FINAL SITE-WIDE
HUMAN HEALTH RISK ASSESSMENT:
UPPER COLUMBIA RIVER SITE

February 10, 2021

Prepared for and with oversight by:
U.S. Environmental Protection Agency Region 10 and
U.S. Environmental Protection Agency Environmental Response Team

Prepared by:
SRC

Contract Number: 68HERH19D0022
EPA Task Order: 68HERH19F0280
Project Number: EH040.0.015.011

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ACRONYMS AND GLOSSARY[1]

95UCL	95% Upper Confidence Limit of the Arithmetic Mean
AALM	All Ages Lead Model
ABA	Absolute Bioavailability
ABS_d	Dermal Absorption Fraction
ABS_{GI}	Gastrointestinal Absorption Fraction
ADA	Aerial Deposition Area
ADI	Average Daily Intake
AI	Adequate Intake
ALM	Adult Lead Methodology
AT	Averaging Time
ATSDR	Agency for Toxic Substances and Disease Registry
B.C.	British Columbia
BERA	Baseline Ecological Risk Assessment
bgs	Below Ground Surface
BLL	Blood Lead Level
BMDL	Benchmark Dose Modeling Lower Confidence Limit
BW	Body Weight
C	Concentration
$CaCO_3$	Calcium Carbonate
CASRN	Chemical Abstracts Service Registry Number
CCT	Confederated Tribes of the Colville Reservation
CCT Tribal Survey	Tribal Consumption and Resource Use Survey
CDC	Centers for Disease Control and Prevention
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act
CF	Confidence Factor
CHIRU	Colville High Intensity Resource User
CI	Confidence Interval
cm	centimeter(s)
cm^2	square centimeter(s)
CnF	Conversion Factor
COI	Chemical of Interest
COPC	Chemical of Potential Concern
Cr(VI)	Hexavalent Chromium
CSM	Conceptual Site Model
CTE	Central Tendency Exposure
DAD	Dermally-Absorbed Dose
DAD_L	Dermally-Absorbed Dose, Averaged Over a Lifetime
DA_{event}	Absorbed Dose per Event
DAF	Dermal Adherence Factor
DAR	Data Analysis Report
DCR	Daily Consumption Rate

[1] Most technical terminology and abbreviations used in this document can be found in EPA reference documents and are defined on https://www.epa.gov/superfund/superfund-glossary.

DDD	Dichlorodiphenyldichloroethane
DDT	Dichlorodiphenyltrichloroethane
DI	Daily Intake
DI_L	Daily Intake, Averaged Over a Lifetime
dL	deciliter(s)
DL-PCB	Dioxin-like PCB
DMP	Data Management Plan
DOI	Department of the Interior
DOJ	Department of Justice
DQO	Data Quality Objective
DSR	Data Summary Report
DU	Decision Unit
dw	dry weight
EC	Exposure Concentration
ECY	Washington State Department of Ecology
ED	Exposure Duration
EF	Exposure Frequency
EMAP	Environmental Monitoring and Assessment Program
EMPC	Estimated Maximum Possible Concentration
EPA	Environmental Protection Agency
EPC	Exposure Point Concentration
ERA	Ecological Risk Assessment
ESI	Expanded Site Inspection
ET	Exposure Time
EV	Event Frequency
FDA	U.S. Food and Drug Administration
FFCMP	Freshwater Fish Contaminant Monitoring Program
FQ	Food Questionnaire
FSCA	Fish Sample Collection Area
g	gram(s)
GI	Gastrointestinal
GPS	Geographic Positioning System
GSD	Geometric Standard Deviation
GSF	Grain Size Fraction
HEAST	Health Effects Assessment Summary Tables
HHRA	Human Health Risk Assessment
HI	Hazard Index
HIF	Human Intake Factor
HQ	Hazard Quotient
HSCA	Hazardous Substances Control Act
HUC	Hydrologic Unit Code
HUD	U.S. Department of Housing and Urban Development
IC	Incremental Composite
ICS	Incremental Composite Sampling
IEc	Industrial Economics, Inc.
IEUBK	Integrated Exposure Uptake Biokinetic

IOM	Institute of Medicine
IQ	Intelligence Quotient
IR	Intake Rate
IRIS	Integrated Risk Information System
ISA	Integrated Science Assessment
ITRC	Interstate Technology & Regulatory Council
IUR	Inhalation Unit Risk
IVBA	*in vitro* Bioavailability
kg	kilogram(s)
KIVCET	Oxygen, Vortex, Cyclone and Electrothermic (in Russian)
KM	Kaplan Meier
K_p	Dermal Permeability Coefficient
L	Liter(s)
Lake Roosevelt	Franklin D. Roosevelt Lake
LCL	Lower Confidence Limit
LOAEL	Lowest-Observed-Adverse-Effect Level
LOE	Level of Effort
LRF	Lake Roosevelt Forum
LRNRA	Lake Roosevelt National Recreation Area
m	meter(s)
m^3	cubic meter(s)
µg	microgram(s)
µm	micrometer(s)
ME	Margin of Error
mg	milligram(s)
mi^2	square mile(s)
mm	millimeter(s)
MRL	Minimal Risk Level
MSD	Mass Fraction of Soil in Indoor Dust
MWL	Mountain Whisper Light
n	Sample Size
NAAQS	National Ambient Air Quality Standards
NAP	National Academies Press
NAS	National Academy of Sciences
NCEA	National Center for Environmental Assessment
NCI	National Cancer Institute
NCP	National Contingency Plan
ND	Non-Detect
NGVD	National Geodetic Vertical Datum
NHANES	National Health and Nutrition Examination Survey
NHEXAS	National Human Exposure Assessment Survey
NIH	National Institutes of Health
NOAEL	No-Observed-Adverse-Effect Level
NPS	National Park Service
NTP	National Toxicology Program
NURE	National Uranium Resource Evaluation

OEHHA	California Office of Environmental Health and Hazard Assessment
OFM	Washington State Office of Financial Management
OLEM	Office of Land and Emergency Management
ORD	Office of Research and Development
OSRTI	Office of Superfund Remediation and Technology Innovation
OSWER	Office of Solid Waste and Emergency Response
P2	5% Probability of Exceeding a PbB of 2 µg/dL
P3	5% Probability of Exceeding a PbB of 3 µg/dL
P5	5% Probability of Exceeding a PbB of 5 µg/dL
P8	5% Probability of Exceeding a PbB of 8 µg/dL
P95	95^{th} Percentile
PAH	Polycyclic Aromatic Hydrocarbon
Pb	Lead
PbB	Blood Lead Concentration
PBDE	Polybrominated Diphenyl Ether
PBT	Persistent, Bioaccumulative, and Toxic
PCB	Polychlorinated Biphenyl
PCDD	Polychlorinated Dibenzo-p-dioxin
PCDF	Polychlorinated Dibenzofuran
PM_{10}	Particulate Matter
ppm	Parts per Million
PPRTV	Provisional Peer-Reviewed Toxicity Value
p-RfD	Provisional Reference Dose
PRG	Preliminary Remediation Goal
QAPP	Quality Assurance Project Plan
QA/QC	Quality Assurance/Quality Control
RAGS	Risk Assessment Guidance for Superfund
RBA	Relative Bioavailability
RBSL	Risk-based Screening Level
RDA	Recommended Dietary Allowance
RE	Relative Error
RecUse Survey	Recreational Consumption and Resource Use Survey
REL	Reference Exposure Level
RfC	Reference Concentration
RfD	Reference Dose
RfDabs	Absorbed Reference Dose
RFDA	Relict Floodplain Deposition Area
RI/FS	Remedial Investigation and Feasibility Study
RM	River Mile
RME	Reasonable Maximum Exposure
ROS	Regression on Order Statistics
RPF	Relative Potency Factor
RSE	Removal Site Evaluation
RSL	Regional Screening Level
SA	Surface Area
SAB	Science Advisory Board

SD	Standard Deviation
SDU	Sediment Decision Unit
SE	Standard Error
SF	Slope Factor
SF_{ABS}	Absorbed Slope Factor
SiO_2	Silicon Dioxide
Site	UCR Site
SLERA	Screening-level Ecological Risk Assessment
SO_2	Sulfur Dioxide
SRL	Savannah River Laboratory
STI	Spokane Tribe of Indians
SVOC	Semi-volatile Organic Compound
t_{event}	Event Duration
TAI	Teck American Incorporated
TAL	Target Analyte List
TCDD	2,3,7,8-Tetrachlorodibenzo-para-dioxin (Dioxin)
TCRA	Time Critical Removal Action
TDS	Total Diet Study
Teck	Teck Metals Ltd.
TEF	Toxic Equivalency Factor
TEQ	Toxic Equivalent
TOC	Total Organic Carbon
TRI	Toxics Release Inventory
TRW	Technical Review Workgroup
TSS	Total Suspended Solids
TUIL	Tolerable Upper Intake Level
TWA	Time-Weighted Average
TWF	Time-Weighting Factor
UCL	Upper Confidence Limit
UCR	Upper Columbia River
UDU	Upland Decision Unit
UF	Uncertainty Factor
UF_A	Interspecies (Animal to Human) UF
UF_D	Incomplete to Complete Database UF
UF_H	Intraspecies (Interhuman) UF
UF_L	LOAEL to NOAEL extrapolation UF
UFP	Uniform Federal Policy
UF_S	Subchronic to Chronic UF
UL	Upper Limit
U.S.	United States
USBR	U.S. Bureau of Reclamation
USGS	U.S. Geological Survey
UTL95-95	95% Upper Tolerance Limit with 95% coverage (also 95UTL95)
VRA	Voluntary Removal Action
WDGER	Washington Division of Geology and Earth Resources
WDOH	Washington State Department of Health

Weston	Weston Solutions, Inc.
WHO	World Health Organization
Windward	Windward Environmental LLC
WOE	Weight of Evidence
WRIA	Water Resource Inventory Area
ww	Wet Weight
XRF	X-ray Fluorescence
YAM	Young America Mine
<	Less than
>	Greater than
≤	Less than or Equal to
≥	Greater than or Equal to
%	Percent
=	Equal to

TABLES

FIGURES

APPENDICES

ATTACHMENTS

Attachment 1: EPA Response to Public Comments Received on April 3, 2020 Public Review Draft Site-wide HHRA for the UCR

NOTE TO READERS

This report presents the results of the human health risk assessment (HHRA) for the Upper Columbia River (UCR) Site. The report is a technical document intended for risk assessors and risk managers rather than a risk communication tool for the general public. It is part of the Remedial Investigation/Feasibility Study (RI/FS) for the Site, which is a detailed study to determine the extent of pollution at the Site and evaluate clean-up options if necessary. This report was prepared to evaluate the need for and potential types of actions that may be required to reduce risks to people from pollution at the Site. The report uses standard risk assessment terminology and follows conventions found in Environmental Protection Agency (EPA) guidance such as the *EPA Risk Assessment Guidance for Superfund* (EPA, 1989). Acronyms and abbreviations are provided on Pages v to x. Clarifications to some terminology are provided in this Note to Readers.

The report provides information on selected environmental media: soil, fish, sediment, river water (surface water), air, crayfish, mussels, and plants. These are referred to in the report as "exposure media." These media were analyzed by laboratories to determine the concentrations of hazardous chemicals in each medium. These chemicals are referred to in the report as "chemicals of potential concern," or "COPCs."

Risks from exposure to COPCs were evaluated for hypothetical populations that represent subsets of populations of people who live near the UCR, visit or work on its beaches, or eat fish, shellfish, game, or cultural/wild plants harvested from the area. These hypothetical populations are referred to in the report as "receptors." Receptor populations are assigned exposure parameters (e.g., intakes of selected media, exposure frequencies and durations) to represent typical (most likely) exposures, referred to as "central tendency exposures" (CTE), or greater than typical exposures, referred to as "reasonable maximum exposures" (RME). Receptor populations are hypothetical because they are restricted to exposures to selected media and defined media intakes; therefore, they may not represent any single individual or real population, whose members engage in a broader diversity of activities and intensities (duration, frequency) than assumed for the receptor population. For example, the outdoor worker receptor is assumed to be exposed exclusively during occupational activities. However, workers may live in the area and may recreate at the UCR. The recreational visitor receptor is assumed to be exposed exclusively during intermittent visits to the Site. However, some recreational visitors may also be residents or outdoor workers.

Although a more complex exposure scenario (work plus residence plus recreation) has not been directly assessed in this HHRA, the risks for contributing exposures (work, or residence, or recreation) provide some information about risks of combined exposures. For example, if risks from residential exposure exceed risk benchmarks, then risks for the resident who works at the Site are also likely to be of concern, but not necessarily from exposures at work. Risks for individual receptors should not be summed to estimate risks for "hybrid" receptors (e.g., resident-recreator-worker) because exposures from each exposure scenario do not necessarily sum (e.g., a person at work is not at home or recreating).

The five categories of receptors (hypothetical populations) evaluated in this risk assessment are:

1. Residents (who live in the area now or could in the future),
2. Recreational visitors to the river,
3. Outdoor workers at public beaches,
4. Colville high intensity resource users, and
5. Residents of the Spokane Tribe of Indians (STI) Reservation (the Spokane Reservation).

Receptor populations 4 and 5 above are populations whose diet consists largely of fish, game, and cultural/wild plants collected locally. These two populations are evaluated separately, and the results are reported in Appendices 1 and 2.

Receptor populations represent plausible exposure scenarios at the Site, rather than individuals. The exposure scenarios are the ways in which people could be exposed to Site-related chemical contamination. “Exposure pathways” in this report include eating fish, other animals, or plants (“consumption”); breathing the air (“inhalation”); touching soil, sediment, or surface water (“dermal contact”); and/or accidentally getting soil/dust, sediment, or surface water in their mouths and swallowing it (“incidental ingestion”).

Risks to receptor populations are estimated at specific places, referred to in this report as exposure areas. An exposure area is an area within which the receptor has an equal probability of being exposed to contaminants. In the residential soil sampling program, these exposure areas were termed Decision Units (DUs). The assumption of random exposure in each exposure area is the basis for using an average concentration to represent the exposure concentration within the exposure area (referred to in this report as the exposure point concentration [EPC]). Examples of exposure areas evaluated in this assessment include DUs on residential properties as well as individual private and public beaches. Here again, the EPC applies to receptor populations, recognizing that it may not represent individuals or subpopulations who do not have an equal probability of being exposed to contaminants in the exposure area (i.e., who preferentially spend time at certain locations within the exposure area). In order to assess such subpopulations, the exposure areas and receptor definitions would have to be reconstructed.

EPA equations and models are used to estimate risks to receptors from exposures to COPCs by comparing calculated exposures to risk-based thresholds. For lead, this is done by using computer models to estimate concentrations of lead in blood and comparing those blood lead concentrations (PbBs) to risk benchmarks. Because the science and regulatory environment regarding lead toxicology is still evolving, three lead risk benchmarks are used in this evaluation, representing a range of PbBs. For COPCs other than lead (referred to as “non-lead COPCs”), risks are separately calculated for cancer and non-cancer health outcomes. For cancer, risks are calculated as the increased probability of an individual getting cancer from exposure to a chemical. For non-cancer outcomes, risks are calculated as how much the estimated receptor exposures exceeds (or does not exceed) an exposure that is assumed to be safe based on toxicological studies. Some non-lead COPCs may have the potential to produce both cancer and non-cancer effects; for these chemicals, risk is calculated both ways. Lead and non-lead COPCs are assessed and presented separately in this report because they are evaluated using different methods. Lead is the main soil contaminant at the Site because most soil DUs that exceeded risk

benchmarks are attributable to lead exposures. Therefore, risks from soil exposure estimated for non-lead COPCs may be considered in the context of lead risk (e.g., those exposure areas that exceed lead risk benchmarks and also exceed non-lead risk benchmarks).

Risk estimates are the result of several calculations performed on various types of data. The final result is presented with one significant digit (e.g., "0.9," "1," "2"). Intermediate values in calculations carry additional digits to minimize rounding errors. These final result values are compared to benchmarks. The following are examples of how calculated results are reported as one significant digit and then compared to the non-cancer benchmark of 1:

- Calculated result of 0.92 is reported as 0.9 – it does not exceed benchmark
- Calculated result of 0.96 is reported as 1 – it does not exceed benchmark
- Calculated result of 1.3 is reported as 1 – it does not exceed benchmark
- Calculated result of 1.5 is reported as 2 – it exceeds benchmark

The comparison of risk estimates to benchmarks provides a means to categorize risk as a concern (i.e., within the risk range or above the threshold) or not (i.e., below the threshold). Consistent with other EPA risk assessments, this assessment includes numerous health-protective assumptions (described in the report) that may result in an overestimation of risk. This is done to ensure that there is little chance of concluding that there is little or no risk at a DU, beach, or other exposure area, when risks are actually above a level of concern (false negative decision error).

