumed that they spend 100 percent of their time outside at the site and therefore are exposed to windblown sediments for 65 days per year, for 9 hours per day. According to the interviews that we conducted, these archaeologists do not typically return to the site from year to year. Therefore, the exposure duration assumed for these workers was one year.

Seasonal archaeologists are only exposed to site contamination for a single three-month period. This exposure duration is consistent with that of a subchronic exposure, according to RAGS F, since it lasts for several weeks. Therefore, we assessed exposures to this receptor using Equation 4-1 to calculate subchronic ECs. We used IURs to assess cancer risk and used subchronic toxicity values, when available, to assess non-cancer hazards.[26]

Maintenance Workers: The Park employs full-time maintenance workers at the site to perform a variety of tasks such as plumbing and electrical work, road work, tree thinning in campgrounds, and removing grasses from swim areas. During the drawdown period, these individuals spend additional time outside near the water performing projects such as cleaning boat ramps and checking docks and anchor systems. We assumed that these employees could be exposed to contaminants in air at the site 4 days per week for 50 weeks per year, for a total of 200 days of exposure per year. We assumed that these employees spend 9 hours per day outside during these days. Based on interview responses, we determined that the average tenure for this position is about 25 years, which we used as the exposure duration.

The exposure pattern of a full-time maintenance worker follows that of a chronic exposure in that the duration consists of several years and the frequency is generally as frequent as a chronic toxicity test or occupational study. Therefore, we assessed this receptor as a chronic exposure, using Equation 4-1 along with IURs to assess cancer risks and with chronic toxicity values to assess non-cancer hazard.

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[25] We assumed that any vacation time taken by archaeologists would occur during the three-month period in which they do not perform field work.

[26] When subchronic toxicity values were not available for a contaminant, we used chronic toxicity values to evaluate hazards, in accordance with RAGS F (USEPA, 2009c).

Park Visitors

The shores of Lake Roosevelt draw a large number of visitors every year, most of whom visit during the summer months. We assumed that a typical visitor will come to the area for two weeks per year (14 days), and that the visitor will return year after year for the same two-week vacation for 30 years. During the vacation, the visitor is not likely to be exposed continually to the windblown sediment concentrations at the site, as some time would potentially be spent away from the shores (i.e., visitors might stay at a hotel further away from the lake). Therefore, during the two week stay, we assumed that a vacationer will be outdoors at the site for an average of 8 hours per day. Visitors may include adults and children, but we assumed the same exposure parameters regardless of age and therefore only present a single risk estimate for the park visitor receptor.

When evaluating cancer risks for park visitors, we used Equation 4-1 along with IURs for each carcinogen. However, to determine how to evaluate non-cancer hazard for these receptors, we utilized the recommended process outlined in RAGS F, Figure 2 (USEPA, 2009c). We first assessed the duration of the exposure. Since the exposure occurs every year for 30 years, it is consistent with a chronic exposure. However, the frequency does not match that of a chronic toxicity test or occupational study, because it only occurs two weeks per year. Therefore, we assessed each two week exposure period as a separate subchronic event, consistent with the recommendations in RAGS F. We used Equation 4-1 to calculate subchronic ECs and used subchronic toxicity data, when available, to calculate non-cancer hazards.[27]

4.5.2 RADIONUCLIDES

We generally used the same exposure parameters for the assessment of both radionuclides and non-lead metals. The main difference is that exposure to radiation is dependent on total concentration inhaled (not averaged over the period of exposure) and thus varies with inhalation rate. Exposure time, frequency, and duration for all receptors are the same in our radionuclide analysis as they are in our analysis of non-lead metals, with the exception of residents and park visitors. For these receptors we specified separate exposure durations for adults and children, because their inhalation rates differ. The exposure duration for children is 6 years and the exposure duration for adults is 24 years, for a total lifetime exposure of 30 years. We used an inhalation rate for adult residents and visitors of 0.55 m^3/hour.[28] For children, we assumed an inhalation rate of 0.3 m^3/hour.[29] All occupational receptors have the same inhalation rate of 1.3 m^3/hour.[30]

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[27] When subchronic toxicity values were not available for a contaminant, we used chronic toxicity values to evaluate hazards, in accordance with RAGS F (USEPA, 2009c).

[28] Average of adult male and female inhalation rates, from U.S. EPA's Exposure Factors Handbook, Volume I, page 5-24 (USEPA, 1997a).

[29] Weighted average of inhalation rates for children under the age of six, from U.S. EPA's Exposure Factors Handbook, Volume I, page 5-24 (USEPA, 1997a).

[30] Hourly average for outdoor workers, from U.S. EPA's Exposure Factors Handbook, Volume I, page 5-24 (USEPA, 1997a).

4.5.3 LEAD

To assess lead hazards, we used site-specific lead concentration data in for media sampled to date which include air, beach sediment, and fish tissue. We used the highest annual mean concentration measured at the site for each media.[31] In addition, we ran the two biokinetic models for the residential scenario, as this is the most highly exposed receptors. Parameters specific to each of the two lead models are described in further detail below.

IEUBK

We used the default IEUBK values for all concentration and exposure pattern assumptions for all media, with a few exceptions. For the outdoor air pathway, we replaced the default concentration with the mean concentration from the samples taken from the Marcus monitor, which had the highest annual mean (0.0052 µg/m^3).[32] We also changed the default dietary patterns in the model to reflect the potential presence of fish from Lake Roosevelt in a child's diet. Our inputs reflect a diet where 12.9 percent of all meat in the diet consists of fish from the site, and that this fish has a lead concentration of 0.112 µg/g.[33] We also input site-specific values for an average outdoor soil lead concentration of 230 µg/g, the highest mean beach sediment concentration at the site from EPA's Phase I sediment sampling effort (USEPA, 2006a).

ALM

For the ALM, we used default recommended values for several parameters, such as the BKSF, the AF, and the ratio of fetal-to-maternal BLL (USEPA, 2003b). For the baseline BLL (1.0 µg/dL) and GSD (1.8 µg/dL), we used the values listed in Table 5-14 of the UCR HHRA Workplan, from the SRC update of default parameters for the ALM (SRC, 2006). Because we only examined the residential scenario for lead, we used the same exposure parameters we defined for residents for the non-lead metal and radionuclide analyses. For instance, to assess the inhalation pathway, we used the same adult residential inhalation rate that was employed in the radionuclide analysis (13.2 m^3/day). For consumption of fish, we used the same lead concentration in fish that was used for the IEUBK analysis (0.112 µg/g). We also assumed an intake rate for fish of 26 g/day.[34] For sediment, we used the same concentration as the one input into the IEUBK model (230 µg/g).

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[31] Non-detect samples were assigned a concentration equal to one-half the reported detection limit when calculating the mean.

[32] Note that this concentration is lower than the default value (0.1 µg/m^3).

[33] The estimate of the percentage of total meat intake consisting of fish was derived from an assumption of a consumption of 13 grams per day of fish from Lake Roosevelt, calculated from the Lake Roosevelt Washington State Department of Health (WSDH) angler survey (WSDH, 1997) and a WSDH survey of Lake Whatcom anglers (WSDH, 2001). This was divided by the total meat intake assumed in the IEUBK model of 101 grams/day. The concentration in fish tissue represents the highest mean concentration of lead in fish tissue from EPA's Phase I fish tissue sampling effort for two species of fish caught and consumed at the lake, trout and walleye (USEPA, 2006b).

[34] Value calculated from the Lake Roosevelt WSDH angler survey (WSDH, 1997) and a WSDH survey of Lake Whatcom anglers (WSDH, 2001).

CHAPTER 5 | CONCENTRATION-RESPONSE ASSESSMENT

5.1 TOXICITY VALUES FOR NON-LEAD METALS

In order to estimate the cancer risk and non-cancer hazard from inhaled metals at the site, we selected toxicity values for each of the COPCs identified through our screening analysis (see Chapter 3). As described in Chapter 4, we assessed both chronic and subchronic exposure scenarios. Therefore, we selected available chronic and subchronic non-cancer inhalation toxicity values (e.g., RfCs) for analyzing non-cancer risks and IURs for our cancer risk analysis. Table 5-1 below presents the toxicity values that we applied during the risk characterization portion of the risk assessment.

TABLE 5. TOXICITY VALUES USED IN THE RISK ASSESSMENT

METAL	CHRONIC NON-CANCER TOXICITY VALUE		SUBCHRONIC NON-CANCER TOXICITY VALUE		IUR	
	µg/m^3	SOURCE/TYPE	µg/m^3	SOURCE/TYPE	(µg/m^3)$^{-1}$	SOURCE
Arsenic	0.015	C/Chronic REL	-	-	0.0043	I
Barium	0.5	H	-	-	-	-
Cadmium	0.01	A/Chronic MRL	-	-	0.0018	I
Chromium (total)[1]	-	-	-	-	0.012	I
Chromium (VI)[2]	0.1	I/RfC	0.3	A/Intermediate MRL	-	-
Chromium (III)	-	-	5	A/Intermediate MRL	-	-
Cobalt	0.006	P/p-RfC	0.02	P/p-RfC	0.009	P
Lead[3]	N/A	-	N/A	-	N/A	-
Manganese	0.05	I/RfC	0.17	C/8-hr REL	-	-
Nickel	0.05	C/Chronic REL	0.2	A/Intermediate MRL	0.00024	I
Thorium	-	-	-	-	4.33E-08[4]	H
Vanadium	0.007	P/p-RfC	0.1	P/p-RfC	0.0083	P

Key: A = ATSDR; C = CalEPA; H = HEAST; I = IRIS; MRL = Minimal Risk Level; P = Provisional Peer Reviewed Toxicity Values; p-RfC = Provisional Reference Concentration; REL = Reference Exposure Level; RfC = Reference Concentration.

[1] The IUR on IRIS is published in the chromium (VI) profile. However, the background documentation for this toxicity value indicates that it is in fact based on occupational studies with workers exposed to total chromium. Therefore, we have applied this toxicity value to the total chromium concentrations measured at the site.

[2] Toxicity values are for chromium (VI) particulates.

[3] Lead hazards were assessed through the IEUBK model and the ALM (see Chapter 4).

[4] Thorium was assessed through the cancer risks due to radiation (see Chapter 4). The value listed is the lifetime excess total cancer risk per unit of exposure (risk/pCi).

In order to select the values in Table 5-1, we utilized the inhalation toxicity data collected during the Hazard Identification phase of our risk assessment (see Chapter 2). If multiple toxicity values were available for a particular contaminant, we first applied the Superfund program's recommended hierarchy to make our selection (USEPA, 2003a). Therefore, we preferentially selected values from IRIS (Tier 1). If values were not available from IRIS, we then sought PPRTVs (Tier 2), which are developed by EPA's Superfund Health Risk Technical Support Center (STSC). If PPRTVs were not available, we obtained toxicity values from other sources that are classified by EPA as Tier 3, such as ATSDR's MRLs, CalEPA's RELs, or EPA's HEAST values (USEPA, 1997b).

However, for nickel and cadmium, multiple chronic non-cancer toxicity values were available from within the same tier of the hierarchy (Tier 3). An ATSDR MRL as well as a CalEPA REL was available for these two metals. For both nickel and cadmium, the MRL and REL values appeared to be of a similar quality, in that they were based on the same type of data (human epidemiological data for cadmium and rat studies for nickel) and were published fairly recently (i.e., within the past 10 years). Therefore, we selected the more conservative of the two values.[35]

5.2 TOXICITY VALUES FOR RADIONUCLIDES

Thorium was the only radionuclide that did not pass our initial screen. Therefore, we assessed cancer risks for this contaminant using slope factors published in Federal Guidance Report Number 13 (USEPA, 2001). To date at the site, no information on specific radionuclide concentrations has been collected. Therefore, we assumed that the entire concentration of Thorium consisted of the 232 isotope (Th-232), as this is the most common naturally occurring isotope (greater than 99 percent of naturally occurring Thorium is Th-232).[36] The specific activity for this isotope is low, 1.1E-07 Ci/g, which indicates that it is not highly radioactive.[37] The cancer slope factor for this isotope from the HEAST table is 4.33E-08 per pCi.

As noted in Section 4.1.1, the production of fertilizer took place at the Trail facility, and it is likely that uranium was present in the phosphate ores used and released into the river as phosphogypsum waste (USEPA, 2009a). The UCR HHRA Workplan notes that several radionuclides are part of the decay chain for uranium (U-238). Therefore, EPA includes all radioactive isotopes in the decay chain for U-238 as contaminants of interest, which includes Th-230, a thorium isotope with much higher levels of radioactivity than Th-232.[38] We do not have data on the specific isotopes present in the samples.

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[35] We selected the CalEPA REL for nickel (0.05 $\mu g/m^3$) instead of the ATSDR MRL of 0.09 $\mu g/m^3$. For cadmium, the more conservative value was the ATSDR MRL (0.01 $\mu g/m^3$). The CalEPA chronic REL for this metal is 0.02 $\mu g/m^3$.

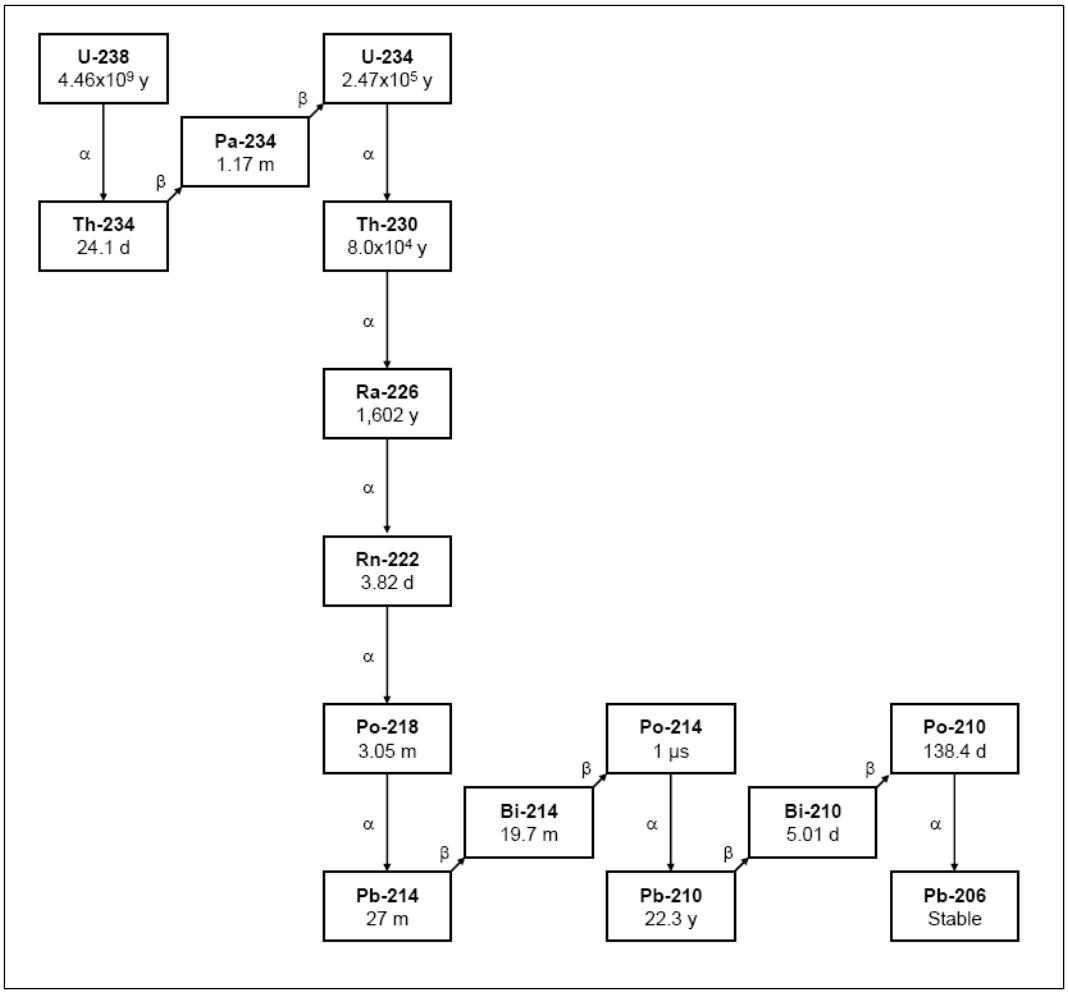
[36] http://www.atsdr.cdc.gov/toxprofiles/tp147-c3.pdf.