The report contains a large number of tables and figures presenting available concentration data, parameters used to conduct the risk assessment, and results for all of the receptor populations and exposure areas. These tables and figures are at the end of the report, rather than embedded in the text where they are first referenced.

A draft of this report was made publicly available on April 3, 2020. EPA held two public meetings (via webinar) on June 10 and July 15, 2020, to provide an overview of the draft report and answer questions. Comments on the draft HHRA were accepted through August, 2020. Comments and responses, webinar questions and answers, and letters received and replied to, are provided in Attachment 1 to this HHRA.[2]

[2] Appendix 2 was under development and not included in the Public Review Draft of this report or the public comment period.

EXECUTIVE SUMMARY

This report presents the Site-wide human health risk assessment (HHRA) for the Upper Columbia River (UCR) Site, which is in northeast Washington State and includes approximately 150 river miles of the Columbia River, extending downstream from the United States (U.S.) – Canadian border south to the Grand Coulee Dam. The Site includes land and waters within the boundaries of the Colville Reservation and the Spokane Reservation, as well as land and waters administered by the National Park Service and Bureau of Reclamation within the U.S. Department of the Interior (U.S. Environmental Protection Agency [EPA], 2006a). This HHRA was conducted as part of an ongoing remedial investigation and feasibility study (RI/FS) in response to concerns regarding historical discharges of hazardous substances into the Columbia River as a result of smelting processes and facility operations by Teck Metals Ltd. ("Teck") and its affiliated predecessors at the facility in Trail, British Columbia (B.C.). This work is being performed under a Settlement Agreement signed by the U.S. and Teck American Incorporated (TAI) in 2006 (EPA, 2006a).

Multiple rounds of data have been collected at the Site over the past 15 years, including samples of UCR surface water, beach sediment, soil, air, and tissue (fish, macroinvertebrates, and plants). Site-related chemicals of interest (COIs) sampled in these media were screened against risk-based screening levels (RBSLs) to determine the chemicals of potential concern (COPCs) for each medium. The COPCs evaluated in this HHRA are metals in UCR surface water; surface sediment at public beaches, and residential beaches[3]; surface soil from relict floodplains and adjacent to Bossburg Flat Beach ("beach soil"); subsurface sediment and subsurface beach soil on public beaches; surface soil in upland areas and on residential properties; air; indoor dust; plants; and waterfowl, upland birds, and wild game (mammals). COPCs in fish and macroinvertebrate tissue include some organic chemicals as well as metals.

Exposures to these COPCs were evaluated for the following receptor populations:

- *Residents* who contact soil in their yards and dust in their homes, who breathe air, who may eat fish from the UCR, and who may contact UCR surface water and sediment if they have a beach on their property;
- *Outdoor workers* who contact surface and subsurface sediment or beach soil at public beaches, who contact UCR surface water, and who breathe air;[4]
- *Recreational visitors* who use the UCR for beach day trips, boating, camping, swimming, and fishing and thereby contact surface sediment and beach soil at public beaches and relict floodplains, who contact UCR surface water, who breathe air, and who may eat fish harvested from the UCR;

[3] The term "residential beach" is used in this HHRA to mean a beach on private property that was sampled as part of the 2014 or 2016 Residential Soil Studies.

[4] Workers in the area may also work full-time in upland areas away from the river (e.g., in the forest or agricultural industries). Upland areas are evaluated for residential land use in this HHRA; the potential future residential exposure evaluation is protective of the upland outdoor worker population and a separate evaluation for upland workers is not warranted.

- *Colville High Intensity Resource Users* (CHIRU) who have contact with soil, beach sediment, and UCR surface water, who breathe air, and who may eat fish, mussels, crayfish, amphibians/reptiles, waterfowl, wild/cultural plants, upland birds, and wild game (mammals) harvested from the Site; and
- *Spokane Tribe of Indians* (STI) who have contact with soil, beach sediment, and UCR surface water, who breathe air, and who may eat fish harvested from the UCR.

Residential and outdoor worker populations are commonly evaluated in HHRAs. The recreational visitor and tribal populations at the Site, however, are more unique. To inform potential exposures of these populations, two surveys were conducted at the Site. A Site-specific Recreational Consumption and Resource Use Survey ("RecUse Survey") was conducted and provided estimates of frequency of use and fish consumption that were incorporated into this HHRA (Industrial Economics, Inc. [IEc], 2012; SRC, 2019a). Additionally, a Site-specific Tribal Consumption and Resource Use Survey ("CCT Tribal Survey") was conducted to provide exposure parameters that were used to evaluate the CHIRU population of the Confederated Tribes of the Colville Reservation (CCT) (Westat Inc., 2012; SRC, 2019b). The HHRA for the CHIRU population of the CCT is presented in Appendix 1, and the HHRA for the STI population is presented in Appendix 2. Exposure parameters provided by the STI from the 2006 STI Hazardous Substances Control Act (STI HSCA, 2006) were used to evaluate the STI population. Risk estimates in these two appendices may also be informative to other non-tribal residents and visitors to the area who engage in similar activities. While Appendix 1 evaluates the high-intensity resource users within the CCT population, the non-subsistence CCT population is represented by the residential population evaluated in this HHRA.

The following is a summary of the results of this HHRA:

Receptor[1]	**Lead[2]**	**COPCS other than Lead**		**Major Contributors to Risk**
		Non-cancer	**Cancer**	
Current resident without beach	↑ P8[3]	↑	↓	• Lead in soil • Methylmercury[5], thallium and dioxins and dioxin-like polychlorinated biphenyls (PCBs) in fish
Current resident with beach	↑ P5	↑	↓	• Lead in soil • Methylmercury, thallium and dioxins and dioxin-like PCBs in fish
Potential future resident	↑ P8	↑	↓	• Lead in soil • Methylmercury, thallium and dioxins and dioxin-like PCBs in fish

[5]Mercury in fish tissue was analyzed using methods that only measure total mercury. Because the majority of mercury in fish is present as methylmercury, as explained in detail in Section 3, mercury in fish is referred to as methylmercury in this HHRA.

Receptor[1]	Lead[2]	COPCS other than Lead		Major Contributors to Risk
		Non-cancer	Cancer	
Recreational visitor, public beach sediment	↑ P5	↑	↓	• Lead in sediment • Methylmercury, thallium and dioxins and dioxin-like PCBs in fish
Recreational visitor, public beach soil	↑ P8	↑	↓	• Lead in soil • Methylmercury, thallium and dioxins and dioxin-like PCBs in fish
Recreational visitor, relict floodplain soil	↑ P3	↑	↓	• Lead in soil • Methylmercury, thallium and dioxins and dioxin-like PCBs in fish
Outdoor worker	↑ P3	↓	↓	• Lead in beach soil and sediment

↑ at least one DU was above risk benchmark
↓ no DUs above benchmark
[1]Results for CHIRU and STI receptor populations are found in Appendices 1 and 2, respectively.
[2]All scenarios have greater than 5% probability of exceeding a blood lead level of 3 micrograms per deciliter (µg/dL) (P3).
[3]"P3," "P5," "P8:" Indicates the level of lead risk, not including fish. P3 = probability of exceeding a blood lead level of 3 µg/dL is >5%; P5 = probability of exceeding a blood lead level of 5 µg/dL is >5%; P8 = probability of exceeding a blood lead level of 8 µg/dL is >5%.

The following generalizations can be made based on the results of the HHRA for the residential, recreational, and worker populations:

- Residential exposure to soil sampled from 588 residential areas and 142 larger randomly selected areas exceeded lead and non-lead risk benchmarks. Removal actions were taken between 2015 and 2018 at 18 properties that were heavily contaminated. Lead in soil poses the greatest risk, the soil is generally more contaminated in areas that are closer to the international border and closer to the river, and undeveloped lands are generally more contaminated than developed (residential) land.
- Residents in impacted areas are advised to follow state recommendations on reducing exposure to lead.[6]
- None of the three metals (arsenic, cadmium, and lead) monitored in air from 2002 to 2009 near Northport or at the international border exceeded risk benchmarks.
- Open public beaches and the UCR are safe for recreation. Bossburg Flat Beach is closed to the public due to high lead levels, and the State of Washington is remediating the Northport waterfront. Human health risks from recreating in river water and sediment are low in other areas of the river.
- Fish, other than sucker, have low levels of lead. Consumption of some fish species contributes to the potential for non-cancer effects in children. Aside from sucker, which

[6] https://ecology.wa.gov/Spills-Cleanup/Contamination-cleanup/Dirt-Alert-program/Healthy-actions.

contributes to unacceptable lead risk, fish consumption is primarily a concern due to methylmercury (which may have developmental effects and effects on the nervous system) and dioxins and dioxin-like PCBs (which may affect the reproductive system).

- Fish consumers are encouraged to follow the Washington Department of Health Fish Advisories for the UCR and Lake Roosevelt.[7]
- Risk to outdoor workers is minimal. Upland soil does not present a risk to outdoor workers.

Methods Employed

Risks to receptor populations were estimated at specific places, referred to in this report as exposure areas. An exposure area is an area within which the receptor has an equal probability of being exposed to contaminants. In the residential soil sampling program, these exposure areas were termed Decision Units (DUs). The assumption of random exposure in each exposure area is the basis for using an average or high-end concentration (for lead and other chemicals, respectively) to represent the exposure concentration within the exposure area (referred to in this report as the exposure point concentration [EPC]). Examples of exposure areas evaluated in this assessment include DUs on residential properties as well as individual private and public beaches.

Two pathways of potential exposure to COPCs that were not evaluated quantitatively in this HHRA were consumption of cultivated garden fruits and vegetables by residents, and inhalation of sweat lodge air by residents of the Colville and/or Spokane Reservations. Risk to residents consuming cultivated/garden plants was not assessed because all non-beach soil DUs from the 2014 and 2016 Residential Soil Studies (CH2MHill, 2016a; TAI, 2017a) were assessed assuming full-time residential exposure, and full-time residential soil exposures are expected to be protective of gardening exposures.[8] People who participate in sweat lodge activities may contact COPCs in UCR surface water that is heated by contact with heated rocks in the sweat lodges. Because non-volatile metals will not vaporize at sweat lodge temperatures, intake of metals would be from ingestion of inhaled water droplets rather than from absorption through the respiratory tract. Relative to ingestion of UCR surface water from direct contact during other water activities such as swimming and wading, ingestion of COPCs in surface water spray in a sweat lodge would be a negligible contributor to risk. As such, this exposure pathway was not quantitatively evaluated (see Appendix 3 for more detailed discussion).

Consistent with other EPA risk assessments, risks from exposure to lead (Pb) in Site media were evaluated and presented separately from risks from exposure to other COPCs. Risks from exposure to lead were assessed using the Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children for residential and recreational exposures, and the Adult Lead Methodology (ALM) for the outdoor worker exposure pathway. Risk benchmarks are risk levels that EPA uses

[7] https://www.doh.wa.gov/Portals/1/Documents/Pubs/334-305.pdf.

[8] The Office of Superfund Remediation and Technology Innovation (OSRTI) determined that this is an acceptable approach for gardens, where direct soil ingestion is the source of the majority of risk. Garden soils tend to have lower soil lead concentrations and lower bioavailability which may result from using soil amendments such as compost and fertilizer (e.g., Brown and Chaney, 2016) in cultivated gardens. Finally, the most sensitive receptor for the lead evaluation is the young child, who is not likely to be an avid gardener.

to distinguish risks that are a potential concern from risks that are below the level of concern. Recognizing the recent advances in lead toxicology (Agency for Toxic Substances and Disease Registry [ATSDR], 2020), this risk assessment has evaluated a range of blood lead levels and the associated soil concentrations within which the risk management decision will most likely be made, from 3 to 8 µg/dL. For this HHRA, the risk evaluation benchmarks selected for elevated lead risk were defined as greater than (>) 5% probability of exceeding a PbB of 3, 5, or 8 µg/dL (referred to as "P3," "P5," and "P8," respectively). The IEUBK model cannot be used with a risk benchmark below P3 (such as P2) because the risk goal would be exceeded even if the soil lead concentration is 0 ppm due to dietary lead exposure.[9] P3 was selected as a lead risk benchmark to quantitatively evaluate the lowest end of the risk range of child PbBs associated with adverse health effects and P8 was selected as a less protective benchmark.

The evaluation used a version of the IEUBK model (version 1.1, build 11) with updates that reflected EPA's Superfund Technical Review Workgroup (TRW) for Lead (Pb) recommended changes to IEUBK version 1.1 input parameter default values, that were based on recent advances in scientific information and will be incorporated into IEUBK (version 2). The differences between IEUBK (version 2) and IEUBK (version 1.1) with the updated input parameter values shown in Tables 3-30 and 3-31 are too small to meaningfully impact the results of the HHRA. An evaluation of the performance of IEUBK (version 2) found strong support for applications of the IEUBK (version 2) in Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA)-related HHRAs (Vandenberg, 2020).

The IEUBK model was used to derive Preliminary Remediation Goals (PRGs) for residential soil lead exposures for each of the three risk benchmarks as follows:

- Utilizing the IEUBK model, a 5% probability of exceeding a PbB of 3 µg/dL was associated with a soil concentration of approximately 50 ppm lead
- Utilizing the IEUBK model, a 5% probability of exceeding a PbB of 5 µg/dL was associated with a soil concentration of approximately 200 ppm lead
- Utilizing the IEUBK model, a 5% probability of exceeding a PbB of 8 µg/dL was associated with a soil concentration of approximately 400 ppm lead

The PRGs associated with each of these risk benchmarks is based on an assumed default relative bioavailability (RBA) of 60% for lead. Site-specific *in vitro* bioavailability (IVBA) information was used to derive RBA-adjusted lead concentrations for samples collected on-Site, which allows direct comparison with these PRGs.

Because the IEUBK model requires a complete exposure scenario, the user must input a residential soil concentration even when the exposure pathway of interest (i.e., exposure to beach sediment) is not residential. Exposure of children to lead in sediment at residential beaches, public beaches, and relict floodplains (beach soil) was therefore assessed using the time-weighted approach recommended by EPA (2003a). This approach used Site-specific exposure frequency (EF) information to apportion exposure between the beach sediment/soil or relict floodplain soil and the soil at the "residence." The residential soil EPC used for lead in this

[9] The estimated background soil lead concentration for this Site is approximately 35 ppm (Appendix 4).

approach was either the average of the lead concentration in the house DU(s) (or the nearest appropriate DU or DUs) on that property (for residential beaches), or the average residential soil EPC for the study area (129 milligrams [mg]/kilogram [kg]; for residential beaches with no associated "house" DU, for public beaches, and for relict floodplains). Surface water exposure (i.e., incidental ingestion of surface water while swimming) and exposure from consuming fish harvested from the UCR were assessed as additional exposures to lead that would occur while recreating at a public or private beach.

For media other than soil, sediment, and surface water, lead risk may be considered in terms of how exposure to the medium (i.e., fish consumption) contributes to lead intake given a selected concentration of lead in residential soil. The IEUBK model predicts that lead intakes of at least 1 µg of lead per day[10] are needed to decrease the soil PRG by 10%. For example, the P5 soil PRG would decrease from approximately 200 ppm to approximately 180 ppm when lead intake because of fish consumption increases from 0 to 1 µg Pb/day. In this HHRA, risk from exposures that were predicted to contribute less than (<) 1 µg lead intake per day (i.e., fish consumption), which would change the PRG by <10%, were classified as minimal.

Risks from exposures to COPCs other than lead ("non-lead COPCs") were estimated using exposure pathways and parameters based on EPA guidance and Site-specific information. Risks were estimated two ways for each receptor: using high-end exposure parameters (termed the "Reasonable Maximum Exposure" [RME]) and using mean or average values for exposure parameters (termed the "Central Tendency Exposure" [CTE]). Risk benchmarks used in this HHRA for non-lead COPCs were as follows: a non-cancer hazard quotient (HQ) >1 or an excess cancer risk $>10^{-4}$ for individual COPCs (EPA, 1997). HQs for individual COPCs were also summed across COPCs within an exposure pathway, and across exposure pathways for a specific exposure scenario, to calculate a hazard index (HI). The risk benchmark for non-cancer hazard based on the HI was HI >1. As recommended in Exhibits 8-2 and 8-3 of EPA's *Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual, Part A* (EPA, 1989), final risk results were presented with one significant digit and compared to benchmarks. Intermediate calculations retained additional digits to minimize rounding errors. The following are examples of how calculated results were reported as one significant digit and then compared to the non-cancer benchmark of 1:

- Calculated result of 0.92 was reported as 0.9 – it does not exceed benchmark
- Calculated result of 0.96 was reported as 1 – it does not exceed benchmark
- Calculated result of 1.3 was reported as 1 – it does not exceed benchmark
- Calculated result of 1.5 was reported as 2 – it exceeds benchmark

The assumption of dose additivity in the HI approach is most relevant to chemicals that induce the same effect by the same mode of action. If an HI >1 because of summing several HQs across pathways, then the chemicals can be segregated by effect and mode of action, and an HI can be calculated for each target organ group (EPA, 1989). Target organ HIs were calculated in this HHRA for receptors and pathways where the HI >1 if summed across COPCs within an

[10] Assuming 30% absolute bioavailability (ABA).

exposure pathway, or across exposure pathways for a specific exposure scenario. The risk benchmark for non-cancer hazard based on target organ HIs was HI >1. Cancer risks were summed across a lifetime to calculate a time-weighted average (TWA) cancer risk. The risk benchmark for TWA excess cancer risk was $>10^{-4}$.