[37] The specific activity for Th-232 can be found at: http://www.iem-inc.com/toolspa.html.

[38] The specific activity level for Th-230 is 2.0E-02 Ci/g (http://www.ead.anl.gov/pub/doc/Thorium.pdf).

We assumed that all of the thorium detected at the site is Th-232, despite the historical release of U-238, for two reasons: 1) the levels of uranium detected in the air samples are very low (below health screening levels; see Chapter 3); and 2) the half-life of the U-238 isotope is very long (4.5 billion years), as is the half-life of the U-234 isotope (233,000 years), both of which would need to decay first to produce Th-230. The small mass of uranium present coupled with extremely slow production of Th-230 suggests to us that amounts of Th-230 present are likely to be far below levels of concern for the air pathway, despite the fact that Th-230 has a high level of radioactivity. The decay chain for U-238 is shown below in Figure 3.

FIGURE 3. URANIUM-238 DECAY CHAIN



Source: http://www.health.state.ny.us/environmental/radiological/radon/chain.htm [As cited in HHRA Workplan, Figure 5-2]

5.3 LEAD TOXICITY

As described in Section 4.4, we have assessed lead exposures to children and fetuses using the IEUBK model and the ALM. These models predict the distribution of BLLs of children that are exposed to contamination at the site. In order to determine whether lead exposures are of concern at a site, EPA has identified 10µg/dL as a BLL level to avoid. In addition, it has set a goal of no more than a five percent chance that a child or fetus will have a BLL above 10 µg/dL. Therefore, we have used this target benchmark to assess whether lead exposures at the site warrant concern.

CHAPTER 6 | RISK CHARACTERIZATION

6.1 APPROACH FOR ASSESSING RISKS

6.1.1 NON-LEAD METALS

Non-Cancer

The potential for non-cancer health effects is evaluated by comparing the estimated exposure level of a receptor at the site over a specific time period to a reference threshold that represents an exposure level below which it is unlikely for even sensitive populations to experience adverse health effects. The resulting ratio is known as the Hazard Quotient (HQ). EPA defines an HQ as "the ratio of a single substance exposure level of a specified time period (e.g., subchronic) to a reference dose for that substance derived from a similar exposure period" (USEPA, 1989). If an HQ exceeds one, there is a possibility that some non-cancer effects may occur. Generally, larger HQs indicate greater levels of concern. If the HQ is less than one, it is believed that there is no increased risk of non-cancer health effects at a site.

In order to evaluate non-cancer effects due to inhalation of non-lead metals in re-suspended sediment at the UCR site, we compared the time-weighted ECs (as described in Chapter 4) with chronic inhalation RfCs or other inhalation toxicity values with exposure durations matching that of the scenario being assessed (e.g., subchronic), using the following equation:

Equation 6-1: $HQ = EC/(RfC \times CF)$

Where:

EC ($\mu g/m^3$) = Exposure Concentration;

RfC (mg/m^3) = Inhalation Reference Concentration or other toxicity value that is of appropriate duration for the exposure scenario (e.g., subchronic).

CF ($\mu g/mg$) = Conversion Factor = 1000.

Cancer

The excess risk of cancer over a lifetime as a result of exposure to a particular contaminant is expressed in terms of a probability of developing cancer due to that exposure. EPA considers cancer risks between 1E-06 (i.e., 1 in 1,000,000) and 1E-04 (i.e., 1 in 10,000) to be generally acceptable (USEPA, 1991b). However, EPA evaluates this on a case-by-case basis and risks below 1E-04 may not be considered sufficiently protective and many warrant remedial action (USEPA, 2009a).

We calculated the excess risk of cancer from inhalation to non-lead, non-radionuclide metals using the following equation:

Equation 6-2: Risk = EC x IUR

Where:

EC ($\mu g/m^3$) = Exposure Concentration; and

IUR $(\mu g/m^3)^{-1}$ = Inhalation Unit Risk.

In order to calculate the excess risk of cancer from radionuclide exposure, we used the following equation:

Equation 6-3: Risk = TR x SF

Where:

TR (pCi) = Total Radioactivity Inhaled; and

SF (risk/pCi) = Slope Factor for the inhalation exposure route.

Estimating Cumulative Risks and Hazards Across Chemicals

Since the receptors at the UCR site are potentially exposed to more than one chemical simultaneously through the inhalation route, we calculated estimates of total cancer risks and non-cancer hazards. For carcinogens, we summed the chemical-specific cancer risks across all carcinogenic contaminants. For non-carcinogens, we first summed the HQs across all chemicals to calculate a Hazard Index (HI). If the HI was less than one, we assumed that non-cancer effects would not be expected to occur from any chemical individually or in combination with others. However, if the HI exceeded one, we calculated organ-specific HIs by combining HQ values for contaminants with the same target organ or tissues.

6.1.2 LEAD

We assessed cumulative risks to children and fetuses at the site due to inhalation of lead in air and ingestion of lead-contaminated fish tissue and beach sediment. For the latter two, we used data collected by EPA as part of their Phase I sampling effort (USEPA, 2006a & b).

We calculated risks for children aged 0-7 years that are potentially exposed to lead at the site using the IEUBK model. We input site-specific mean lead concentrations for each of the three media for which data were available along with site-specific exposure parameters (see Chapter 4). As a conservative first-pass approach, we used the highest sampled mean for each media as well as the exposure parameters for the residential receptor, as these are the most highly exposed individuals. If lead levels were above levels of concern, we assessed additional monitor means and receptors to identify all of the potentially affected areas. We used the IEUBK default values for all other parameters and concentrations.

Because children as well as adults are expected to be exposed to lead at the UCR site, we assessed risks to fetuses in utero due to maternal exposure using EPA's ALM. Again, we assessed exposure to inhaled lead particulates as well as exposure through fish ingestion and beach sediment, ingestion, and dermal contact. See Chapter 4 for detailed model inputs.

If greater than five percent of the population of exposed children or fetuses at the site were expected to have BLLs over 10 µg/dL, we would conclude that lead levels may warrant further evaluation. If less than five percent of children and fetuses were predicted to have BLLs above the target level, than we assumed that lead exposure was not of concern at the site.

6.2 RESULTS

6.2.1 NON-CANCER HAZARDS

The non-cancer hazards that we calculated at the site due to inhalation of contaminated particulates were below levels of concern for each individual contaminant (i.e., all HQs were below one). In addition, the HIs calculated across all of the non-lead COPCs were below one, with the highest value estimated for residents living near the Seven Bays site (with an HI of 0.91). The largest contributors to the HI were barium, manganese, and vanadium, with HQs of 0.35, 0.27 and 0.22, respectively. Table 6 below presents the total HIs for each sampling location and receptor. Appendix D provides the individual HQs for each contaminant, site and receptor combination.

TABLE 6. HAZARD INDICES FOR RECEPTORS AT THE UCR SITE DUE TO INHALATION EXPOSURE TO ALL COPCs

RECEPTOR	INCHELIUM	MARCUS	SEVEN BAYS
RESIDENT	0.86	0.47	0.91
FULL-TIME ARCHAEOLOGIST	0.07	0.04	0.08
SEASONAL ARCHAEOLOGIST	0.01	0.01	0.02
FULL-TIME MAINTENANCE WORKER	0.18	0.10	0.20
PARK VISITOR	0.08	0.07	0.06

6.2.2 CANCER RISKS

The cancer risks due to inhalation exposures at the site were within EPA's acceptable risk range (i.e., 1E-06 to 1E-04) for each individual carcinogen as well as across all carcinogens. The highest total cancer risks are experienced by residents living in proximity to the Marcus site, with risks of 1.79E-05, primarily due to exposures to chromium, arsenic, and vanadium. Table 7 below presents the total cancer risks for each sampling location and receptor. Appendix D provides the individual cancer risk estimates for each contaminant, site and receptor combination.

The total cancer risk estimates include exposure to thorium at the site, which is assumed to be the Th-232 isotope (as explained in section 5.2).

TABLE 7. TOTAL CANCER RISKS FOR RECEPTORS AT THE UCR SITE DUE TO INHALATION EXPOSURE OF PARTICLES

RECEPTOR	INCHELIUM	MARCUS	SEVEN BAYS
RESIDENT	1.8E-05	1.8E-05	1.4E-05
FULL-TIME ARCHAEOLOGIST	1.0E-06	1.0E-06	7.8E-07
SEASONAL ARCHAEOLOGIST	4.2E-08	4.7E-08	3.0E-08
FULL-TIME MAINTENANCE WORKER	3.2E-06	3.2E-06	2.4E-06
PARK VISITOR	3.1E-07	1.7E-07	2.0E-07

6.2.3 LEAD

The results of our IEUBK model run indicate that only 0.52 percent of children exposed at the site to lead via inhaled particles, fish ingestion and beach sediment would have BLLs above the level of concern, 10 µg/dL. This is well below the target of 5 percent. The predicted geometric mean BLL was 3.0 µg/dL for children aged 0-7 years living near the site.

The ALM predicted that only 0.36 percent of fetuses would have BLLs greater than 10 µg/dL if their mothers lived near the site and were exposed to lead via fish ingestion, recreating on the UCR beaches, and inhaling re-suspended sediments. Geometric mean fetal BLLs were predicted to be 2.1 µg/dL.

6.3 UNCERTAINTIES

The following section provides our assessment of the uncertainties associated with our inhalation risk estimates at the UCR site. Overall, the assumptions that we made in our analysis are likely to lead to an overestimation of risk to the average receptor at the site, rather than an underestimate. The risk estimates we report represent RME risks, which combine upper end exposure assumptions and a conservative estimate of the contaminant concentration (95UCL on the mean). We include a discussion of specific uncertainties below, first outlining those related to estimating the exposure concentrations receptors would be exposed to at the site. Second, we list uncertainties related to the toxicity values we used to assess cancer risk and non-cancer hazard at the site.

6.3.1 UNCERTAINTIES RELATED TO ESTIMATING EXPOSURE CONCENTRATIONS

The following outline the uncertainties that we have identified affecting the ECs we calculated as part of our exposure assessment at the site.

- The USGS dataset measured the concentrations in air of 33 trace metals as well as PM_{10}. However, the UCR HHRA Workplan identifies several additional contaminants of interest (COIs) not measured by USGS, such as other common and trace metals, radionuclides, semivolatile organic compounds (SVOCs), polycyclic aromatic hydrocarbons (PAHs), pesticides, and organics (e.g., polychlorinated biphenyls (PCBs)). EPA indicates that since pesticides, PAHs, SVOCs, and organic contaminants have relatively low volatility, it is possible to predict their presence from measures of PM_{10} (USEPA, 2009a). Therefore, EPA does not see this as an important data gap. We did not calculate risks from inhaled organic contaminants at the site since PM levels did not exceed our screening levels. Therefore, it is likely that the concentration of organics would be below a level of concern at the site. However, it would potentially be useful to measure the concentration of the additional metals identified as COIs by EPA (see USEPA, 2009a, Table 5-1) as well as radionuclides in the U-238 decay chain in order to fully assess risks to human health from outdoor air at the site. Therefore, it is possible that our risk estimates are underestimated, due to the omission of these contaminants.
- The USGS air monitors were placed in three locations along Lake Roosevelt. These monitors are located in three of the river reaches defined by the RI/FS for risk assessment (Reaches 3, 4, and 5). The small number of monitors may not provide sufficient information on the spatial trends in particulate air pollution at the site and do not cover all of the areas where human exposures may occur (USEPA, 2009). In addition, the data was collected over a single four-year period, which may not be sufficient to determine temporal trends of contamination in air.
- The air samples taken at the site between 2002 and 2005 consisted of 24-hour composite samples. Although the sampling frequency increased during the drawdown period, only a handful of samples actually indicate that a HWE occurred during a sampling day. Therefore, it is possible that the dataset from these years does not adequately represent the concentration of metals in air during HWEs. In 2006, the sampling strategy was altered so that the monitors were only activated when wind speeds exceeded a specific threshold level. The concentrations measured during this time are not consistently higher than other years and do not exceed acute toxicity values. Therefore, this uncertainty may not affect the overall conclusions of our risk assessment, but additional HWE sampling would be needed to confirm this.

- Our subchronic risk estimates for seasonal employees and park visitors rely upon exposure estimates derived from samples taken only during the season in which these receptors would be expected to have exposures at the site. However, during this process, we identified a temporal pattern in the data that we did not expect. We found that higher concentrations were measured in the summer months, with summertime average concentrations (May-September) often exceeding the yearly average concentrations, as well as average concentrations for the drawdown period (March through June). One possible explanation we have considered is that more sediment is re-suspended in the dry summer months compared to drawdown periods, because the greater area of exposed sediment during drawdown may be too damp to become re-suspended when subjected to wind. However, additional data is needed to more fully explore this temporal variability in exposure.
- The USGS dataset included some non-detected samples. We expect this to have a minimal effect on the majority of our risk estimates, as the proportion of detected samples was very high for the majority of the chemicals have a high percentage of detects (the average is approximately 87 percent detected). The exceptions are barium and thorium (for which roughly 60-70 percent of samples were detected). Therefore, the risk estimates for these two metals may be associated with greater uncertainty.

6.3.2 UNCERTAINTIES RELATED TO TOXICITY ASSESSMENT

Below, we explain the uncertainties related to assessing the potency of the metals that we included in our analysis.

- We were unable to identify inhalation toxicity values for 18 contaminants detected at the site. This included lithium, phosphorus, scandium, titanium, iron, zinc, gallium, rubidium, strontium, niobium, molybdenum, silver, antimony, cesium, lanthanum, cerium, thallium, and bismuth. Therefore, we were unable to calculate quantitative risk estimates for these chemicals. It is possible that the omission of these metals from our analysis may result in an underestimate of risk from windblown sediment at the site, if one or more of these exhibit toxicity via inhalation at levels found along the UCR.
- We were unable to identify subchronic inhalation toxicity values for three COPCs - arsenic, barium, and cadmium. We assessed the subchronic exposure scenarios for these metals using chronic non-cancer toxicity values, as per EPA guidance (USEPA, 2009b). The use of chronic toxicity values may lead to overestimates of risk, since these tend to be lower than subchronic toxicity values.
- As described in Section 5.2, we assumed that all of the thorium detected at the site consisted of the Th-232 isotope, since specific radionuclide concentrations were not measured at the site, and Th-232 is the most common isotope found in the environment. However, because uranium-containing fertilizer production waste was released to the site, it is possible that other, more highly radioactive thorium

isotopes are present (e.g., Th-230). Therefore our assumption could potentially underestimate risk. However, the small mass of uranium present at the site (concentrations did not exceed health-based screening levels) coupled with an extremely long half-life of U-238 (billions of years) would result in very minute amounts of Th-230 being present, even if we assume all the uranium at the site is U-238. This suggests to us that amounts of Th-230 present are likely to be well below levels of concern for the air pathway, despite the fact that Th-230 has a high level of radioactivity.