This HHRA considered potential risk from all COPCs. Some COPCs that contribute a large portion of the risk are commonly found in watersheds in Washington State. Examples include dioxins, dibenzofurans, and polychlorinated biphenyls (PCBs). While these chemicals contribute to the estimated total risk to receptor populations of interest at the Site, they do not constitute risks unique to the Site. Additionally, there are uncertainties associated with the COPCs that are the major contributors to risk (such as conservative assumptions made regarding available toxicity data). An analysis of Site-specific background data illustrates that, for lead, consideration of the estimated background concentration of lead in soil at the Site (approximately 35 ppm; see Section 6.4) is unlikely to influence results at P5 or P8 because the P5 and P8 are well above background (approximately 200 or 400 ppm, respectively). However, because the background soil lead concentration is very close to the P3 soil PRG (~50 ppm), consideration of background may be influential for that lead risk benchmark. Measured concentrations of arsenic and antimony also exceeded estimated background concentrations in many DUs and upland aerial deposition areas (ADAs). In addition to arsenic and lead, the COPCs that contribute the most to calculated risk from exposure to soil (thallium, manganese, cobalt, and iron) are below estimated background concentrations.

The risk estimates reported in this HHRA assumed that no additional steps are taken to remediate the environment or to reduce human contact with contaminated environmental media. Subsequent to the collection of residential soil data from the Site in 2014 and 2016, Time Critical Removal Actions (TCRAs) and/or Voluntary Removal Actions (VRAs) were completed on 28 sampled residential soil DUs.[11] Those DUs were included in the evaluations in this report using their post-removal soil concentrations to represent current exposure conditions.

Summary of Results

Risks from exposure to lead and non-lead COPCs by the CHIRU population and the STI population are given in Appendices 1 and 2. Risks for the resident, recreational visitor, and outdoor worker populations are summarized below.

Current Resident Population (Not Beach DUs)

The current resident population was evaluated for exposure to outdoor soil and indoor dust, outdoor and indoor air, and consumption of fish caught from the UCR (evaluated for each species individually). This was done on a DU-by-DU basis (i.e., conservatively assuming that the resident lived full-time on that DU). Table ES-1 summarizes the number of DUs that exceeded risk benchmarks for both non-lead and lead COPCs for the current resident population without a beach on the property. While Appendix 1 evaluates the high-intensity resource users within the CCT population, the non-subsistence CCT population is represented by the residential population evaluated in this HHRA.

[11] List of TCRAs and VRAs is current as of June 20, 2019.

Lead

Of 588 residential DUs evaluated, 389 DUs exceeded the lead benchmark of P3 (66%), 87 DUs exceeded P5 (15%), and 12 exceeded P8 (2%), not including the consumption of fish from the UCR (see Figures 5-1 through 5-3). Consuming fish from the UCR may pose an additional lead risk to current residents (see Appendix 5 for batch mode lead results). As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

COPCs Other Than Lead

For the non-lead evaluation, exposures from each pathway were summed to evaluate cancer and non-cancer effects to the current residential population (without a beach) as a whole. When cancer risk was summed across a lifetime and across exposure pathways for the current residential population (without beaches on their property), no DUs exceeded the cancer risk benchmark of 10^{-4} for either the RME or CTE scenario.

For the non-cancer evaluation for the current adult resident population, no exposure pathway on its own resulted in exceedances of target organ risk benchmarks under either the RME or CTE scenario. When these pathways were summed to look at total exposure to current adult residents without beaches on their property, one house DU had a target organ HI >1 for the skin/hair/nails system when the fish species consumed was Walleye (this DU also exceeded the lead risk benchmark of P8), and all 588 DUs had non-cancer target organ HIs >1 for the nervous system when the fish consumed was sucker (see Figure 5-11). These exceedances occurred under the RME exposure scenario; consumption of all other fish species evaluated did not result in non-cancer benchmark exceedances. When CTE exposure conditions were assumed, no DUs exceeded non-cancer benchmarks for current adult residents (without beaches).

For the non-cancer evaluation for the current child resident population, inhalation of outdoor and indoor air and consumption of Northern Pike or White Sturgeon, as individual exposure pathways, did not result in exceedance of non-cancer benchmarks at any residential DUs under either RME or CTE scenarios. For the exposure to soil/dust pathway, one house DU had a target organ HI >1 for the skin/hair/nails system with RME exposure assumptions but did not exceed benchmarks with CTE exposure assumptions. This house DU exceeds the lead risk benchmark of P8 as well, as shown in Figure ES-1.

Under the RME scenario for the fish consumption pathway for the current child resident population, consumption of Burbot resulted in target organ HIs >1 for the developmental and nervous systems; consumption of kokanee or Rainbow Trout resulted in the skin/hair/nails target organ system HI >1; consumption of Smallmouth Bass or Walleye resulted in target organ HIs >1 for the developmental, nervous, and skin/hair/nails systems; sucker consumption resulted in developmental, nervous, and reproductive target organ system HIs >1; and consumption of whitefish resulted in target organ HIs >1 for the reproductive and skin/hair/nails systems. None of these fish consumption exposure pathways resulted in exceedance of non-cancer benchmarks under CTE exposure assumptions. When the exposure pathways were summed together under the RME scenario for the current child residential population, at least one residential DUs had non-cancer target organ HIs >1 for each fish species consumed:

- Consumption of sucker: 588 DUs had HI >1 for developmental, nervous, and reproductive target organ systems; 578 DUs had HI >1 for skin/hair/nails target organ system (see Figure 5-16)
- Consumption of Walleye: 588 DUs had HI >1 for developmental, nervous, and skin/hair/nails target organ systems; 9 DUs had HI >1 for endocrine system; 2 DUs had HI >1 for cardiovascular system (see Figure 5-17)
- Consumption of Smallmouth Bass: 588 DUs had HI >1 for developmental, nervous, and skin/hair/nails target organ systems
- Consumption of whitefish: 588 DUs had HI >1 for nervous, reproductive, and skin/hair/nails target organ systems
- Consumption of Burbot: 588 DUs had HI >1 for developmental and nervous target organ systems; 43 DUs had HI >1 for skin/hair/nails target organ system (see Figure 5-12)
- Consumption of Northern Pike: 588 DUs had HI >1 for nervous and skin/hair/nails target organ systems
- Consumption of Rainbow Trout: 588 DUs had HI >1 for skin/hair/nails target organ system; 577 DUs had HI >1 for nervous system (see Figure 5-14)
- Consumption of kokanee: 588 DUs had HI >1 for skin/hair/nails target organ system; 219 DUs had HI >1 for nervous system (see Figure 5-13)
- Consumption of White Sturgeon: 588 DUs had HI >1 for nervous system; 18 DUs had HI >1 for skin/hair/nails target organ system (see Figure 5-15)

The major contributor to skin/hair/nails target organ risk was thallium, and the major contributor to developmental and nervous system target organ risk was methylmercury.

Under CTE exposure assumptions, when all exposure pathways were summed, no target organ HIs exceeded 1 when the fish species consumed was Burbot, Northern Pike, Rainbow Trout, Smallmouth Bass, White Sturgeon, sucker, or whitefish. Target organ HIs >1 at one house DU for the skin/hair/nails system when the fish consumed was kokanee or Walleye (see Figure 5-18). This DU exceeded the lead risk benchmark of P8.

Current Resident Population (Beach DUs)

The current resident-with-beach population was evaluated at 21 residential beaches sampled in 2014 and 2016 for exposure to outdoor soil and indoor dust, surface sediment, UCR surface water, outdoor and indoor air, and consumption of fish caught from the UCR (by individual fish species). Table ES-2 summarizes the number of DUs that exceeded risk benchmarks for both non-lead and lead COPCs for the current resident population with a beach on the property without including consumption of fish. For COPCs other than lead, fish consumption drives the non-cancer risk results, and the major contributors to the increased risk from fish ingestion were methylmercury, thallium, and total toxic equivalents (TEQ). Beach sediment is less contaminated than soil.

Lead

Of 21 residential beach DUs evaluated, 19 exceeded the lead risk benchmark of P3, and 5 exceeded P5 not including consumption of local fish (see Figure 5-7). No residential beach DUs exceeded P8. Consuming fish from the UCR may pose an additional risk to current residents with beaches (see Appendix 5 for lead batch mode results). As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

COPCs Other Than Lead

For the non-lead evaluation, exposures from each pathway were summed to evaluate both cancer and non-cancer effects to the current residential population (with a beach) as a whole, by beach DU. When cancer risk was summed across a lifetime and across exposure pathways for the residential population, no residential beach DUs exceeded the cancer risk benchmark for either the RME or CTE scenario.

For the non-cancer evaluation for the adult resident population (with residential beaches), no exposure pathway on its own resulted in exceedances of target organ risk benchmarks under either the RME or CTE scenario. When these pathways were summed to look at total exposure to current adult residents with beaches on their property, all residential beach DUs had non-cancer target organ HIs >1 for the nervous system when the fish consumed was sucker. These exceedances occurred under the RME exposure scenario, and the major contributor to risk was methylmercury. Consumption of all other fish species evaluated did not result in non-cancer benchmark exceedances. When CTE exposure conditions were assumed, no DUs exceeded non-cancer benchmarks at any residential beach DU.

For the non-cancer evaluation of the child resident-with-beach population, the only individual exposure pathways with target organ HIs >1 under the RME scenario were consumption of individual fish species other than Northern Pike and White Sturgeon. Consumption of Burbot resulted in target organ HIs >1 for the developmental and nervous systems; consumption of kokanee or Rainbow Trout resulted in the skin/hair/nails target organ system HI >1; consumption of Smallmouth Bass or Walleye resulted in target organ HIs >1 for the developmental, nervous, and skin/hair/nails systems; sucker consumption resulted in developmental, nervous, and reproductive target organ system HIs >1; and consumption of whitefish resulted in target organ HIs >1 for the reproductive and skin/hair/nails systems. None of these fish consumption exposure pathways resulted in exceedance of non-cancer benchmarks under CTE exposure assumptions. When all exposure pathways (air, water, sediment, soil/dust, and fish) were summed together for the child resident with a beach, all 21 residential beach DUs had non-cancer target organ HIs >1 under RME assumptions for:

- The nervous system for consumption of all individual fish species except kokanee and Rainbow Trout. Twenty residential beach DUs had HIs >1 for consumption of Rainbow Trout.
- The developmental system for consumption of Burbot, Smallmouth Bass, sucker, and Walleye
- The reproductive system for consumption of sucker and whitefish

- The skin/hair/nails system for consumption of kokanee, Northern Pike, Rainbow Trout, Smallmouth Bass, sucker, Walleye, and whitefish. One residential beach DU also had HI >1 for the skin/hair/nails system for consumption of Burbot and White Sturgeon.

For the CTE scenario, no target organ HIs were >1 for children at residential beach DUs when exposure pathways were summed.

Potential Future Resident Population

The potential future resident population was evaluated for exposure to outdoor soil and indoor dust, outdoor and indoor air, and consumption of fish caught from the UCR (evaluated as individual fish species). This was done on an ADA-by-ADA basis (i.e., conservatively assuming that the potential future resident lived full-time on that ADA). Table ES-3 summarizes the number of ADAs that exceeded risk benchmarks for both non-lead and lead COPCs for the potential future resident population.

Lead
Of 142 upland ADAs evaluated, 139 ADAs exceeded the lead benchmark of P3 (98%), 68 ADAs exceeded P5 (48%), and 15 exceeded P8 (11%) not including consumption of UCR fish (see Figures 5-4 through 5-6). Consuming fish from the UCR may pose an additional risk to potential future residents (see Appendix 5 for lead batch mode results). As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

COPCs Other Than Lead
For the non-lead evaluation, exposures from each pathway were summed to evaluate both cancer and non-cancer effects to the potential future residential population as a whole. When cancer risk was summed across a lifetime and across exposure pathways for the potential future residential population, no ADAs exceeded the cancer risk benchmark of 10^{-4} for either the RME or CTE scenario.

For the non-cancer evaluation for the future adult resident population, no exposure pathway on its own resulted in exceedances of target organ risk benchmarks under either the RME or CTE scenario. When these pathways were summed to look at total exposure to future adult residents, all 142 ADAs had non-cancer target organ HIs >1 for the nervous system when the fish consumed was sucker. These exceedances occurred under the RME exposure scenario; the major contributor to risk was methylmercury. Consumption of all other fish species evaluated did not result in non-cancer benchmark exceedances. When CTE exposure conditions were assumed, no DUs exceeded non-cancer benchmarks for potential future adult residents.

For the non-cancer evaluation for the future child resident population, inhalation of outdoor and indoor air and consumption of Northern Pike or White Sturgeon, as individual exposure pathways, did not result in exceedance of non-cancer benchmarks at any ADAs under either RME or CTE scenarios. For the exposure to soil/dust pathway, two ADAs had a target organ HI >1 for the skin/hair/nails system with RME exposure assumptions but did not exceed benchmarks with CTE exposure assumptions. These two ADAs exceed the lead risk benchmark of P8 as well, as shown in Figure ES-2.

Under the RME scenario for the fish consumption pathway for the future child resident population, consumption of Burbot resulted in target organ HIs >1 for the developmental and nervous systems; consumption of kokanee or Rainbow Trout resulted in the skin/hair/nails target organ system HI >1; consumption of Smallmouth Bass or Walleye resulted in target organ HIs >1 for the developmental, nervous, and skin/hair/nails systems; sucker consumption resulted in developmental, nervous, and reproductive target organ system HIs >1; and consumption of whitefish resulted in target organ HIs >1 for the reproductive and skin/hair/nails systems. None of these fish consumption exposure pathways resulted in exceedance of non-cancer benchmarks under CTE exposure assumptions. When the exposure pathways were summed together under the RME scenario, at least some ADAs had non-cancer target organ HIs >1 for each fish species consumed:

- Consumption of sucker: 142 ADAs had HI >1 for developmental, nervous, reproductive, and skin/hair/nails target organ systems
- Consumption of Walleye: 142 ADAs had HI >1 for developmental, nervous, and skin/hair/nails target organ systems; 3 ADAs had HI >1 for the endocrine target organ system; 1 ADA had HI >1 for cardiovascular system (see Figure 5-22)
- Consumption of Smallmouth Bass: 142 ADAs had HI >1 for developmental, nervous, and skin/hair/nails target organ systems
- Consumption of whitefish: 142 ADAs had HI >1 for nervous, reproductive, and skin/hair/nails target organ systems
- Consumption of Burbot: 142 ADAs had HI >1 for developmental and nervous target organ systems; 55 ADAs had HI >1 for skin/hair/nails target organ system (see Figure 5-19)
- Consumption of Northern Pike and Rainbow Trout: 142 ADAs had HI >1 for nervous and skin/hair/nails target organ systems
- Consumption of kokanee: 142 ADAs had HI >1 for skin/hair/nails target organ system; 122 ADAs had HI >1 for nervous system (see Figure 5-20)
- Consumption of White Sturgeon: 142 ADAs had HI >1 for nervous system; 21 ADAs had HI >1 for skin/hair/nails target organ system (see Figure 5-21)

The major non-lead contributors to risk were methylmercury (developmental and nervous systems), thallium (skin/hair/nails system), and dioxins and dioxin-like PCBs (reproductive system).

Under CTE exposure assumptions for the future child resident, when all exposure pathways were summed, no target organ HIs were >1 when the fish species consumed was Burbot, kokanee, Northern Pike, Rainbow Trout, Smallmouth Bass, White Sturgeon, or whitefish. Target organ HIs were >1 at two ADAs for the nervous system when the fish consumed was sucker, and one ADA had a target organ HI >1 for the skin/hair/nails system when Walleye was the fish species consumed (see Figure 5-23). One of the ADAs with nervous system HI >1 when sucker was consumed exceeded the lead risk benchmark of P8, and the other ADA exceeded the lead risk benchmark of P3. The ADA with the skin/hair/nails HI >1 when Walleye was consumed

exceeded the lead risk benchmark of P8. The major non-lead contributors to risk were methylmercury (developmental, nervous system) and thallium (skin/hair/nails).

Recreational Visitor Population

The adult and child recreational visitor population was evaluated for exposure on beach day trips, boating and camping trips to UCR surface water, outdoor air, public beach surface sediment or beach surface soil, relict floodplain surface soil, and consumption of fish caught in the UCR (evaluated by individual species).

Lead

For day trips to public beaches, the lead risk benchmark of P3 was exceeded at all public beaches with or without consumption of fish. As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead. Lead batch mode results for individual fish species are presented in Appendix 5. The P5 benchmark was only exceeded at Bossburg Flat Beach (based on 2011 sampling and 2013 reanalysis data) with or without fish consumption (Figure 5-8). No public beaches exceeded P8. These lead results are the same for boating and camping trips as well. Consuming fish from the UCR may pose an additional lead risk to recreational visitors. For beach day trips, camping and boating trips to Bossburg Flat Beach and exposure to surface soil in upland DUs (UDUs), all UDUs exceeded P3 and one UDU exceeded P5 and P8 with or without consumption of UCR fish. For relict floodplains, with or without UCR fish consumption, all relict floodplain deposition areas (RFDAs) exceeded the lead risk benchmark of P3, and no RFDAs exceeded P5 or P8.

COPCs Other Than Lead

For the non-lead evaluation, exposures from each pathway were summed to evaluate both cancer and non-cancer effects to the recreational visitor population as a whole. When cancer risk was summed across a lifetime and across exposure pathways for the recreational visitor population, no public beaches or relict floodplains exceeded the cancer risk benchmark regardless of trip type for both the RME and CTE scenarios.

For the non-cancer evaluation for the adult recreational visitor population, no exposure pathway on its own resulted in exceedances of target organ risk benchmarks under either the RME or CTE scenario. When these pathways were summed to look at total exposure to adult recreational visitors, there were no target organ HIs >1 at any public beach, SDU, RFDA, or UDU for the RME and CTE scenarios, regardless of trip type.

The only individual exposure pathway that exceeded non-cancer risk benchmarks under the RME scenario for target organ HIs for the child recreational visitor population was the consumption of fish species except Northern Pike and White Sturgeon. For the RME scenario, consumption of fish species resulted in non-cancer target organ HIs >1 for the following systems at each public beach, SDU, RFDA, and UDU for each trip type:

- Developmental system: Burbot, Smallmouth Bass, sucker, and Walleye
- Nervous system: Burbot, Smallmouth Bass, sucker, and Walleye
- Reproductive system: sucker and whitefish

- Skin/hair/nails system: kokanee, Rainbow Trout, Smallmouth Bass, Walleye, and whitefish

When exposure pathways were summed, there were no target organ HIs >1 at any public beach, SDU, relict floodplain, or UDU (regardless of trip type) for the child recreational visitor, under RME exposure assumptions, when the fish species consumed was Northern Pike or White Sturgeon. There were non-cancer target organ HIs >1 under the RME scenario at each public beach, SDU, relict floodplain, or UDU (regardless of trip type) for the developmental, nervous, reproductive, and skin/hair/nails system for the child recreational visitor consuming the following species:

- Developmental system: Burbot, Smallmouth Bass, sucker, and Walleye
- Nervous system: Burbot, Smallmouth Bass, sucker, and Walleye
- Reproductive system: sucker and whitefish
- Skin/hair/nails system: kokanee, Rainbow Trout, Smallmouth Bass, Walleye, and whitefish

The major non-lead contributors to risk were methylmercury (developmental and nervous systems), thallium (skin/hair/nails system), and dioxins and dioxin-like PCBs (reproductive system).

For the CTE scenario, none of the exposure pathways evaluated had target organ HIs >1 for child recreational visitors at any public beach, SDU, RFDA, or UDU (regardless of trip type). No target organ HIs >1 at any public beach, SDU, RFDA, or UDU for child recreational visitors when exposure was summed across pathways, regardless of trip type.

Outdoor Worker Population

The adult outdoor worker population was evaluated for exposure to surface and subsurface sediment or soil at public beaches, UCR surface water, and outdoor air. This included each public beach sampled in 2009-2011, 2013, and 2015.

Lead
Subsurface sediment or soil exposure at public beaches exceeded P3 at three exposure areas at Bossburg Flat Beach and did not exceed P5 or P8.

COPCs Other Than Lead
Under both the RME and CTE scenarios, non-cancer target organ HIs did not exceed the non-cancer benchmark of 1 at any public beach for the outdoor worker population. Cancer risks summed across exposure pathways were below the cancer risk benchmark of 10^{-4} at all public beaches evaluated.

Exposure to Air at the UCR Site

Exposure to Site-related COPCs in air has been a public concern at the Site because of the source of contamination. However, as discussed in detail in the body of the report, exposure to COPCs

in the air pathway alone did not exceed any risk benchmarks for lead, non-cancer, or cancer. The concentration of lead in UCR air is an order of magnitude lower than the default air lead concentration in the IEUBK model. Air data used in this HHRA were collected in 2009 near the Highway 25 Bridge in Northport. However, emissions from the Trail smelter as reported to the National Pollutant Release Inventory from 2002 to 2017 show that while individual metals may be emitted at varying rates, emissions of arsenic and lead have decreased since 2009 (Figure 2-13). Exposure to airborne contaminants from the Teck smelter do not pose an unacceptable risk to Site residents, recreators, or workers.

1 INTRODUCTION

1.1 Overview of the Site

The Upper Columbia River (UCR) Site ("the Site") is located wholly within the state of Washington, in the northeast portion of the state, and includes approximately 150 river miles (RMs) of the Columbia River, extending downstream from the United States (U.S.) – Canada border south and west to the Grand Coulee Dam. The Site "…consists of the areal extent of hazardous substances contamination within the United States in or adjacent to the Upper Columbia River, including the Franklin D. Roosevelt Lake ("Lake Roosevelt"), from the border between the United States and Canada downstream to the Grand Coulee Dam, and all suitable areas in proximity to such contamination necessary for implementation of response actions…" (U.S. Environmental Protection Agency [EPA], 2006a). The Site includes land and waters within the boundaries of the Colville Reservation and the Spokane Reservation, over which the Tribes have civil regulatory jurisdiction, as well as land and waters administered by the National Park Service (NPS) and the Bureau of Reclamation within the U.S. Department of the Interior (DOI) (EPA, 2006a).

The construction of the Grand Coulee Dam, a federal reclamation project, was completed in 1940 on a portion of the Columbia River that forms the southern boundary of the Colville Reservation (U.S. Bureau of Reclamation [USBR], 2006). A multi-purpose project, it provides flood control, irrigation, hydropower production, recreation, stream flows, and wildlife benefits (EPA, 2003b). Located immediately behind the Grand Coulee Dam is Lake Roosevelt, a large reservoir extending approximately 133 RMs north of the dam at full pool and bordered by over 600 miles of shoreline, approximately 312 miles of which are part of the Lake Roosevelt National Recreation Area (LRNRA) (NPS, 2019, EPA, 2003b).

In August 1999, the Confederated Tribes of the Colville Reservation (CCT) petitioned EPA to conduct an assessment of the UCR. The petition expressed concerns about risks to people's health and the environment from contamination in the river. In December 2000, EPA completed a preliminary assessment (Ecology and Environment [E&E], 2000), which indicated that further data collection was warranted. In 2001, EPA conducted an expanded site inspection and collected sediment samples to learn more about the types and amounts of pollution present (EPA, 2003b). The results showed that contamination was present and that a remedial investigation and feasibility study (RI/FS) was warranted.

On December 11, 2003, EPA issued a Unilateral Administrative Order (UAO) under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), directing Teck Metals Ltd. ("Teck") to perform an RI/FS at the Site. The U.S. contends that discharges from the Trail Smelter, situated approximately ten RMs north of the U.S.- Canada border, have contributed to releases of hazardous substances, as defined in CERCLA, at the Site. The U.S. acknowledges that other entities may have contributed to releases of hazardous substances at the Site. While Teck and Teck American Incorporated (TAI) deny that they have liability under CERCLA for the Site, Teck and TAI have offered to enter into this contractual agreement with EPA to perform the tasks set forth in the Settlement Agreement (EPA, 2006a). On June 2, 2006, the U.S., on behalf of the EPA and Department of Justice (DOJ), and TAI signed a Settlement Agreement requiring Teck to perform an RI/FS at the Site (EPA, 2006a). Per

the Settlement Agreement, "This Agreement concerns the Upper Columbia River Site ("Site"), which consists of the areal extent of hazardous substances contamination within the United States in or adjacent to the Upper Columbia River, including the Franklin D. Roosevelt Lake ("Lake Roosevelt"), from the border between the United States and Canada downstream to the Grand Coulee Dam, and all suitable areas in proximity to such contamination necessary for implementation of the response actions described below. The Site may include land and waters within the boundaries of the Colville Reservation and the Spokane Reservation, over which the Tribes have civil regulatory jurisdiction, as well as land and waters administered by the NPS and the Bureau of Reclamation within the DOI. The Parties enter into this Agreement to provide for the implementation of the activities described herein at the Upper Columbia River Site" (EPA, 2006a).

The RI/FS is currently underway in response to concerns regarding historical discharges of hazardous substances into the Columbia River, including but not limited to discharges of granulated slag, liquid effluents, emissions, and accidental spills and "upsets" from smelting processes and facility operations by Teck and its affiliated predecessors at the Trail facility located in Trail, British Columbia (B.C.). In accordance with the Settlement Agreement, TAI will complete the RI/FS and baseline ecological risk assessment (BERA) and EPA will complete the human health risk assessment (HHRA).

1.2 Purpose

This report presents the Site-wide HHRA. The purpose of this HHRA is to assess the potential risks to humans, both under current conditions and expected conditions in the future, from exposure to Site-related contaminants present in environmental media associated with the UCR, assuming no steps are taken to remediate the environment or to reduce human contact with contaminated environmental media.[1] The results of this assessment are intended to help inform risk managers and the public about potential human risks attributable to exposure to Site-related contaminants and to help determine if there is a need for action at the Site (EPA, 1989). The overall management goal is to ensure protection of humans from deleterious effects from exposures to Site-related chemicals for both current and future land uses. This report was developed based on a current understanding of the Site, including nature and extent of contamination, chemicals of interest (COIs), and human exposure scenarios. The methods used to evaluate risks in this HHRA are consistent with current EPA guidelines for HHRA at Superfund sites (EPA, 1989, 1991a, 1991b, 1992, 1997, 2001a, 2002a, 2002b, 2004a, 2004b, 2009a, 2011a, 2014a).

[1] Subsequent to the collection of residential soil data from the Site in 2014 and 2016, Time Critical Removal Actions (TCRAs) and/or Voluntary Removal Actions (VRAs) were conducted on 28 decision units (DUs) as of June 20, 2019 (see Table 2-3). Those DUs were included in the HHRA using post-removal soil concentrations as representative of current exposure conditions, as described in detail in this report.

1.3 Organization

The remainder of this report is organized into the following sections:

Section 2	Description of the Site and a review of data that have been collected to characterize the nature and extent of environmental contamination at the Site.
Section 3	Description of the exposure assessment for human exposure scenarios of potential concern at the Site. Identifies chemicals of potential concern (COPCs) for exposure media associated with those exposure pathways and presents the equations and exposure parameters used to derive estimates of exposure to lead and non-lead COPCs.
Section 4	Summaries of cancer and non-cancer toxicity values used in the assessment, and adjustments for relative bioavailability (RBA).
Section 5	Presentation of estimated cancer risk and non-cancer hazard to humans from exposures to lead and non-lead COPCs at the Site.
Section 6	Discussion of uncertainty associated with the analysis, focusing on influential factors that may lead to possible overestimation or underestimation of risk.
Section 7	Summary of overall findings of risks from exposure to lead and non-lead COPCs at the Site.
Section 8	Full citations for EPA guidance documents, Site-related documents, and scientific publications referenced in this document.

2 SITE CHARACTERIZATION

This section provides a general characterization of the Site, including descriptions of Site history and usage, physical characteristics, and ecological resources. This Site Characterization includes summaries of all Site-related data collected to date.

2.1 Site Location and Description

The Site is in north eastern Washington (Figure 2-1). The Site extends along the Columbia River from the border between the U.S. and Canada downstream to the Grand Coulee Dam (EPA, 2006a). Immediately upstream of the Grand Coulee Dam, the impounded river forms Lake Roosevelt reservoir. At full pool, Lake Roosevelt extends at least 133 miles upriver to U.S. Geological Survey (USGS) RM 730, within 15 miles of the Canadian border, and is bordered by over 600 miles of publicly available shoreline (EPA, 2005a; Lake Roosevelt Forum [LRF], 2006a; NPS, 2019). The Colville National Forest is west of the UCR from the northern border of the Colville Reservation to Marcus Flats. The southeast corner of the Colville National Forest is approximately one mile from the UCR between the Onion Creek confluence and Northport. Highway 25 runs roughly adjacent to the eastern shore of the river from the confluence of the Spokane and Columbia Rivers upstream to Northport, where it crosses the river and continues north to the Canadian border. North of Northport, the Waneta Road continues on the eastern shore to the international border. This portion of the Site is characterized by sparsely developed public and private land. As described in the Settlement Agreement for implementation of the RI/FS, the Site "…consists of the areal extent of hazardous substances contamination within the United States in or adjacent to the Upper Columbia River, including the Franklin D. Roosevelt Lake ("Lake Roosevelt"), from the border between the United States and Canada downstream to the Grand Coulee Dam, and all suitable areas in proximity to such contamination necessary for implementation of response actions…" (EPA, 2006a).

The area surrounding the Site is generally thinly populated and consists of forests, farmland and residential properties. Communities located along Highway 395 to the west of the UCR include Barstow and Boyds. Communities located to the east of the UCR, along Highway 25, include, from north to south, Northport, Evans, Marcus, Kettle Falls, Rice, Daisy, Gifford, Cedonia, Hunters, Fruitland, and Enterprise. The northern extent of the Colville Reservation is approximately 3.5 miles north of Rice but on the opposite shore of Lake Roosevelt. The Colville Reservation borders Lake Roosevelt downstream to the Grand Coulee Dam. This area includes several communities, the largest of which are Coulee Dam, Inchelium, and Keller (EPA, 2003b). The Spokane Tribe of Indians (STI) Reservation (the Spokane Reservation) borders approximately 8 miles of the reservoir to the east, just south of the community of Enterprise and north of the Spokane River. South of the Spokane River downstream to the Grand Coulee Dam are the communities of Seven Bays, Lincoln Mill, and Grand Coulee.

2.2 Physical Setting and Land Use

The physical characteristics of the UCR influence the distribution of potential contaminants released to the Site, potential exposure to those contaminants, and the development and evaluation of potential remedial alternatives. This section presents an overview of Site geology, hydrogeology, hydrology, river reach characteristics, and climate. A detailed description of the

physical setting of the Site and primary land uses by residents and visitors is provided in the HHRA work plan (SRC, 2009).

2.2.1 Physical Setting

2.2.1.1 Geology

The UCR is situated within two geologic provinces: the Okanogan Highlands and the Columbia Basin (Figure 2-2). The UCR is located along the division between the eastern and western Okanogan Highland regions. The Okanogan Highlands, which are typified by rounded mountains and deep, narrow valleys, include both shores of the Columbia River above the confluence with the Spokane River. Below the confluence with the Spokane River, the Columbia Basin borders the southern shore of the Columbia River.

The UCR region was extensively modified by glacial activities during the Pleistocene era. The UCR is located within the footprint of the ancestral glacial Lake Columbia, which formed at least three times during the Pleistocene glacial period. The glacial lake and its tributaries deposited coarser materials interbedded with silt and clay, forming deltas. More recently, with the construction of Grand Coulee Dam and the flooding of Lake Roosevelt, the higher river levels have resulted in saturation of these glaciofluvial terraces and their consequent collapse; more than 300 landslides have been documented along the UCR (Jones et al., 1961).

2.2.1.2 Hydrogeology

Aquifers occur in the Columbia Plateau Basalts (south of the lower reach of Lake Roosevelt) and alluvial deposits adjacent to and in valleys of tributaries to the reservoir. Except for the Columbia Plateau Basalts, much of the Site is underlain by geologic formations that do not yield significant quantities of groundwater for water supply uses (USGS, 1985). Limited local aquifers are present in the Site vicinity in permeable glacial alluvial deposits and in permeable sedimentary rocks (sandstones and limestones) (Whitehead, 1994). The aquifer that supplies the City of Northport is a permeable glacial deposit that contains useable quantities of groundwater. The sand and gravel deposits that comprise this aquifer extend from ground surface to depths greater than (>) 60 meters (m), with static water levels approximately 23 m below ground surface (bgs) (Weston Solutions, Inc. [Weston], 2004a).

Groundwater at the Site occurs in pore spaces between sand and gravel particles of unconsolidated aquifers and in fractures or voids of rock aquifers. These aquifers receive recharge from percolation of precipitation into the ground and leakage from surface water bodies (Whitehead, 1994). Groundwater in the Columbia Plateau Basalts aquifer discharges to Lake Roosevelt at the northern edge of the south-sloping Columbia basalts (Whitehead, 1994). Lower reaches of the Columbia River farther to the south (and outside of the UCR study area) subsequently receive discharge from this extensive basalt aquifer. Groundwater from wells and springs in the Site vicinity (e.g., Fort Spokane spring, EPA, 2007a) is used for public and domestic potable water supply, irrigation, power generation, and industry. The Washington State Department of Health (WDOH, 2006) identified 131 water systems within 5 miles of the UCR and Lake Roosevelt shoreline that utilize groundwater (springs or wells) (Table 2-1).