- Our risk assessment focused on the inhalation of re-suspended sediments at the UCR site, measured in terms of PM_{10}. If the particle sizes in the studies underlying the inhalation toxicity values we used are different from those measured at the site, this could introduce some uncertainty into our risk estimates, the direction of which differs by specific chemical.
- We do not know the precise form of the metals captured by the monitors, nor the bioavailability of the metal compounds or complexes present. Where possible, we have used toxicity values from studies of metal compounds generally (e.g., barium and compounds). In some cases, the toxicity values we apply are for specific compounds (e.g., vanadium pentoxide). If the compounds actually present differ from those on which the toxicity values are based, risks may be under- or overestimated.

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APPENDIX A:

HAZARD IDENTIFICATION SUMMARY TABLE

	Cancer	Non cancer												
Metal	**Tumor Type**	**Respiratory**	**Cardiovascular**	**Gastrointestinal**	**Hematological**	**Immune**	**Musculoskeletal**	**Hepatic**	**Renal**	**Dermal**	**Ocular**	**Neurological**	**Reproductive**	**Developmental**
Antimony		A, S, C	A, S, C	C					A, C		A, C [1]			
Arsenic	Lung	A	C							A		A, **C**	**A**	A, **C**
Barium		C	C	C			C							
Beryllium	Lung	**C**	C					C	C					
Bismuth	Elevated total cancer risk			C			C			C				
Cadmium	Lung, trachea, bronchus	A, **C**							**C**					
Cerium		C												
Cesium			C [2]	C [2]										
Chromium	Lung, nasal	S, **C**												
Cobalt	Lung	**C, S**	C							C				
Copper		**A**, C												
Gallium					U					U				
Iron		C												
Lanthanum														
Lead			C		C							C	C	C
Lithium		U		U					U			U		
Manganese		C										S, **C**	C	
Mercury												**A, S, C**	**A**	
Molybdenum				U	U						U			U
Nickel	Lung, nasal	**A, C**				C								
Niobium														
Phosphorus				U	U									
Rubidium														

	Cancer	Non cancer												
Metal	Tumor Type	Respiratory	Cardiovascular	Gastrointestinal	Hematological	Immune	Musculoskeletal	Hepatic	Renal	Dermal	Ocular	Neurological	Reproductive	Developmental
Scandium														
Silver		C		C					C					
Strontium						C [3]								
Thallium							C					C		
Thorium	Elevated total cancer risk				C									
Titanium		U												
Uranium	Elevated total cancer risk								S,C					
Vanadium [4]	Alveolar, bronchiolar	**A, S, C**									A			
Yttrium		U						U			U			
Zinc		A												
PM10		A, C	A											
PM2.5		A, C	A, C											

A = Acute (1 hour to 14 days of exposure), S= Subchronic (14 to 364 days of exposure, or repeated 8-hour exposures), C= Chronic (Over one year of exposure), U= Uncertain (See notes to Hazard ID table for specifics of these exposure types. Subchronic exposures include both 8-hour RELs and Intermediate MRLs)

Bold entries are for the critical effect. Generally, this also means a toxicity value was obtained for the listed endpoint.

Notes:

[1] Ocular irritation reported in humans following exposure to airborne antimony. This could be due to exposure through the dermal route, rather than the inhalation route (ATSDR Toxicological Profile for Antimony: http://www.atsdr.cdc.gov/toxprofiles/tp23.html).

[2] Based on select human case studies.

[3] For Radiostrontium, ^{90}Sr.

[4] For Vanadium Pentoxide.

APPENDIX B:

SCREENING ANALYSIS MEMO

MEMORANDUM | July 27, 2009

TO Tino Tafoya, Bureau of Reclamation

FROM Tyra Walsh, Mikael Gentile, and Henry Roman, Industrial Economics, Inc. (IEc)

SUBJECT Screening Analysis for Lake Roosevelt Inhalation Risk Assessment

INTRODUCTION

This memo provides a brief overview of the methodology that we used to conduct a screening analysis as part of an inhalation risk assessment of contamination at Lake Roosevelt. This memo and the accompanying spreadsheet represent the deliverable for Task 3 of the Statement of Work (SOW) for Reclamation Delivery Order 09PE107660. We first describe the process we used to identify human health inhalation screening levels and how we compared these to the monitoring data from Lake Roosevelt. We then describe the results of the screening analysis and our list of contaminants of potential concern (COPC) that we will carry through to the risk assessment.

METHODS

We performed a screening analysis in order to narrow the focus of the risk assessment to only include those metals that would pose a potential risk to receptors at Lake Roosevelt through the inhalation exposure route. This consisted of comparing the maximum contaminant concentrations measured by the three United States Geological Survey (USGS) monitors on-site between 2002 and 2006 with conservative human health risk-based (HHRB) screening values when available, or, in the case of particulate matter (PM) concentrations, with the current PM National Ambient Air Quality Standards (NAAQS). If the maximum concentration for a contaminant at the site exceeded the screening value, then we considered it a COPC and will continue to evaluate potential risks from inhalation exposure to this contaminant going forward. The results of the screening analysis are presented in the attached spreadsheet (UCR Inhalation Screening Analysis_062409.xls).

According to an initial assessment of the data on wind speed collected by USGS, we determined that receptors at Lake Roosevelt (e.g., workers, local residents and recreators) would likely be exposed to inhaled particles in two main ways. First, high wind events (HWEs) could cause short periods of exposure (e.g., less than 8 hours) to higher concentrations of particles.[39] Second, receptors could be exposed to varying

[39] We define a HWE as four or more consecutive hours with mean wind speed at or above 5.14 m/s. This definition is consistent with that used by USGS when designing the sampling protocol for 2006, which was targeted to collect data only during HWEs.

concentrations of these contaminants over longer periods of time. Therefore, we first performed a screen using acute toxicity benchmark concentrations, to ensure protection against extreme exposures during HWEs. We also compared these concentrations against chronic HHRB screening values, to protect against longer-term exposure to particles at the site. If we could not identify appropriate acute toxicity values or chronic HHRB screening values for a contaminant, we attempted to locate data on background concentrations of that contaminant.

CONTAMINANT CONCENTRATIONS

We first calculated summary statistics (e.g., mean, median, minimum, maximum) for each contaminant by observation site (Kettle Falls, Marcus, Inchelim or Seven Bays) and year (see the "Summary Statistics" tab of the attached spreadsheet) using the air monitoring data provided to us by USGS for 2002-2006. We then calculated the maximum observed concentration across all years and sites for each contaminant. We noted the date and site of the observation, and also if the observation was taken during a HWE. We then compared this overall maximum concentration to the screening values described below.

ACUTE TOXICITY SCREENING ANALYSIS

To determine whether any of the monitored contaminants could potentially pose an unacceptable acute risk, we compared the maximum concentration to the acute inhalation toxicity values we identified in our Hazard Identification (Task 2 of the SOW). If multiple acute toxicity values from different sources were identified for a contaminant, we compared to the lowest value.

CHRONIC TOXICITY SCREENING ANALYSIS

To determine which contaminants could pose risks through chronic exposure, we compared each maximum contaminant concentration to its corresponding chronic HHRB screening level for residential ambient air from EPA's Regional Screening Levels for Chemical Contaminants at Superfund Sites (hereafter, "Regional Screening Table") (http://www.epa.gov/reg3hwmd/risk/human/rb-concentration_table/index.htm).[40] These screening values are based on chronic toxicity values that were selected using the same hierarchy as we employed in our Hazard Identification.[41] In addition, they are based on conservative assumptions, such as a hazard quotient of 0.1 for non-cancer and a one-in-one million cancer risk as well as conservative exposure assumptions (e.g., receptors are exposed 350 days per year, 24 hours per day for 30 years).

[40] Note that comparing a one-time maximum concentration to a chronic screening value is a conservative approach, in that longer term average exposures are likely to be significantly lower than this maximum value.