2.2.1.3 Hydrology

The Columbia River watershed is large and complex, with an area of approximately 260,452 square miles (mi^2) that encompasses parts of Washington, Oregon, Nevada, Utah, Idaho, Wyoming, and Montana, as well as B.C., Canada. The headwaters of the Columbia River are at Columbia Lake in Canal Flats, B.C. The river flows approximately 1,245 miles (approximately 470 miles in Canada) before reaching the Pacific Ocean along the border between Oregon and Washington. The river enters the U.S. in northeastern Washington, just south (downstream) of the confluence with the Pend Oreille River.

Grand Coulee Dam was built to provide flood control, irrigation, and power generation. Construction began in the 1930s and was completed in 1941. In June 1942, the impounded reservoir of Lake Roosevelt reached its full pool water surface elevation of 393 m above mean sea level (USBR, 2006) (393 m National Geodetic Vertical Datum [NGVD] of 1927). Major tributaries that influence hydraulic conditions at the U.S. – Canadian border are the Columbia and Pend Oreille rivers. Principal tributaries that join the UCR within the study area are the Kettle, Colville, Spokane, and Sanpoil rivers.

Flow regimes in the UCR have varied over time. Over the past century, three distinct flow regimes have existed, as described below:

1. **Unregulated (before Grand Coulee Dam or upstream flow control)**. Before flow regulation began, UCR flows were governed by precipitation and runoff, particularly the amount of snowpack and snowmelt. During the unregulated era, the river was free-flowing and subject to large, periodic high-flow (flood) events.
2. **Downstream Control (after construction of Grand Coulee Dam but before upstream flow control**). During the period of downstream control, UCR flows were determined by unregulated upstream flow and water-level regulation at Grand Coulee Dam. Although periodic high-flow events still occurred, the extent of the Lake Roosevelt impoundment and backwater effects in upstream areas were controlled entirely by operations at Grand Coulee Dam.
3. **Regulated (after construction of Grand Coulee Dam and after upstream flow control**). During the contemporary era of regulation (post-1972), river flows are controlled by the operation of upstream dams in addition to management operations at Grand Coulee Dam (EPA, 2007b). As a result of the combined effects of dam operations, the size and frequency of large flood events has been reduced.

2.2.1.4 Flow Regulation across the U.S. – Canadian Border

The UCR has been divided into six reaches that correspond to relatively distinct physiographic units (Figure 2-1). Boundaries for the six reaches were selected based on consideration of distinct geomorphic features (e.g., channel width, sinuosity, confluence with major tributaries), general hydraulic or hydrodynamic characteristics (depth, location of the reservoir pool, riverbed characteristics, flow velocity), and expected differences regarding the principal mechanisms for transport or deposition of particle-bound COIs. Detailed descriptions of each river reach can be found in the HHRA work plan (SRC, 2009) and the RI/FS Work Plan (EPA, 2008); brief descriptions are provided below. As previously described, UCR hydrology changed significantly

with the construction of Grand Coulee Dam and again with the implementation of coordinated flood control operations at upstream dams beginning in 1973. These flow regime differences are expected to have influenced the initial transport of sediment and COIs in the UCR and continue to influence their redistribution. Therefore, the changing nature of flow in the UCR was also considered as the boundaries for river reaches were selected.

- Reach 1 (USGS RM 745 to RM 730)
  Reach 1 extends from the U.S. – Canadian border (USGS RM 745) southward past the city of Northport to USGS RM 730, near Onion Creek (Figure 2-1). The northern section of the reach – approximately 3 miles in length – is relatively shallow and narrow, retaining much of its historical hydraulic characteristics, and flows freely much of the time. The southern section of the reach – approximately 12 miles in length – is just upstream of the Lake Roosevelt reservoir and is influenced by the pool level. This section of the river is a free-running riverine reach, though it may be pool-like depending on the level of Lake Roosevelt.
- Reach 2 (USGS RM 730 to RM 711)
  Reach 2 extends from near Onion Creek (USGS RM 730) to Evans (USGS RM 711) (Figure 2-1). Historically, Reach 2 was a swift riverine reach, running southwest from USGS RM 730, first through a narrow, deep canyon and a series of rapids called the Little Dalles, then broadening slightly over the remainder of the run down to USGS RM 711 (Symonds, 1883). The constriction at Little Dalles was widened as part of Grand Coulee Dam construction efforts (1933 to 1942) by removing a rock island down to 383 m along with part of the southern riverbank (McKay and Renk, 2002). This section of the UCR is inundated by the Lake Roosevelt pool approximately 70 percent (%) of the time (EPA, 2004c). However, currents through the widened canyon are swift at lower pool levels. Although more sinuous than upstream areas, Reach 2 is still a relatively narrow channel with few embayments or shoreline irregularities.
- Reach 3 (USGS RM 711 to RM 699)
  Reach 3 extends from Evans (USGS RM 711) to just downstream of Kettle Falls (USGS RM 699; Figure 2-1). The characteristics of Reach 3 include distinct geomorphic features that are believed to favor deposition (and corresponding chemical transport and fate) under historical and contemporary flow regimes. At USGS RM 710 and again between USGS RM 706 and 707, the UCR thalweg makes two sharp (90-degree) bends while passing through a relatively broad floodplain in the area of Marcus Flats. To the north of the second bend, the Kettle River joins the UCR. The Kettle River is the first significant tributary confluence downstream of the U.S. – Canadian border, with a mean annual flow of approximately 3,000 cubic feet per second (USGS, 2006) (Table 2-2). Between USGS RM 704 and 703, the UCR thalweg descends through a steep, narrow constriction. Prior to the construction of Grand Coulee Dam, this was a powerful series of cascades known as Kettle Falls. Kettle Falls is now typically inundated by the Lake Roosevelt pool. However, during occasions of extreme drawdown (e.g., during construction of the third powerhouse at Grand Coulee Dam), Kettle Falls re-emerges. Downstream of the Kettle Falls constriction, the UCR runs through a relatively straight, narrow channel until the confluence with the Colville River at USGS RM 699.

- Reach 4 (USGS RM 699 to RM 640)
  Reach 4 extends from just downstream of Kettle Falls (USGS RM 699) to just upstream of the confluence with the Spokane River (USGS RM 640). Because of the length and expected differences in sediment and contaminant transport regimes, exposure, and habitat over time, this reach is further divided into two subreaches. Reach 4a extends from USGS RM 699, at the confluence of the Colville River, to USGS RM 676, just upstream of Inchelium. Reach 4b extends from USGS RM 676 to USGS RM 640 near the confluence with the Spokane River. Reach 4b borders the Spokane Reservation. These reaches collectively represent the middle reservoir.

- Reach 5 (USGS RM 640 to RM 617) and Reach 6 (USGS RM 617 to near RM 597)
  Reach 5 extends from USGS RM 640 to USGS RM 617. Within Reach 5, the Spokane River joins the Columbia River at USGS RM 639. Reach 6 extends from USGS RM 617 to the Grand Coulee Dam (near USGS RM 597). Within Reach 6, the Sanpoil River joins the UCR between USGS RM 615 and 614. These reaches collectively represent the Lower Reservoir. Reaches 5 and 6 both border the Colville Reservation. Both reaches can be characterized as lacustrine with slow-moving water.

2.2.1.5 Climate/Meteorology

The UCR study area lies in the rain shadow of the Cascade Mountains. The northern areas of the Site receive about 50 centimeters (cm) of precipitation a year (NPS, 2019). Moving south, the climate becomes far more arid with average annual precipitation at Grand Coulee Dam of approximately 25 cm. This precipitation occurs mostly in the winter and spring, while summer months are generally hot and dry.

The Site is in a transitional climate zone receiving moist marine air, continental air, and dry arctic air. Strong, gusty wind can occur during transitions between continental and marine air masses, mainly in spring and summer (Ferguson, 1996). It has been reported that in particularly warm and dry years, 8 to 20 gusty wind events can occur within the Site. The cool, moist air masses from Pacific storms, which progress eastward, are dramatically different than the hot, dry continental air masses. As the air masses meet, the associated fronts can be very strong. These weather fronts often bring strong, gusty local winds. Analysis of meteorological monitoring data collected along the UCR indicates that the dominant wind directions are from northeast to southwest and from southwest to northeast (DOI, 2006). However, wind direction distributions show strong variation with season and topography.

2.2.1.6 Ecological Resources

Aquatic life, wildlife, and vegetation within the Site are discussed in the HHRA work plan (SRC, 2009), the screening-level ecological risk assessment (SLERA; TAI, 2010a), and the BERA work plan (TAI, 2011a). As discussed above, the climate of portions of the Site and the surrounding area is semi-arid and varies a great deal from one end of the Site to the other (LRF, 2006b), with the southern (lower) portion near Grand Coulee Dam being generally hotter and drier. Vegetation in this area (Grand Coulee Dam to Keller Ferry) includes steppe (bunch grass grassland) and shrub-steppe. Between Keller Ferry and the upper end of the Spokane River Arm at Little Falls Dam is a transition from shrub-steppe to ponderosa pine (*Pinus ponderosa*) forest (Hebner et al., 2000). Areas around the middle and upper reservoir, between the Spokane River

and Kettle Falls, receive approximately 43 to 50 cm of precipitation a year (LRF, 2006b). This area is covered with a dense mix of ponderosa pine and Douglas fir (*Pseudotsuga menziesii*; Hebner et al., 2000; LRF, 2006b). The upper portion of the UCR (i.e., north of Kettle Falls to the U.S. – Canadian border) is primarily forested, consisting of Douglas fir, western larch (*Larix occidentalis*), grand fir (*Abies grandis*), Oregon grape (*Berberis repens*), and red-stem ceanothus (*Ceanothus velutinus*) (UCR Natural Resource Trustee Council, 2009).

2.2.2 Land Use

Land use near the Site is residential, recreational, and commercial, including timber lands. The HHRA work plan (SRC, 2009) provides an extensive description of human settlement, cultural resources, and demographics of the Site. As mentioned above, a portion of the Site is within the Colville Reservation and Spokane Reservation. In addition, the Site area includes several towns and communities that are adjacent to or near the river. Much of Lake Roosevelt has been designated as the Lake Roosevelt National Recreation Area (LRNRA), which is managed by the NPS. The LRNRA attracts more than 1.3 million visitors per year on average (NPS, 2019). Designated recreational uses of the LRNRA include boating, fishing, swimming, wading, camping, canoeing, and hunting. The park has a staff of approximately 72 permanent and seasonal employees and receives around 4,000 hours of volunteer labor annually (Foster, 2019). Maintenance and administrative offices for the park are located in Coulee Dam, Spring Canyon, Fort Spokane, and Kettle Falls (NPS, 2019).

All of Lake Roosevelt is within the federally proclaimed LRNRA, even those portions that are within tribal boundaries. However, through the Lake Roosevelt Cooperative Management Agreement of 1990 (the 5-party agreement), which designates the NPS, USBR, Bureau of Indian Affairs, CCT, and STI as managing partners, those portions of Lake Roosevelt within reservation boundaries are managed by the respective tribal entities (CCT or STI).

Figure 2-3 is a reproduction of an NPS map showing water management zones of the lake and recreational facilities along the UCR (LRF, 2019; EPA, 2007a). Developed areas overseen by the NPS include 22 boat launches, 27 campgrounds, and 3 concessionaire-operated marinas (Seven Bays, Keller Ferry, and Kettle Falls Marinas) to provide moorage, boat rental, fuel, supplies, food service, and other services. Two Rivers Marina, which lies within the LRNRA, is owned and operated by the STI.

The remainder of the Lake Roosevelt shoreline managed by the NPS is undeveloped. The NPS allows camping on any undeveloped shoreline, provided it is at least one-half mile away from developed areas (Foster, 2019). The Colville and Spokane Reservations also provide opportunities for recreational visitors to fish and camp at the UCR (NPS, 2019). Recreational users may include occasional visitors, residents, and tribal members. NPS employees and volunteers are also present at the Site as part of their work responsibilities and may use the Site for recreation on a regular basis.

2.3 Site History

Indigenous people have occupied the vicinity of the UCR Site for more than 10,000 years. Prior to the influx of Canadian and European settlers in the mid-1850s, the ancestors of the twelve

indigenous nations now comprising the CCT (spaƛmuləxʷəxʷ/Methow, Sʔukʷnaʔqín/Okanogan, Sn̓ʕay̓ckstx/Arrow Lakes, Sn̓pʕawílx/Sanpoil, Sx̌ʷy̓ʔiłpx/Colville, Nspiləm/Nespelem, c̓əlámәxәxʷ/Chelan, šniyátkʷәxʷ/Entiat, šnq̓áẇsәxʷ/Moses-Columbia, Np̓ešqʷáẇšәxʷ/Wenatchi, nímípuʔ/Nez Perce, and Snq̓ʷaʔmitx/Palus) were nomadic, following the seasons and their sources of food. Their aboriginal territories were grouped primarily around waterways such as the Columbia River, the Sanpoil River, the Okanogan River, the Snake River and the Wallowa River (CCT, 2008). The STI's ancestors were also a river people, living a semi-nomadic way of life hunting, fishing, and gathering, living along the banks of the Spokane and Columbia rivers and up their tributaries. Their primary diet consisted of what was taken from the waterways in the form of salmon, steelhead, eel, and shellfish.[2]

The Colville Reservation was created by Executive Order in 1872; the Spokane Reservation was created by Executive Order in 1881. Portions of the UCR Site are located within the Colville and Spokane reservations. The present boundaries of the Colville Reservation include approximately 1.4 million acres (2,200 mi^2), including northern and western shorelines of approximately 93 miles of the UCR extending upstream from Grand Coulee Dam (Figure 2-1) (CCT, 2008).

The original north boundary of the Colville Indian Reservation was the Canadian border; this former "North Half" of the Colville Indian Reservation continues to be an important homeland to the CCT. The CCT exercises certain management and regulatory authority in this area from the northern boundary of the current reservation north to the Canadian border, bounded by the Okanogan and Columbia rivers. CCT-owned land and individual tribal members reside on the North Half and use the lands, waters, and natural resources for cultural and subsistence uses as they do on the reservation. In Antoine v. Washington, the Supreme Court affirmed the Colville Tribes' rights to hunt and fish on the North Half (Alexander Antoine v. State of Washington 420 US 194. 1975). The total population of the Colville Reservation in the year 2000 was estimated at 7,600 people (Washington State Office of Financial Management [OFM], 2006).

The Spokane Indian Reservation, located at the confluence of the Spokane and Columbia Rivers, was established on August 18, 1877, as affirmed by Executive Order of President Hayes on January 18, 1881.[3] In 1951, the STI officially became 1 of 574 recognized tribal governments within the U.S. following the passage of their formal Constitution that governs the Tribe to this day.[4] Pursuant to its inherent sovereignty, Constitution, and exercise of federally delegated authority to administer Clean Water Act Section 301(c) and 401, the STI has promulgated and administers federally-approved water quality standards and water quality certifications, respectively, as to all waters within Reservation boundaries, including UCR Site waters within those boundaries.

Many of the modern towns near the Site were founded from the late 1850s through the 1880s as mining communities or as supply centers for the mining districts. A western power shortage associated with World War II led Franklin D. Roosevelt to authorize the Columbia Basin Project, including the Grand Coulee Dam and Banks Lake, a holding reservoir. The implementation of

[2] https://spokanetribe.com/history/.
[3] *Northern Pac. Ry. Co. v. Wismer*, 246 U.S. 283, 288 (Supreme Court of the United States [SCOTUS], 1918).
[4] https://spokanetribe.com/history/.

this project altered the historical, cultural, and natural resources of the UCR, leading to present day conditions. In addition to the fishing and hunting rights the STI retains within its Reservation, the Tribe was granted "paramount use" rights for a portion of Lake Roosevelt for fishing, hunting, and boating when Grand Coulee Dam inundated a portion of the Spokane Reservation creating what is now Lake Roosevelt.[5] The HHRA work plan (SRC, 2009) details known and potential chemical sources near the Site, including mine, mill, and smelting operations; pulp mill operations; Toxics Release Inventory (TRI) sources; water quality discharge permitted sites; and municipal and nonpoint sources.

Mining and mineral processing has been occurring in the UCR region, in both the U.S. and Canada, since at least the late 1800s. Most of the operations in the U.S. portion of the region took place in Stevens and Ferry counties (Orlob and Saxton, 1950; Wolff et al., 2005). Mining activities in the drainage basin also occurred in the Metaline mining district in Pend Oreille County, Washington. As part of the UCR Expanded Site Inspection (ESI) conducted by EPA in 2001 and 2002, EPA collected sediment samples and visited several U.S. mine and mill sites in the northern portion of the study area, including mines and mills along tributaries to the UCR, plus several additional mines and mills located in Republic, Washington to the west and along the Pend Oreille River to the east.

The ESIs and the Phase 1 RI (EPA, 2003b, 2006b, 2006c) documented sediment contamination along the UCR Site from the U.S. – Canada border to the Grand Coulee Dam. Hart Crowser (2013) conducted a soil sampling study in upland areas of the UCR Site as an initial assessment of surface and shallow subsurface conditions in the UCR Valley and demonstrated the presence of elevated heavy metal concentrations in the upper horizons of minimally disturbed soils. In a memorandum resolving disputes raised by TAI (EPA, 2017a), TAI agreed to prepare a technical assessment in response to EPA's Level of Effort (LOE) for Assessment and Estimations of Upland Soils (Background). Final Data Quality Objectives (DQOs) for the background evaluation were summarized in EPA (2018a). Based on these results, EPA concluded that both the smelter in Trail, B.C., and the former Le Roi Smelter in Northport, Washington, are sources of contamination to the UCR Site; however, the Trail smelter was identified as the "primary source of contamination" (EPA, 2003b). The mines and mills along the tributaries to the UCR were not identified as current sources of contamination to the Site.[6] A removal assessment study was conducted at the Young America Mine (YAM; TechLaw Inc., 2012a) and at Bossburg Flat Beach (TechLaw Inc., 2012b) which found no evidence that contamination moved down-river from the YAM mill impoundment. A removal action conducted at the YAM Site consolidated and capped the contaminated material (EPA, 2012a). Except for the Spokane River, Phase 1 sediment sampling by EPA (2005b) near the mouth of selected major UCR tributaries did not identify the presence of notably elevated COI concentrations indicative of major watershed sources of contamination from historical mine and mill sites. In addition, ESI sediment sampling by EPA (2003b) at the mouth of 110 tributaries to the UCR between RM 675 and the U.S. –

[5] 16 U.S.C. § 835d.

[6] Some of these mines and mills had localized contaminant concentrations that met EPA requirements for time-critical and non-time-critical removal actions. The following sites have been addressed under EPA's removal program: Anderson-Calhoun Mine and Mill, Bonanza Mill, LeRoi Smelter, Colville Post and Pole, Cleveland Mine and Mill, Josephine Mill No. 1, Grandview Mine and Mill, and Sierra Zinc Mine and Mill.

Canada border did not identify the presence of elevated COI concentrations indicative of major watershed sources of contamination.

The following subsections provide summary information for the Teck facility in Trail, B.C., and the former Le Roi Smelter in Northport, Washington.

2.3.1 Trail, B.C., Teck Facility

The Teck facility in Trail, B.C., is located on the Columbia River approximately 10 miles upstream from the U.S. – Canada border. Smelter operations have been underway in Trail since 1896 (G3 Consulting, 2001a) to smelt copper and gold ores from the Rossland Mines (G3 Consulting, 2001b). On-site operations thermally extracted gold and copper from ore. At that time, roasting technology was crude and limited to the heap method where ore was piled up with cordwood and limestone intermixed and set aflame. With such crude processes, the smelter produced a matte of 50% copper (i.e., industrially worthless until further refined), while the lead, which was prevalent within local ores, could not be extracted. As a result, further refining was required at Heinze's refinery in Butte, Montana (www.crowsnest-highway.ca). The Spokane Falls & Northern Railway company was reluctant to transport the copper matte and offered an alternative to surrounding area mining companies willing to construct a smelter in Northport, Washington. The owners of the Le Roi Gold Mining Company of Spokane registered in the state of Washington in August 1897, and the Le Roi smelter (described in Section 2.3.2 below) was operational by February 1898 (www.crowsnest-highway.ca).

The resulting competition (i.e., lack of ore and manpower) temporarily halted smelting operations in Trail. On March 1, 1898, the Canadian Pacific Railway negotiated the purchase of the Trail smelter and associated railway rights and immediately began modernization activities. By July 1898, the facility, under the name Canadian Smelting Works, was tied into the West Kootenay power grid and by December of that year smelting operations were underway (Cominco, 2000). As the number of lead mines within the surrounding area (i.e., Canada and the western U.S.) grew, in 1901 lead furnaces were added to facility operations. The new furnaces were unsophisticated, however, and until 1902 the resulting impure bullion was transported to the American Smelting and Refinery Company's plant in Tacoma, Washington, for further processing. With the development of the Betts electrolytic process in 1902, the facility could produce pure lead, fine silver, and gold. Recognizing the value of securing a source of ore and concentrate, Canadian Smelting Works began working toward the consolidation of surrounding area mines with the smelting facility. This consolidation process culminated in 1906, and the Canadian Smelting Works became known as the Consolidated Mining and Smelting Company of Canada (www.crowsnest-highway.ca). Zinc production began in 1916. By 1925, the facility consisted of a complex of structures housing a lead plant, an electrolytic zinc plant, a foundry, a machine shop, and a copper-rod mill (www.crowsnest-highway.ca). Fertilizer plants were built at the Trail smelter in 1930, facilitating the production of both nitrogen- and phosphorus-based fertilizers (MacDonald, 1997). The facility constructed and operated a heavy water plant from 1944 to 1955 (www.crowsnest-highway.ca).

The smelter was renamed Cominco in 1966 (G3 Consulting, 2001b). In addition to lead, zinc, cadmium, silver, gold, bismuth, antimony, indium, germanium, and arsenic, the Cominco facility

also produced sulfuric acid and liquid sulfur dioxide (SO_2). Ammonia, ammonium sulfate, and phosphate fertilizers were produced at the plant until August 1994, at which time production of the phosphate-based fertilizer was terminated (MacDonald, 1997). In 2008, Teck Cominco changed their name to Teck Metals Ltd. or Teck.

Major operations at the facility include primary smelting of zinc and lead concentrates and secondary smelting for production of a variety of metal products (e.g., antimony, bismuth, cadmium, cobalt, copper, germanium, gold, indium, mercury, silver, and thallium), arsenic products, granular and crystallized ammonium sulfate fertilizers, sulfur, sulfuric acid, SO_2, and ferrous granules (i.e., granulated slag) (EPA, 2003b).

While information regarding releases at the Teck Trail facility prior to the 1970s has not been provided, known discharges and emissions from the Trail facility, historic and current, that have relevance to the UCR Site include but are not limited to:

- Discharges of granulated slag to the Columbia River
- Liquid effluent discharges to the Columbia River
- Atmospheric emissions (stack and fugitive)
- Potential discharges to the Columbia River from surface water runoff
- Accidental spills and releases to the Columbia River from Trail facility operations

These emissions are described in detail in the HHRA work plan (SRC, 2009) and the RI/FS Work Plan (EPA, 2008).

2.3.2 Le Roi/Northport Smelter

The historic Le Roi/Northport Smelter (Le Roi) was located approximately 7 RMs downstream of the U.S. – Canada border in Northport, Washington. The Le Roi Smelter property encompasses approximately 32 acres and is accessed from the Northport-Waneta Road near Highway 25. The former smelter area occupied the eastern two-thirds of the property, and a former lumber mill occupies the remaining portion. The smelter buildings, which have been removed, included a furnace building, roaster building, and ore crushing mill (Heritage, 1981).

In 1892, D.C. Corbin, owner of the Spokane Falls and Northern Railroad, built a rail line to Northport, then consisting of a lumber mill and several tents. The railroad tracks were located adjacent to the Le Roi site. In 1896, Mr. Corbin donated the property to the Le Roi Mining and Smelting Company for the construction of the Breen Copper Smelter. In 1896, the Breen Copper Smelter began refining copper and gold ores from mines in northeast Washington, as well as copper ore from B.C., for the Le Roi Mining and Smelting Company. In 1901, the Le Roi Company smelting operations reorganized with the Red Mountain smelting operations to become the Northport Smelting and Refining Company (Northport Pioneers, 1981).

The copper and gold ore was processed by heap roasting, which involves open burning of the raw ore prior to placing it in a furnace. Some of the waste was formed into slag bricks that were then used as construction materials for on-site as well as off-site buildings. The gold and copper

concentrate was shipped from the facility by rail for further refining. At the peak of operation (1908), preliminary estimates suggest that the Le Roi Smelter processed 500 tons of ore per day until operations were suspended in 1909 (Weston, 2005). In 1914, the Le Roi Smelter reopened to process lead ore from Silver Valley, Idaho, to meet government demand during World War I (Child et al., 2018). Lead smelting operations during this period produced up to approximately 30 tons per day of airborne sulfur emissions (Weston, 2004b). Slag was the main byproduct of smelting operation at the site. This material was sorted in piles at the facility or flumed to the river via underground launders (EPA, 2004d). Operations ceased permanently in 1921. The furnace, roaster, and crusher and ore buildings were removed from the site. From 1953 to 2001, the western portion of the site was used as a lumber mill; no wood treatment or chemical use was reported for this period of operation (Weston, 2004b).

Emissions from the Le Roi facility that have potential relevance to the UCR Site include:

- Discharges of slag to the Columbia River
- Drainage to the Columbia River from seepage and surface runoff of materials stored on-site
- Stack emissions
- Effluent discharge and accidental spills

From 1993 to 2004, EPA conducted preliminary assessments, site inspections, and a removal site evaluation (RSE). Removal assessment activities included sampling of residential and commercial properties in and around the Northport community, sampling of public areas, and collecting drinking water samples from the municipal well located near the former smelter site. Northport residential and commercial properties with lead concentrations in soil greater than 1,000 mg/kg (removal action level) were identified in 2003 and 2004 for a Time Critical Removal Action (TCRA). A removal action was conducted on the Le Roi property and residential areas within the town of Northport by EPA in 2004 (Weston, 2004b, 2005). Contaminated soils were consolidated at the smelter site (11-acre area), covered with a polyethylene sheet and clean soil, and vegetated.

As described in greater detail in Section 2.4, TAI conducted TCRAs in 2015, 2017, and 2018 at residential properties and a CCT tribal allotment at the UCR Site that were sampled in 2014 and 2016 using a removal action level of 700 mg/kg lead in soil. Given the lower removal action level of lead used in 2015, 2017 and 2018, in 2019 EPA conducted an RSE of properties within the town of Northport that had been sampled in 2003 and 2004 as described above and found to have lead in soil near or above 700 mg/kg.[7] The RSE identified 16 residential properties and common use areas, which are publicly owned or to which the general public has access and the right to use, within the Northport town limits that met the criteria for a TCRA. Each property has one or more DUs designated for removal activities.

At each property, the portions of the property subject to investigation and subsequently designated for removal action (i.e., the DUs) were selected based on their proximity to the

[7] https://response.epa.gov/site/site_profile.aspx?site_id=14843.

residences and the likelihood that the DUs would be used frequently by residents or visitors. The DUs include yard, garden, and play areas used by residents for property access, recreation, lawn and house maintenance, and gardening; activities conducted within these DUs result in an increased risk of exposure to the elevated levels of lead in the soil. Soil cleanup activities were scheduled to begin in August 2020 (EPA Region 10, 2020).

2.4 Investigations and Response Actions

Numerous investigations have been conducted at the UCR Site to assess and evaluate potential contamination in surface water, sediment, soil, air, and biota. Operations at the Trail facility began in 1896 and continue today. One of the changes to Trail facility operations was the cessation of granulated slag discharges to the Columbia River in mid-1995. Additionally, the KIVCET[8] (which stands for "oxygen, vortex, cyclone and electrothermic," in Russian) smelter installation in 1997 and subsequent operational improvements led to a significant reduction in stack lead emissions (Teck, 2017). Therefore, a cut-off date of 2002 was utilized for all environmental data sets evaluated for use in the HHRA (i.e., no data collected prior to 2002 were used in the HHRA).

Summaries of the findings and recommendations of EPA ESIs conducted in 2001 and 2002 are provided in the following reports:

- EPA, 2001b (2001 Sediment Investigation Trip Report, Upper Columbia River/Lake Roosevelt Expanded Site Inspection. December 2001. Prepared by Roy F. Weston Inc. for EPA Region 10, Seattle, WA)
- EPA, 2002c (Preliminary Assessments and Site Investigations Report, Lower Pend Oreille River Mines and Mills, Pend Oreille County, Washington. April 2002. Prepared by Ecology and Environment, Inc. for EPA Region 10, Seattle, WA)
- EPA, 2002d (Preliminary Assessments and Site Inspections Report, Upper Columbia River Mines and Mills, Stevens County, Washington. October 2002. Prepared by Ecology and Environment, Inc. for EPA Region 10, Seattle, WA)
- EPA, 2003b (Upper Columbia River Expanded Site Inspection Report, Northeast Washington. March 2003. Prepared by Ecology and Environment, Inc. for EPA Region 10, Seattle, WA)
- EPA, 2004e (Hecla Knob Hill Mine Site Inspection Report, Ferry County, Washington. July 2004. Prepared by Weston Solutions, Inc. for EPA Region 10, Seattle, WA)
- EPA, 2004f (South Penn Mine Site Inspection Report, Ferry County, Washington. September 2004. Prepared by Weston Solutions, Inc. for EPA, Region 10, Seattle, WA)
- EPA, 2004g (Mountain Lion Mine Site Inspection Report, Ferry County, Washington. September 2004. Prepared by Weston Solutions, Inc. for EPA, Region 10, Seattle, WA)

[8] http://www.totalmateria.com/page.aspx?ID=CheckArticle&site=ktn&NM=366.

- EPA, 2004h (Republic Mine and Mill Combined Preliminary Assessment/Site Inspection Report, Ferry County, Washington. July 2004. Prepared by Weston Solutions, Inc. for EPA, Region 10, Seattle, WA)

Additional investigations that provided the data used in the HHRA include:

- Fish tissue sampling in 2005, 2009, 2016, and 2018 (CH2MHill, 2007; Exponent, 2013a; TAI, 2013a; CH2MHill, 2016b; Windward Environmental LLC [Windward], 2017a, 2018; TAI, 2018)
- Surface water sampling in 2009 and 2010 (Exponent, 2013b)
- Public beach sediment and soil sampling in 2005, 2009, 2010, 2011, 2013 (reanalysis), and 2015 (EPA, 2005b, 2006b, 2006c, 2013a; TAI, 2010b, 2014a, 2016a)
- Outdoor air monitoring data collected from 2002 through 2009 by Teck (CH2MHill, 2015; Washington State Department of Ecology [ECY], 2017a)
- Upland and relict floodplain soil sampling in 2014 and 2015 (TAI, 2015a, 2015b, 2016a)
- Residential soil sampling (including residential beaches[9]) in 2014 and 2016 (CH2MHill, 2016a; TAI, 2016b, 2017a)
- Macroinvertebrate sampling in 2016 (Exponent et al., 2016; Windward, 2017b)
- Wild/cultural plant sampling conducted in 2018 (TAI, 2019a)

Removal actions that have been conducted and access restrictions that have been implemented at the Site include:

- Sediment removal action at Black Sand Beach in 2010 (TAI, 2011b). This voluntary action was conducted by Teck under ECY oversight. The beach sediment used in the calculation of the exposure point concentration (EPC) for this beach was collected in 2009, before the removal action; however, the 2009 data were the only sediment data available for this beach.
- Bossburg Flat Beach and Upland refined sediment and soil sampling conducted by Teck under EPA oversight. Bossburg Flat Beach and Upland Area was closed in 2012 by DOI NPS (NPS, 2012) because of elevated levels of lead found in beach sediment and upland soil samples (TAI, 2015b).
- Young American Mill site removal action in 2012 conducted by EPA Removal Program.

[9] The term “residential beach” is used in this HHRA to mean a beach on private property that was sampled as part of the 2014 or 2016 Residential Soil Study.

- Mine and mill risk reduction actions conducted at Anderson-Calhoun in 2010,[10] Sierra Zinc in 2001 and 2002,[11] Van Stone in 2017,[12] and Bonanza Mine in 2002.[13]
- TCRAs and Voluntary Removal Actions (VRAs) on 28 decision units (DUs) from the 2014 and 2016 residential soil sampling area as of June 20, 2019 (20 from the 2014 sampling, 6 from the 2016 sampling, and 2 sampled in 2018). The TCRA action level for lead in soil was 700 mg/kg. These DUs are listed in Table 2-3. The TCRAs and VRAs were conducted by Teck under EPA oversight. For DUs where a removal was conducted, the maximum measured concentration of each COPC in backfill used during that removal action was used as the soil EPC in risk calculations (Table 3-12). The COI screen and risk calculations in this HHRA include TCRA or VRA DUs at their post-removal concentrations. In addition, three DUs are part of the Soil Amendment Technology Evaluation Study. For these DUs (258, 401, and 441), the original measured concentration and bioavailability data were used in this HHRA, since the study is in progress.