[41] U.S. Environmental Protection Agency (2003). *Human Health Toxicity Values in Superfund Risk Assessment*. Office of Solid Waste and Emergency Response, Washington, D.C. Publication 9285.7-53.

For contaminants lacking inhalation-based HHRB screening levels, we first considered alternative means of screening using other toxicity information. Because some fraction of the PM_{10} particles inhaled by an exposed individual would be trapped by the mucous lining the respiratory tract and ultimately ingested, we felt it was reasonable to perform a screening analysis using HHRB screening levels based on ingestion exposures and oral toxicity data if inhalation-based values were lacking.[42] We assumed that 50 percent of inhaled particles would be ingested. We then calculated chronic ingestion screening values using the equations provided on the Regional Screening Table website along with chronic ingestion toxicity data and compared these to the maximum ingested concentration (see the "Ingestion SLs" tab of the attached spreadsheet).

RADIONUCLIDE EXPOSURE

Thorium and uranium were the two radionuclides detected among the USGS PM samples. We compared the maximum detected uranium concentration to a chronic screening level in air from the Regional Screening Tables; it did not exceed the screening level and will be excluded from further analysis.

The Regional Screening Tables do not include a screening level for thorium, however. To assess the carcinogenic potential of radiation exposure from inhaled thorium, we calculated a screening level for the most common thorium isotope, Th-232, based on a cancer slope factor for that radionuclide from EPA's Health Effects Assessment Summary Tables (HEAST) - Radionuclides Table.[43] The inhalation cancer slope factor in HEAST is expressed in terms of lifetime excess cancer risk per picocurie (pCi), where pCi is a measure of the radioactivity to which an individual may be exposed.[44] We divided this value (4.33E-08 per pCi) into 1E-06 to calculate a HHRB screening level in total pCi corresponding to a one-in-one million incremental lifetime cancer risk (approximately 23 pCi). We then developed a total radiation exposure estimate for Th-232 using conservative exposure assumptions. First, we assumed that all thorium detected in the USGS data is Th-232, and that adults in the area would be exposed to the maximum Th-232 concentration 350 days per year, with a breathing rate of 20 m^3 per day. This results in the inhalation of 26 µg of Th-232 in one year, and 780 µg over a 30-year exposure period.[45] We then converted the total mass of Th-232 to a measure of total radiation activity in pCi using the Th-232 Specific Activity factor of 1.1E-7 pCi/g, and found the total radiation exposure of an adult over 30-years to the maximum Th-232 concentration to be 86 pCi, which exceeds the one-in-one million cancer risk-based screening level. Thus, we will include thorium as a COPC in the risk assessment.

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[42] We assumed that the risks due to incidental ingestion of inhaled particles would be encompassed by the inhalation based screening values when available.

[43] See http://www.epa.gov/radiation/heast/. Searched June 2009.

[44] A Curie (Ci) is a measure of the number of disintegrations per second of a radionuclide (One Ci = 3.7 x 10^{10} disintegrations per second). One picocurie = 1E-12 Ci.

[45] We used the following equation: 0.00371 µg/m^3 x 20 m^3/day x 350 days/yr = 26 µg/yr.

COMPARISON TO BACKGROUND LEVELS

We attempted to perform a comparison to background levels for contaminants detected by the USGS monitors at least once for which we identified no toxicity information from EPA's Superfund hierarchy of data sources. We obtained estimates of background concentrations of the USGS analytes in sediment in the Lake Roosevelt area from a 2001 USGS sediment sampling report.[46,47] If background data were not available from the USGS report for a contaminant, we attempted to identify background concentrations in sediment from other sources.[48]

The comparison to background levels proved problematic because of different particle size distributions between the measured contaminant concentrations on the monitor filters and the various background measurements in sediment or soil. For instance, the 2002 – 2006 data represent concentrations of these contaminants in the PM_{10} fraction of soil/sediment (particles with a diameter of 10 μm or less), whereas the USGS background levels contain particles that are 63 μm in diameter and smaller. As a result, we did not eliminate any of these contaminants based on a background comparison at this time, though we will continue to investigate potential adjustments to the background data that may allow for a more informative comparison.

PARTICULATE MATTER SCREENING ANALYSIS

To determine whether receptors may be at risk from exposure to coarse (PM_{10}) and fine ($PM_{2.5}$) particles at the site, we performed a screen using the National Ambient Air Quality Standards (NAAQS) for these particle size fractions. We compared measured PM_{10} concentrations to the current acute 24-hour standard, and compared our estimated $PM_{2.5}$ concentrations to both the 24-hour and annual average NAAQS.[49] In order to convert the measured PM_{10} concentrations to an equivalent $PM_{2.5}$ concentration, we calculated a ratio of $PM_{2.5}/PM_{10}$ using EPA monitor values from a nearby site in Colville, WA.[50]

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[46] Majewski, M. et al. (2003). *Concentrations and Distribution of Slag-Related Trace Elements and Mercury in Fine-Grained Beach and Bed Sediments of Lake Roosevelt Washington, April-May 2001*. U.S. Department of the Interior, U.S. Geological Survey. Tacoma, Washington. Our background estimate consisted of an average of five reference sites.

[47] We converted the maximum observed concentrations to parts per million (ppm) or percentage of sample (depending on how the element was reported in the USGS report) using data on sample volume and net sample weight in order to compare them to the background concentrations.

[48] All levels were found in the following book: Kabata-Pendias, A. (2001) *Trace Elements in Soils and Plants*, Third Edition. New York; CRC Press LLC.

[49] Note that there is not currently an annual average PM_{10} standard (it was revoked in 2006).

[50] We obtained monitoring data from EPA's AirData website (http://www.epa.gov/oar/data/). Monitoring values were available for these two pollutants at the Colville site for the years 2001 and 2002. We calculated the ratio of $PM_{2.5}/PM_{10}$ at the monitor for both of these years individually. We then calculated a mean ratio, which we applied in our analysis (25 percent).

RESULTS AND CONCLUSIONS

Below is a summary of the results and implications of our screening assessment (also see the "Summary" tab of the attached spreadsheet). We also present the results in Exhibits 1 and 2 below.

- None of the maximum measured concentrations exceeded available acute toxicity values. Therefore, our risk assessment will focus on chronic exposures.[51]
- Eight contaminants (chromium, manganese, cobalt, nickel, arsenic, cadmium, barium and thorium) exceeded chronic HHRB screening values, and therefore will be carried through the risk assessment as COPCs.
- We are currently unable to quantitatively estimate acute or chronic inhalation risks from exposure to 19 contaminants listed in Exhibit 2 because they lack inhalation toxicity data. Copper lacks a chronic inhalation toxicity data; however, the maximum concentration of this contaminant passed the acute toxicity screen.
- Of those contaminants in Exhibit 2 with available oral toxicity data (lithium, iron, zinc, strontium, molybdenum, silver, antimony, and thallium), none exceeded the oral screening values for risk from chronic exposure via incidental ingestion of inhaled particles. This result was not sensitive to our assumption about the fraction of inhaled particles ingested.
- We are currently unable to exclude any of the contaminants in Exhibit 2 based on a comparison with background levels.
- None of the PM_{10} or estimated $PM_{2.5}$ concentrations exceeding the corresponding NAAQS level for short-term or long-term exposures. Our findings for the annual average $PM_{2.5}$ concentrations are sensitive to our assumed fraction of $PM_{2.5}$ in each sample, however.
- We will assess risks from lead in our assessment using EPA's Integrated Exposure Uptake Biokinetic (IEUBK) model (http://www.epa.gov/superfund/lead/products.htm). The maximum lead concentration in the USGS data did not exceed the current 3-month rolling average lead NAAQS. However, given that lead is a COPC in other media at the UCR site and that children are a sensitive subgroup for the health effects of lead, we propose to evaluate the overall lead exposures to children including inhalation of lead particles.

[51] Note that subchronic toxicity values exist for some contaminants. However, since chronic toxicity values are more conservative, we based our screen on chronic HHB screening values. If we determine that HWEs are best modeled as intermediate exposures, we will assess risks using intermediate toxicity values and exposure assumptions.

EXHIBIT 1. CONTAMINANTS THAT EXCEED SCREENING LEVELS

Contaminants that exceed screening levels
Arsenic
Barium
Cadmium
Chromium
Cobalt
Lead[1]
Manganese
Nickel
Thorium[2]
Vanadium

[1] Due to a lack of screening values for lead, we could not determine that concentrations exceed human health-based screening levels. However, due to the sensitivity of children to lead exposure, we have opted to include it in the risk assessment.
[2] We assessed Thorium on the basis of excess cancer risk due to radiation.

EXHIBIT 2. CONTAMINANTS WITHOUT INHALATION TOXICITY VALUES

Contaminants without inhalation toxicity values
Antimony*
Bismuth
Cerium
Cesium
Copper (lacks chronic only)**
Galium
Iron*
Lanthanum
Lithium*
Molybdenum*
Niobium
Phosphorus
Rubidium
Scandium
Silver*
Strontium*
Thallium*
Titanium
Zinc*

*We also assessed incidental ingestion for these chemicals. Based on our analysis, none of the contaminants posed unacceptable risks at detected levels via this route of exposure.
**Passed acute toxicity screen.

APPENDIX C:

CONTAMINANT CONCENTRATIONS IN AIR

TABLE C-1. EXPOSURE POINT CONCENTRATIONS FOR ALL DATA (CHRONIC EXPOSURE SCENARIO)

	INCHELIUM	MARCUS	SEVEN BAYS
Arsenic	0.0005	0.00075	0.0003
	95% KM (BCA) UCL	95% KM (BCA) UCL	95% KM (BCA) UCL
Barium	0.15	0.016	0.18
	95% KM (Chebyshev) UCL	95% KM (Percentile Bootstrap) UCL	95% KM (Chebyshev) UCL [4]
Cadmium	0.00014	0.00034	0.000067
	95% KM (Chebyshev) UCL	95% H-UCL [1]	95% KM (BCA) UCL
Chromium	0.0019	0.0024	0.0013
	95% KM (BCA) UCL	95% KM (Chebyshev) UCL	95% KM (BCA) UCL
Cobalt	0.00025	0.00019	0.00026
	95% KM (Chebyshev) UCL	95% KM (Chebyshev) UCL	95% KM (Chebyshev) UCL
Lead	0.0022	0.0052	0.0015
	Mean w/ NDs as DL/2	Mean w/ NDs as DL/2	Mean w/ NDs as DL/2
Manganese	0.012	0.0088	0.014
	95% KM (Chebyshev) UCL	95% KM (Chebyshev) UCL	95% KM (Chebyshev) UCL
Nickel	0.00054	0.00044	0.00043
	95% KM (BCA) UCL	95% KM (BCA) UCL	95% KM (BCA) UCL
Thorium	0.00012	0.000061	0.00012
	95% KM (Percentile Bootstrap) UCL	95% KM (Percentile Bootstrap) UCL	95% KM (BCA) UCL
Vanadium	0.0018	0.0011	0.0016
	95% KM (Chebyshev) UCL	95% KM (Chebyshev) UCL	95% KM (Chebyshev) UCL

All values, unless noted, are 95 percent Upper Confidence Limits (UCL) on the mean calculated in ProUCL 4.0, in $\mu g/m^3$. Values are rounded to two significant figures.

Unless otherwise noted, calculation of the 95 percent UCL is based on a nonparametric distribution and calculated using the method listed below the estimate in light gray.

[1] Assuming a lognormal distribution.

[2] Assuming a normal distribution.

[3] Assuming a gamma distribution.

[4] Recommended data was a 97.5 percent UCL, we use the 95 percent UCL using the same method.

[5] ProUCL recommended two UCLs; reported value is the first one.

TABLE C-2. EXPOSURE POINT CONCENTRATIONS FOR SUMMER DATA (SUB-CHRONIC EXPOSURE SCENARIO)

	INCHELIUM	MARCUS	SEVEN BAYS
Arsenic	0.0008	0.00074	0.00037
	95% KM (Chebyshev) UCL	95% KM (Chebyshev) UCL	95% KM (Chebyshev) UCL
Barium	0.025	0.025	0.019
	95% KM (BCA) UCL	95% KM (Percentile Bootstrap) UCL	95% KM (Percentile Bootstrap) UCL
Cadmium	0.00012	0.00036	0.000068
	95% KM (Chebyshev) UCL	95% Chebyshev(Mean, Sd) UCL [4]	95% KM (Chebyshev) UCL
Chromium	0.0027	0.0015	0.0016
	95% KM (Chebyshev) UCL	95% KM (BCA) UCL	95% KM (BCA) UCL
Cobalt	0.00028	0.00015	0.00027
	95% KM (Chebyshev) UCL	95% KM (Chebyshev) UCL	95% H-UCL
Lead	0.0019	0.0033	0.0012
	Mean w/ NDs as DL/2	Mean w/ NDs as DL/2	Mean w/ NDs as DL/2
Manganese	0.015	0.0086	0.014
	95% KM (Chebyshev) UCL	95% H-UCL [1]	95% H-UCL [1]
Nickel	0.00058	0.00031	0.00046
	95% KM (BCA) UCL	95% KM (Percentile Bootstrap) UCL	95% KM (Percentile Bootstrap) UCL
Thorium	0.00013	0.000063	0.00021
	95% KM (BCA) UCL	95% KM (Percentile Bootstrap) UCL	95% KM (Chebyshev) UCL
Vanadium	0.0022	0.00088	0.0016
	95% KM (Chebyshev) UCL	95% Student's-t UCL [2]	95% H-UCL [1]

All values, unless noted, are 95 percent Upper Confidence Limits (UCL) on the mean calculated in ProUCL 4.0, in $\mu g/m^3$. Values are rounded to two significant figures.

Unless otherwise noted, calculation of the 95 percent UCL is based on a nonparametric distribution and calculated using the method listed below the estimate in light gray.

[1] Assuming a lognormal distribution.

[2] Assuming a normal distribution.

[3] Assuming a gamma distribution.

[4] Recommended data was a 97.5 percent UCL, we use the 95 percent UCL using the same method.

[5] ProUCL recommended two UCLs; reported value is the first one.

TABLE C-3. EXPOSURE POINT CONCENTRATIONS FOR DRAWDOWN DATA (SUB-CHRONIC EXPOSURE SCENARIO)

	INCHELIUM	MARCUS	SEVEN BAYS
Arsenic	0.00046	0.00077	0.00028
	95% KM (BCA) UCL	95% KM (BCA) UCL	95% KM (BCA) UCL
Barium	0.022	0.015	0.095
	95% KM (Percentile Bootstrap) UCL	95% KM (Percentile Bootstrap) UCL	95% KM (Chebyshev) UCL [4]
Cadmium	0.00015	0.00035	0.00007
	95% KM (Chebyshev) UCL	95% H-UCL	95% KM (Chebyshev) UCL
Chromium	0.0021	0.0029	0.0014
	95% KM (BCA) UCL	95% KM (Chebyshev) UCL	95% KM (BCA) UCL
Cobalt	0.00025	0.00021	0.00027
	95% KM (Chebyshev) UCL	95% KM (Chebyshev) UCL	95% Chebyshev(Mean, Sd) UCL [4]
Lead	0.0024	0.0058	0.0014
	Mean w/ NDs as DL/2	Mean w/ NDs as DL/2	Mean w/ NDs as DL/2
Manganese	0.0098	0.007	0.015
	95% Approximate Gamma UCL [3]	95% Approximate Gamma UCL [3]	95% Chebyshev(Mean, Sd) UCL [4]
Nickel	0.00075	0.00043	0.00044
	95% KM (Chebyshev) UCL	95% KM (t) UCL [5]	95% KM (BCA) UCL
Thorium	0.00012	0.000057	0.00011
	95% KM (BCA) UCL	95% KM (t) UCL	95% KM (BCA) UCL
Vanadium	0.0016	0.00096	0.0013
	95% Approximate Gamma UCL [3]	95% Approximate Gamma UCL [3]	95% KM (BCA) UCL

All values, unless noted, are 95 percent Upper Confidence Limits (UCL) on the mean calculated in ProUCL 4.0, in $\mu g/m^3$. Values are rounded to two significant figures.