Consumption evaluations conducted for biota harvested at the Site include:

- WDOH evaluated fish tissue data from the 2009 fish tissue sampling event (https://www.doh.wa.gov/Portals/1/Documents/Pubs/334-317.pdf) (WDOH, 2012)
- WDOH evaluated White Sturgeon tissue data collected from the UCR in 2016 (WDOH, 2018a)
- WDOH evaluated macroinvertebrate (crayfish and mussel) tissue data collected from the UCR in 2016 (WDOH, 2018b)
- WDOH evaluated Northern Pike tissue data collected from the UCR in 2018 (WDOH, 2019)

2.5 Basis for Human Health Concern

Smelting operations generate wastes that contain elevated levels of metals. The Teck smelter is a source of contamination to the UCR. Elevated concentrations of metals that are related to smelting operations have been measured in biotic and abiotic media in the UCR recreational areas, surrounding residential communities, and upland areas. Excess exposure to these metals may cause a range of adverse non-cancer and cancer health effects in humans.

2.6 Site-Specific Chemical Data to be Utilized in the Human Health Risk Assessment (HHRA)

For this Site-wide HHRA, the Site-specific data collected by EPA and TAI listed above were utilized as outlined below. Data collected from the UCR, residential, and non-residential areas

[10] https://response.epa.gov/site/site_profile.aspx?site_id=6194.
[11] https://www.dnr.wa.gov/Publications/ger_ofr2003-20_iaml_sierrazinc.pdf.
[12] https://apps.ecology.wa.gov/gsp/Sitepage.aspx?csid=461;
https://www.dnr.wa.gov/Publications/ger_ic100_iaml_vanstone.pdf.
[13] https://www.dnr.wa.gov/Publications/ger_ic109_iaml_bonanza.pdf.

are briefly described below and are summarized in Tables 2-4 through 2-21. A discussion of how these data were used for calculation of EPCs in the HHRA is presented in Section 3.3.

2.6.1 Surface Water

The source of the surface water data that were utilized in the HHRA is the 2009/2010 Surface Water Study conducted as part of the RI/FS for the Site (Exponent, 2013b). While surface water data were available at the time that the HHRA work plan (SRC, 2009) was developed, those data were not representative of disturbed conditions that are more relevant for assessing human exposure. Additionally, surface water data were needed to represent current conditions, provide representative spatial coverage of the UCR Site, and evaluate seasonal variability in surface water conditions throughout the UCR Site. To fill these data gaps, surface water samples were collected in 2009 and 2010 from transects spatially distributed along the UCR to support the HHRA and BERA (Figure 2-4). The study focused on spatially representative reaches of the UCR collected during time periods that represented extreme flow and water level conditions. Three discrete rounds of sampling were conducted to assess surface water concentrations of COIs under various river conditions. The first round of samples was collected between September 1 and October 20, 2009, during high pool, low flow river conditions. The second round of sampling was conducted from February 23 to April 19, 2010 during low pool, low flow river conditions. The third round of sampling occurred between April 24 and June 17, 2010 during rising pool, high flow river conditions.

Transects 1, 8, and 9 were located within UCR Reach 1; for all other UCR reaches, there was one transect per reach. Four types of samples were collected along each transect. Single-point, near surface samples (1 m below water surface) were collected at two to four locations along each transect. Single-point, near bottom samples (approximately 1 m above the river bottom) were collected along each transect at the same locations the near-surface samples were collected. Undisturbed, single-point nearshore samples were collected near the right and left riverbank along each transect. Samples were collected from the middle of the water column in water approximately 0.5 m deep. Disturbed nearshore samples were collected at each end of each transect from approximately 0.25 m below the water surface following sediment disturbance that would reflect shallow water (1 m deep) play during recreation or other nearshore human activity. Triplicate samples (a primary sample and two field replicates) were collected at each disturbed sampling location.

All surface water samples were submitted for analysis of total (unfiltered) and dissolved (filtered) Target Analyte List (TAL) metals[14] plus molybdenum, uranium, and organic carbon. Some surface water samples were also submitted for analysis of total and dissolved other metals

[14] TAL metals: aluminum, antimony, arsenic, barium, beryllium, cadmium, calcium, chromium, cobalt, copper, iron, lead, magnesium, manganese, mercury, nickel, potassium, selenium, silver, sodium, thallium, vanadium, and zinc. Arsenic was analyzed using two methods in most samples: EPA1632 (reported as “Inorganic Arsenic”), and EPA 6020 (total arsenic reported as “Arsenic”).

and metalloids[15]. The following groups of analytes were analyzed in unfiltered surface water samples:

- Organic compounds (pesticides, semi-volatile organic compounds [SVOCs], polycyclic aromatic hydrocarbons [PAHs], polychlorinated biphenyls [PCBs] as congeners, and polybrominated diphenyl ethers [PBDEs])
- Conventional parameters (hardness as calcium carbonate [$CaCO_3$] and total suspended solids [TSS], both disturbed and undisturbed samples; alkalinity as $CaCO_3$, total dissolved solids, TSS, pH, sulfate, chloride, fluoride, and silica [as dissolved silicon dioxide, SiO_2], undisturbed samples only)
- Stable isotopes of water (deuterium and oxygen-18)
- Radionuclides (uranium-238 and radium-226)
- Field measurements (water depth, temperature, pH, dissolved oxygen, conductivity, turbidity, and oxidation-reduction potential)

Table 2-4 summarizes the number of samples by reach and analyses conducted on total and dissolved disturbed surface water samples. Field replicates were not included in the sample number count. Primary samples with field replicate results (duplicate and triplicate samples) were reduced to a single sample result as described in Section 2.7 and as per the Data Management Plan (DMP) (TAI, 2019b). Lab replicates and sample results that were rejected ("R" qualified) were also not included in the sample number count.

2.6.2 Solid Media

As discussed in Section 2.2, the Site encompasses multiple communities near the UCR, the Colville Reservation and Spokane Reservation, commercial forests, and undeveloped public land. Soil and sediment data have been collected from the UCR Site during multiple sampling events. Site-specific soil data were collected by TAI in the 2014 and 2016 Residential Soil Studies (TAI, 2015a, 2017a) and by EPA in the 2014 Upland Soil Study (CH2MHill, 2016a). Soil samples were also collected from soil DUs near Bossburg Flat beach (TAI, 2016a). RFDAs were sampled as part of the Upland Soil Study (TAI, 2015a) and along with the soil samples collected near Bossburg Flat Beach are evaluated herein as "beach soil."

The sources of sediment data for public beaches were the Phase I 2005 Sediment Sampling Investigation (EPA, 2006c), the 2009 Beach Sediment Study (samples collected in 2009, 2010, and 2011) (TAI, 2014a, 2017b), and the 2015 Bossburg Flat Beach Refined Sediment and Soil Study (TAI, 2016a). Some of the archived samples collected in 2011 were re-analyzed for lead, arsenic, and *in vitro* bioavailability (IVBA) lead concentrations in the less than (<) 250 micrometer (µm) size fraction in 2013 (EPA, 2013a). Residential beach sediment data were

[15] Metalloids include: bismuth, boron, cerium, cesium, dysprosium, erbium, Europium, fluoride, gadolinium, gallium, germanium, gold, holmium, indium, lanthanum, lithium, lutetium, molybdenum, neodymium, niobium, praseodymium, rubidium, samarium, scandium, strontium, tantalum, tellurium, terbium, thorium, thulium, tin, titanium, tungsten, ytterbium, yttrium, and zirconium.

collected as part of the 2014 and 2016 Residential Soil Studies (see Sections 2.6.2.6 and 2.6.2.7). A detailed description of these sediment data can be found in Table 2-5.

Each of these studies is described separately below.

2.6.2.1 Phase I 2005 Sediment Sampling Investigation

Several different types of sediment samples were collected by EPA in 2005: baseline sediment samples, sediment core samples, and beach sediment samples (EPA, 2005b, 2006b, 2006c). The baseline sediment samples were collected to better define the current nature and extent of COIs over the length and width of the UCR between the U.S. – Canada border and Grand Coulee Dam. The sediment core samples were collected to characterize vertical variations in COI concentrations within the upper sediment column and to establish the apparent thickness of the contaminated sediment layer. The objective was to be able to assess the potential for future remobilization of COIs in known or suspected scour zones, or the rate of sediment accumulation in non-scour zones. Beach sediment samples were collected from a series of 15 "high use" beaches prioritized by managers from DOI, ECY, CCT, and STI, and distributed throughout the UCR study area. These samples were considered usable for the HHRA. However, only three of the beaches sampled in 2005 warranted further study based on a screening level risk assessment conducted by EPA Region 10 (EPA, 2006d): Black Sand Beach, Northport, and Dalles. These beaches were re-sampled in the 2009-2011 sampling event described below. The 2005 beach sediment sample results were not included in the EPC calculations for this HHRA, as they were not sieved to a grain size representative of human exposure to sediment and more recent data were available for the beaches identified as needing additional study, though they were included in the COI screen.

2.6.2.2 2009-2011 Beach Sediment Study

Three rounds of beach sediment field sampling activities were performed in September 2009, April 2010, and April and May 2011 (TAI, 2014a). The primary objective of the study was to collect additional sediment data to characterize potential risks to humans and the environment. Sediment samples were collected from 33 beach areas (Table 2-6, Figure 2-5).

The September 2009 field event targeted five beaches located within the uppermost reaches of the UCR: Black Sand Beach, Upper Columbia R.V. Park, Northport Beach, Onion Creek, and Dalles Orchard. Samples were successfully collected at the Black Sand Beach[16] and Upper Columbia R.V. Park, but conditions at the other three beaches were not conducive to sample collection. High water levels and substrate issues (presence of cobbles and bedrock) prevented collection of fine-grained beach sediment at Northport Beach, Onion Creek, and Dalles Orchard.

Surface sediment samples (0 to 15 cm) were collected from 60 discrete locations randomly distributed throughout each beach between the water's edge and the maximum beach elevation. The 60 discrete samples were randomly composited into five samples (12 randomly assigned sampling locations were composited into a single surface sediment sample). The composite surface sediment samples were used to evaluate exposure to recreational and occupational populations. Five randomly assigned subsurface sediment cores were also collected from each

[16] It should be noted that samples were collected from Black Sand Beach as part of the 2009 Beach Sediment study, which was followed by a removal action in Fall 2010 (TAI, 2011b).

beach. Core samples were collected at the following depth intervals: 0-15, 15-45, and 45-75 cm. The core samples were collected to evaluate potential effects on receptors who may come in contact with deeper sediment (outdoor workers).

In April of 2010, samples were collected at two beaches located within the uppermost reach of the UCR (Northport Beach and Dalles Orchard), and three beaches located below the uppermost reach of the UCR (Summer Island, Barnaby Island Campground, and China Beach). The sampling design for Dalles Orchard and Northport beach was modified to only collect 3 composites comprised of 12 samples because areas with fine grained sediment were very small; 5 composites of 12 samples were collected at the other 3 beaches. Five subsurface sediment cores were also collected at each beach.

In April and May 2011, sediment samples were collected from 26 beaches located throughout the UCR from the Swimming Hole to Crescent Bay. At 3 of the beaches (Kamloops Island, Crescent Bay and Swimming Hole), 5 composite samples comprised of seven samples per composite were collected; 5 composites consisting of 12 samples each were collected at the other 23 beaches. Five subsurface sediment cores were collected at each beach.

Whole sediment samples were submitted for grain size analysis. All sediment samples were sieved to <2 millimeters (mm). Sieved surface composite samples were submitted for analysis of pH, total organic carbon (TOC), total sulfides, percent moisture, and TAL metals plus uranium. One surface sediment composite from each beach was randomly selected and further sieved into four size fractions: <2 mm to 250 µm, <250 to 125 µm, <125 to 63 µm, and <63 µm. Analyses for TAL metals, elemental uranium, and IVBA of arsenic and lead were conducted on each of these finer fractions. The relationship between particle size class and metals concentration in the fractioned sample was used to estimate the metals concentrations in the <250 µm size fraction of the other 4 samples. IVBA results for arsenic and lead were used to calculate RBA-adjusted arsenic and lead concentrations.

Core sediment samples were sieved to <2 mm and submitted for analysis of pH, TOC, total sulfides, percent moisture, TAL metals plus uranium, radionuclides, PCB Aroclors, pesticides, SVOCs, and PAHs. PCB congeners, dioxins/furans, and PBDEs were analyzed in samples with TOC >1%. While core sediment samples were used in the COI screen for subsurface sediment, they were not included in EPC calculations for this HHRA as they were not sieved to a grain size representative of human exposure to sediment.

The calculated <250 µm composite surface sediment sample results were used to evaluate exposure to surface sediment COPCs during recreational or occupational activities in this HHRA. Table 2-6 summarizes the number of samples and laboratory analyses conducted by beach. Field replicates were not included in the sample number count. Primary samples with field replicate results (duplicate samples) were reduced to a single sample result as described in Section 2.7 and per the DMP (TAI, 2019b). Laboratory replicates and sample results that were rejected (“R” qualified) were not included in the sample number count as they were not utilized in this HHRA.

The <2 mm composite surface sediment sample results were used in the waterfowl uptake models to estimate tissue concentrations in waterfowl in Appendix 1. Primary samples with field

replicate results (duplicate samples) were reduced to a single sample result as described in Section 2.7 and as per the DMP (TAI, 2019b). Lab replicates and sample results that were rejected ("R" qualified) were not included in the sample number count as they were not utilized in this HHRA.

2.6.2.3 2013 Beach Sediment Re-Analysis

In the 2009-2011 Beach Sediment Study, five composite samples were collected at each beach. As described above, 4 of the samples were sieved to <2 mm and 1 was sieved into smaller size fractions. In 2013, EPA requested that the archived samples collected from Bossburg Flat, Evans Campground, Swimming Hole, Flat Creek and Lyons Island be reanalyzed to verify that results based on estimated <250 µm means were comparable to measured results for sediment sieved to <250 µm. These archived samples were sieved to <250 µm and submitted for analysis of lead, arsenic, and IVBA of lead (EPA, 2013a). If a sample was sieved to <250 µm and reanalyzed in 2013, the result reported by the lab in 2013 was used in lieu of the calculated result. For arsenic, because IVBA of arsenic was not analyzed in 2013, the calculated <250 µm IVBA result from 2011 was used to adjust the measured arsenic concentration analyzed in 2013.

2.6.2.4 2015 Bossburg Flat Beach Refined Sediment and Soil Study

Elevated lead concentrations (>1,500 milligrams [mg] per kilogram [kg]) were found in samples collected from Bossburg Flat Beach (RM 716) and Evans Campground Beach (RM 710) during the 2009 Beach Sediment Study, as described in the beach study data summary report (TAI, 2014a). Bossburg Flat Beach was closed by the NPS on January 10, 2012 in response to environmental sampling information provided to NPS by the EPA, which indicated that lead and arsenic levels in this area may be a risk to human health (NPS, 2012). This beach area remains closed today. Subsequent investigations at the adjacent Young America Mine and Mill site also identified elevated lead concentrations (TechLaw Inc., 2012a, 2012b). To investigate further, TAI collected samples from nearshore sediment and soil at the former cable ferry landings near the Young America Mine and Mill site, Bossburg Flat Beach and Evans Campground Beach as described in the Quality Assurance Project Plan (QAPP; HDR et al., 2015a, 2015b) (Tables 2-6 and 2-7; Figures 2-6 and 2-7).

Ten sediment samples were collected using incremental composite (IC) sampling methods (Interstate Technology & Regulatory Council [ITRC], 2012) within selected sediment DUs (SDUs); four nearshore SDUs at Bossburg Flat Beach, three nearshore SDUs at Evans Campground Beach, and three nearshore SDUs on the west side of the UCR near the former cable ferry landing. IC samples were collected at a depth of 0-15 cm. Triplicate IC samples were collected at four SDUs (Bossburg: SD02, SD03; Evans: SD06, SD07). Three sediment cores were collected at all SDUs except SD09 and SD10. Samples were collected from each of the cores at the following depth intervals: 0-15, 15-30, and 30-45 cm.

Two areas associated with the former cable ferry landings were designated for collection of sediment samples (F-01 and F-02). Samples could only be collected near the former cable ferry landing on the east bank of the UCR (F-01). The area around F-01 was sampled in 2012 during the removal assessment for Bossburg Flat Beach. Lead concentrations in sediment ranged from 456 to 24,463 mg/kg (Techlaw Inc., 2012b). Because of cultural sensitivities, no samples were collected from the west bank area. Three sediment cores were collected at F-01. Samples were

collected from each of the cores at the following depth intervals: 0-15, 15-30, and 30-45 cm (TAI, 2016a).

Six soil samples were collected using IC sampling methods within upland DUs (UDUs) at Bossburg Flat Beach: samples UDU-01 through UDU-05 were collected near Bossburg Flat Beach and sample UDU-06 was collected on the west side of the UCR near the former cable ferry landing (Table 2-7, Figure 2-6). IC samples were collected at a depth of 0-15 cm. Triplicate IC samples were collected at one UDU (UDU-04). Three core samples were collected at all UDUs at the following depth intervals: 0-15, 15-30, and 30-45 cm.