Unless otherwise noted, calculation of the 95 percent UCL is based on a nonparametric distribution and calculated using the method listed below the estimate in light gray.

[1] Assuming a lognormal distribution.

[2] Assuming a normal distribution.

[3] Assuming a gamma distribution.

[4] Recommended data was a 97.5 percent UCL, we use the 95 percent UCL using the same method.

[5] ProUCL recommended two UCLs; reported value is the first one.

APPENDIX D:

RISK ESTIMATES FOR INDIVIDUAL CONTAMINANTS

TABLE D-1. RISK ESTIMATES FOR LOCAL RESIDENTS

	INCHELIUM		MARCUS		SEVEN BAYS	
	CANCER RISK	HQ	CANCER RISK	HQ	CANCER RISK	HQ
Arsenic	8.84E-07	0.03	1.33E-06	0.05	5.30E-07	0.02
Barium	N/A	0.29	N/A	0.03	N/A	0.35
Cadmium	1.04E-07	0.01	2.52E-07	0.03	4.96E-08	0.01
Chromium [1]	9.37E-06	0.003	1.18E-05	0.003	6.41E-06	0.002
Cobalt	9.25E-07	0.04	7.03E-07	0.03	9.62E-07	0.04
Manganese	N/A	0.23	N/A	0.17	N/A	0.27
Nickel	5.33E-08	0.01	4.34E-08	0.01	4.24E-08	0.01
Thorium	7.20E-08	N/A	3.66E-08	N/A	7.20E-08	N/A
Vanadium	6.14E-06	0.25	3.75E-06	0.15	5.46E-06	0.22
Total	1.75E-05	0.86	1.79E-05	0.47	1.35E-05	0.91

[1] Cancer risk for Chromium is assessed for total chromium. Chromium is assessed for Cr(VI) only for the non-cancer HQ as the only chronic toxicity data available for chromium is for Cr(VI).

TABLE D-2. RISK ESTIMATES FOR FULL-TIME ARCHAEOLOGISTS

	INCHELIUM		MARCUS		SEVEN BAYS	
	CANCER RISK	HQ	CANCER RISK	HQ	CANCER RISK	HQ
Arsenic	5.05E-08	0.003	7.57E-08	0.004	3.03E-08	0.002
Barium	N/A	0.02	N/A	0.003	N/A	0.03
Cadmium	5.92E-09	0.001	1.44E-08	0.003	2.83E-09	0.001
Chromium [1]	5.35E-07	0.0002	6.76E-07	0.0003	3.66E-07	0.0002
Cobalt	5.28E-08	0.003	4.02E-08	0.003	5.50E-08	0.004
Manganese	N/A	0.02	N/A	0.01	N/A	0.02
Nickel	3.04E-09	0.001	2.48E-09	0.001	2.42E-09	0.001
Thorium	1.07E-08	N/A	5.44E-09	N/A	1.07E-08	N/A
Vanadium	3.51E-07	0.02	2.14E-07	0.01	3.12E-07	0.02
Total	1.01E-06	0.07	1.03E-06	0.04	7.79E-07	0.08

[1] Cancer risk for Chromium is assessed for total chromium. Chromium is assessed for Cr(VI) only for the non-cancer HQ as the only chronic toxicity data available for chromium is for Cr(VI).

TABLE D-3. RISK ESTIMATES FOR SEASONAL ARCHAEOLOGISTS

	INCHELIUM		MARCUS		SEVEN BAYS	
	CANCER RISK	HQ	CANCER RISK	HQ	CANCER RISK	HQ
Arsenic	1.89E-09	0.002	3.16E-09	0.003	1.15E-09	0.001
Barium	N/A	0.003	N/A	0.002	N/A	0.01
Cadmium	2.58E-10	0.001	6.01E-10	0.002	1.20E-10	0.0005
Chromium [1]	2.40E-08	0.0001	3.32E-08	0.0001	1.60E-08	0.0001
Cobalt	2.15E-09	0.001	1.80E-09	0.001	2.32E-09	0.001
Manganese	N/A	0.004	N/A	0.003	N/A	0.006
Nickel	1.72E-10	0.0003	9.85E-11	0.0001	1.01E-10	0.0001
Thorium	4.35E-10	N/A	2.06E-10	N/A	3.98E-10	N/A
Vanadium	1.27E-08	0.001	7.60E-09	0.001	1.03E-08	0.001
Total	4.16E-08	0.01	4.67E-08	0.01	3.04E-08	0.02

[1] Cancer risk for Chromium is assessed for total chromium. Chromium is assessed as combined risk from Cr(III) and Cr(VI) for the non-cancer HQ as sub-chronic toxicity data were available Cr(III) and Cr(VI) separately.

TABLE D-4. RISK ESTIMATES FOR FULL-TIME MAINTENANCE WORKERS

	INCHELIUM		MARCUS		SEVEN BAYS	
	CANCER RISK	HQ	CANCER RISK	HQ	CANCER RISK	HQ
Arsenic	1.58E-07	0.01	2.37E-07	0.01	9.47E-08	0.004
Barium	N/A	0.06	N/A	0.01	N/A	0.07
Cadmium	1.85E-08	0.003	4.49E-08	0.01	8.85E-09	0.001
Chromium [1]	1.67E-06	0.001	2.11E-06	0.001	1.14E-06	0.000
Cobalt	1.65E-07	0.01	1.25E-07	0.01	1.72E-07	0.01
Manganese	N/A	0.05	N/A	0.04	N/A	0.06
Nickel	9.51E-09	0.002	7.75E-09	0.002	7.57E-09	0.002
Thorium	3.34E-08	N/A	1.70E-08	N/A	3.34E-08	N/A
Vanadium	1.10E-06	0.05	6.70E-07	0.03	9.75E-07	0.05
Total	3.15E-06	0.18	3.22E-06	0.10	2.44E-06	0.20

[1] Cancer risk for Chromium is assessed for total chromium. Chromium is assessed for Cr(VI) only for the non-cancer HQ as the only chronic toxicity data available for chromium is for Cr(VI).

TABLE D-5. RISK ESTIMATES FOR PARK VISITORS

	INCHELIUM		MARCUS		SEVEN BAYS	
	CANCER RISK	HQ	CANCER RISK	HQ	CANCER RISK	HQ
Arsenic	1.88E-08	0.02	1.74E-08	0.02	8.72E-09	0.01
Barium	N/A	0.02	N/A	0.02	N/A	0.01
Cadmium	1.18E-09	0.004	3.55E-09	0.01	6.71E-10	0.002
Chromium [1]	1.78E-07	0.001	9.86E-08	0.0003	1.05E-07	0.0003
Cobalt	1.38E-08	0.005	7.40E-09	0.003	1.33E-08	0.005
Manganese	N/A	0.03	N/A	0.02	N/A	0.03
Nickel	7.63E-10	0.001	4.08E-10	0.001	6.05E-10	0.001
Thorium	1.04E-09	N/A	5.04E-10	N/A	1.68E-09	N/A
Vanadium	1.00E-07	0.01	4.00E-08	0.003	7.28E-08	0.01
Total	3.13E-07	0.08	1.68E-07	0.07	2.03E-07	0.06

[1] Cancer risk for Chromium is assessed for total chromium. Chromium is assessed as combined risk from Cr(III) and Cr(VI) for the non-cancer HQ as sub-chronic toxicity data were available Cr(III) and Cr(VI) separately.