All samples were homogenized and sieved into two fractions: <2 mm and either <250 µm (sediment) or <149 µm (soil). Samples were submitted for analysis of TAL metals in both the <2 mm and <250 µm/<149 µm fractions and for IVBA for lead and arsenic in the <250 µm/<149 µm fraction (EPA, 2016a). Grain size, pH and percent solids were measured on the bulk sediment samples before sieving, and TOC was measured in the <2 mm fraction.

The <250 µm IC sediment sample results were used to evaluate exposure to surface sediment (0 to 15 cm) COPCs during recreational or occupational activities in the HHRA. The <250 µm sediment and <149 µm beach soil core sample results were used to evaluate exposure to subsurface sediment and beach soil (0-45 cm) COPCs during occupational activities in the HHRA. The <149 µm IC soil sample results were used to evaluate exposure to beach surface soil (0 to 15 cm) COPCs during recreational or occupational use. Table 2-6 summarizes the number of sediment samples and laboratory analyses conducted by SDU. Table 2-7 summarizes the soil data collected in this study. Field replicates were not included in the sample number count. Three IC samples (a primary sample and two field replicates) were collected at four SDUs (two at Bossburg Flat Beach and two at Evans Campground Beach) and one UDU and were utilized in the HHRA non-lead analysis as individual samples. Lab replicates and sample results that were rejected ("R" qualified) were not included in the sample number count as they were not utilized in this HHRA.

The <2 mm composite surface sediment sample results were used in the waterfowl uptake models to estimate tissue concentrations in waterfowl in Appendix 1.

2.6.2.5 2014 Upland Soil Study

Soil data for the Site were collected between September 8 and October 23, 2014 from upland areas adjacent to the UCR to fill data gaps for the BERA and the Site-wide HHRA as described in the QAPP (TAI, 2014b). These data gaps included upland areas potentially affected by aerial deposition of smelter particulates (Aerial Deposition Areas [ADAs], each approximately 25 acres in size), historical fluvial deposition of sediment onto relict floodplains (evaluated as "beach soil" herein), re-deposition of windblown sediment (Windblown Sediment Deposition Areas), and parameters needed to evaluate RBA of metals. ADAs and RFDAs in relict floodplains were targeted for both HHRA and ecological risk assessment (ERA), and soils were sieved to <2 mm and <149 µm. Windblown Sediment Deposition Areas were targeted only for ERA (EPA, 2012b) and were not sieved to a grain size representative of human exposure to soils. As such, soil data from the Windblown Sediment Deposition Areas were not included in the HHRA and are therefore not included in the remainder of this summary.

Three ADA sampling areas were delineated, as shown in Figure 2-8. *Primary* ADAs were in the northernmost 100 mi^2 of the Site, extending south from the U.S. – Canada border. Within that area, *high-density* ADAs were located within a 23-mi^2 corridor along the UCR immediately downstream of the U.S. – Canada border. The ADA high-density area was designated for more extensive sampling because of the perceived likelihood of higher historical deposition rates in that area. *Reserve* ADAs, collected to mitigate sample collection challenges in specific areas (e.g., impassable roads, flooding, rocky outcrops, lack of landowner permission, steep terrain), were collected from a 16-mi^2 area east of the ADA primary area. ADA samples were used in the HHRA.

Soil samples were collected using IC sampling methods (ITRC, 2012) within specifically selected ADAs. Details regarding the selection of ADAs within these sampling areas are found in the QAPP (TAI, 2014b); samples were not collected within 50 m of roads or railways, or within no-sample buffer zones established for active and abandoned mine sites.[17] As shown in Table 2-8, 142 ADAs were sampled. Samples were collected from the top 7.5 cm of soil at 30 increment locations randomly located within each ADA, and at 90 increment locations within the ADA if the ADA was sampled in triplicate.[18] Once all increments were collected within an ADA, they were composited into one sample for laboratory analysis. Soil samples for HHRA from ADAs were sieved using a No. 100 sieve, which has a mesh size of 149 µm. Three IC samples (a primary sample and two field replicates) were collected at 16 ADAs and were utilized in the HHRA non-lead analysis as individual samples. EPA field splits were collected at 18 ADAs but were not utilized in the HHRA. Lab replicates and sample results that were rejected ("R" qualified) were also not utilized in the HHRA.

Composite soil samples were analyzed for TAL metals,[19] molybdenum, and IVBA. Laboratory analysis was done on the <149 µm and on the <2 mm fractions of soil. IVBA analyses were performed on 20% of soils from ADAs that had lead concentrations >100 mg/kg. Data are summarized in the final soil study data summary report (DSR; TAI, 2015a). The <149 µm ADA sample results were used to evaluate potential future residential exposure to surface soil. Only IVBA results for arsenic and lead were used in the HHRA because reliable models to convert IVBA to RBA are not available for other metals.

Relict floodplains are areas that may have been flooded under past UCR flow conditions but are not expected to be inundated under current pool level management controls (TAI, 2015a). As such, exposure to surface soil ("beach soil") samples collected from these areas was evaluated as exposure to soil. The relict floodplain is the delineated area between high-pool seasonally inundated lands and the maximum pre-1973 strandline. The five largest relict floodplain areas (ranging from 0.13 mi^2 to 0.42 mi^2) are located near Northport, Washington and were targeted for sampling in 2014. Because of lack of access, no samples were collected from one of the five

[17] A 500-m no-sample buffer zone was established for mine sites within ADAs that were sampled as part of the START-2 assessment (START-2, 2002). A 100-m no-sample buffer zone was established around other known mine sites in the vicinity of the Site, including those identified as "producer," "past producer," "occurrence," "prospect," or "unknown."

[18] At ADA-101, only 15 increments were collected due to steep terrain.

[19] TAL metals include aluminum, antimony, arsenic, barium, beryllium, cadmium, calcium, chromium, cobalt, copper, iron, lead, magnesium, manganese, mercury, nickel, potassium, selenium, silver, sodium, thallium, vanadium, and zinc (EPA, 2015a) (see http://www.epa.gov/superfund/programs/clp/ismtarget.htm).

relict floodplains (RFE), and not all targeted samples were collected from RFB, RFC, and RFD. Figure 2-9 shows the location of the relict floodplains sampled in 2014.

A total of 29 RFDAs were targeted for sampling, with 3 to 9 RFDAs selected per relict floodplain. Sixteen RFDAs were sampled using IC sampling. Samples were collected from the top 7.5 cm of sediment at 30 increment locations randomly located within each RFDA, and at 90 increment locations within the RFDA if the RFDA was sampled in triplicate. Three IC samples (a primary sample and two field replicates) were collected at four RFDAs (one from each of the relict floodplains sampled) and were utilized in the HHRA non-lead analysis as individual samples. Prior to sieving, an aliquot was taken from each IC sample for analysis of grain size and pH. The remaining sample was sieved to <2 mm and <149 µm and submitted for analysis of total solids and TAL metals plus molybdenum. Three of the samples with lead concentrations >100 mg/kg were selected for IVBA analysis. Table 2-9 summarizes the RFDA soil samples collected in 2014 and used in the HHRA. The <149 µm composite RFDA surface soil sample results were used to evaluate exposure to beach soil COPCs during recreational activities. The <2 mm composite surface soil sample results were used in the waterfowl uptake models to estimate tissue concentrations in waterfowl in Appendix 1.

2.6.2.6 2014 Residential Soil Study

Rural residential properties within the northernmost reaches of the Site (north of the town of Northport to the U.S. – Canada border) were sampled by EPA in 2014 to collect surface soil and beach sediment data to support the HHRA as described in the QAPP (SRC, 2014a). Landowners in the area were contacted and provided access agreements to allow EPA to perform sampling. EPA conducted field reconnaissance of each residential property with a signed access agreement during the weeks of April 28 and May 12, 2014, to derive property-specific sampling designs. Property owners were interviewed, and exposure areas were defined and mapped according to current and potential future land use, physical property boundaries, and the predicted depth of exposure to potentially contaminated soils. Residential parcels in the soil sampling area were quite large (mean size is 16 acres; range of 0.06 to 158 acres); as such, sampling was focused on locations where there was a high potential for exposure to soil by residents, especially children. Seventy-four properties were sampled between August 18 and October 3, 2014 (Figure 2-10; CH2MHill, 2016a).

The number and location of DUs on each residential property ranged from 1 to 15 (mean = 3) and were based on property size and the likelihood of soil exposure as determined through Site visits, interviews with landowners, and review of aerial photography (CH2MHill, 2016a). Distinct play areas, immediate areas around houses, driplines, gardens, beaches, agricultural areas, and animal pens/riding areas were delineated as separate DUs. Beach IC sample locations were delineated in the field based on water elevation at the time of sampling and interviews with property owners regarding their use of the beach; 15 beach DUs were sampled in 2014. Other frequently used areas were assigned to an “other – not specified” DU category (including CCT tribal allotments which are evaluated in Appendix 1). Areas near paved roadways and railways were avoided, with a 50-foot buffer in both directions from the centerline, and other than driplines, DUs were located to avoid the influence of painted surfaces. The boundaries for each DU were delineated based on land use and geographic positioning system (GPS) data collected during the property visit.

Two types of solid media samples were collected from residential DUs: IC surface samples and discrete (core) samples. IC samples were collected according to methods described above for the Upland Soil Study and outlined in ITRC (2012), and sample depth bgs was based on DU type. In general, IC samples were taken at a depth of 0-2.5 cm bgs starting below the organic litter or sod (EPA, 2003c). Exceptions to this rule included gardens (sampled to tilled depth, which was typically 0-30 cm bgs), disturbed (animal activity) areas (sampled 0-8 cm bgs), beaches (sampled 0-15 cm bgs), and CCT tribal allotments (sampled 0-8 cm bgs). For DUs >1,000 square feet, three IC samples (a primary sample and two field replicates) consisting of 30 increments each were collected. DUs <1,000 square feet had only one IC sample (of 30 increments) collected. For duplicate types of DUs on a property, only one triplicate IC sample was collected (e.g., if there were two gardens on a property, one had three IC samples collected, and one had one IC sample [30 increments] collected).

Increments were located using systematic random sampling with a rectangular grid overlaying the DU and a random starting point. Planned increments located in compacted gravel, other non-soil materials, or obstructions were moved as described in the DSR (CH2MHill, 2016a). Beach IC sample locations were delineated based on water elevation and interviews with property owners as described above. Dripline sample locations were delineated in the field based on field observations.[20] Once all increments were collected within a DU, they were composited into one sample (or three samples, if the DU was sampled in triplicate) for laboratory analysis. Soil samples were sieved using a No. 100 sieve with a mesh size of 149 µm for all DUs except beaches; beach sediment samples were sieved to a mesh size of <250 µm.

Discrete (core) samples were collected from approximately 1 DU per property from a depth of 2.5-15 cm bgs; in most cases, 5 discrete samples were collected in the DU closest to the house (the location of the 5 samples was randomly selected). Discrete solid media samples were sieved to the same mesh size as IC samples (either <250 µm for beach sediment or <149 µm for surface soils). In all, 235 DUs were sampled; from those DUs, 541 IC samples and 402 discrete samples were collected (Table 2-10). The IC surface soil sample results from all DU types except driplines, residential beaches, and CCT tribal allotments were used to evaluate residential exposure to surface soil COPCs. CCT tribal allotment IC surface soil results were used to evaluate CHIRU exposure to surface soil COPCs in Appendix 1. Table 2-11 includes a summary of the beach sediment samples collected in 2014 that were used in the HHRA. The <250 µm composite surface sediment sample results were used to evaluate exposure to surface sediment COPCs during recreational and CCT tribal activities (one beach DU was located on a CCT tribal allotment; see Appendix 1) and to estimate uptake by waterfowl in Appendix 1. Discrete sample results were not utilized in this HHRA because these samples were collected to provide information on the vertical nature and extent of contamination and not to evaluate human exposure.

Three IC samples (a primary sample and two field replicates) were collected at 153 DUs (including all 15 beach DUs) and were utilized in the HHRA non-lead analysis as individual samples. EPA field splits were collected as well but were not utilized in the HHRA. Lab

[20] Dripline samples were not utilized in the HHRA.

replicates and sample results that were rejected ("R" qualified) were also not utilized in the HHRA.

Samples were analyzed for total solids and TAL metals (except mercury). IVBA of arsenic and lead analyses were performed on a subset of IC samples in accordance with the QAPP (SRC, 2014a and 2014b):

1. DUs with a maximum lead concentration greater than or equal to (≥) 100 parts per million (ppm) or a maximum arsenic concentration ≥20 ppm were eligible for IVBA analysis. If more than 1 DU of the same type on a given property were potentially eligible, the DU with the maximum lead concentration was selected as the eligible DU.
2. One IC sample was selected for IVBA analysis from each of the eligible DUs. For DUs with more than one IC sample, the soil sample was randomly selected.
3. In addition, all IC samples with relative percent differences for lead or arsenic concentration >30% were selected for IVBA (SRC, 2014b).

This process selected 122 IC samples from 114 DUs for IVBA analysis (36 DUs had field triplicate IC samples collected and analyzed for IVBA of arsenic and lead). Laboratory analyses and Quality Assurance/Quality Control (QA/QC) procedures, as well as analytical results, are described in detail in the DSR (CH2MHill, 2016a). Each DU had either one or three composite soil samples collected from it.

Twenty DUs sampled in 2014 had lead concentrations that triggered TCRAs or VRAs; 18 DUs were remediated as TCRAs in 2015, and 2 DUs were remediated as VRAs in 2017-2018 (see Appendices 6 and 7 for more information). These DUs, shown in Table 2-3, were evaluated in this HHRA using the maximum COPC concentrations in backfill as of data received on June 20, 2019.

2.6.2.7 2016 Residential Soil Study

Based on results of the 2014 Residential Soil Study, EPA directed TAI to collect additional solid media samples from rural residential properties within and downstream of the 2014 study boundary with the objective of supporting the HHRA (EPA, 2015b, 2015c; TAI, 2016b).[21] As in 2014, properties sampled were identified through voluntary participation. Field reconnaissance was conducted from April 25 through May 19, 2016, to gather information on property use and potential exposure areas, as well as features to be excluded from sampling (e.g., areas that have been excavated or filled, trash burning areas, driveways). Soil sampling took place on 114 properties between August 2 and September 25, 2016 (Figure 2-11).

There were 452 DUs identified through the procedures described in the QAPP (TAI, 2016b). Most properties had one "house" DU that encompassed up to 1 acre immediately surrounding a house or cabin present on the property; other DUs were delineated for driplines, play areas, gardens, agricultural areas, animal/livestock areas, beach areas, and "other" areas (including CCT tribal allotments). Seven residential beach DUs were sampled in 2016. Eight properties (19 DUs) sampled were undeveloped CCT tribal allotments. As with the 2014 residential soil

[21] The 2016 Residential Soil Study did not include the "Northport Exclusion Area," described in Section 2.3.2.

sampling, care was taken to avoid areas potentially influenced by lead paint (other than driplines), roads, driveways, and other areas that might interfere with sampling and data interpretation (TAI, 2017a). One DU (172-O1) may have been used historically as a small ore mill or ore stockpiling site (Roland, 2019). Given the likelihood that lead and other metals found in the surface soil at this DU may be related to previous use and not aerial deposition, this DU was excluded from analyses in the HHRA.

Two types of samples were collected: IC samples from at least one DU per property, and discrete (core) samples from a subset of DUs. IC samples were collected from each DU as described for the 2014 residential soil sampling and in TAI (2017a). Increments for each IC sample were collected from 30 pre-determined locations within the DU; at DUs designated for the collection of three IC samples (a primary sample and two field replicates), increments were taken from 90 pre-determined locations within the DU. Increments were collected from the surface (after removal of any surface debris) to the depth assigned to the DU type (0-2.5 cm bgs for most DU types; 0-30 cm bgs for gardens, 0-15 cm bgs for beaches). After all increments were collected for a 30-increment IC sample, they were composited and sieved using a No. 100 sieve with a mesh size of 149 µm (for non-beach soils) or a No. 60 sieve with a mesh size of 250 µm (for beach sediments).

A total of 740 IC samples were collected from the 452 DUs; this includes three IC samples (a primary sample and two field replicates) collected at 144 of the DUs (including two residential beach DUs). Three IC samples (a primary sample and two field replicates) were utilized in the HHRA non-lead analysis as individual samples. EPA field splits were collected as well but were not utilized in the HHRA. Discrete (core) samples were collected from 29 randomly selected DUs having an IC sampling depth of 0-2.5 cm. In each DU, core samples were collected from 0-2.5 and 2.5-15 cm depth intervals at five locations. After collection, core samples were sieved to the same mesh sizes used for IC samples (No. 60 or No. 100). The IC surface soil sample or beach surface sediment results from all DU types except driplines were used to evaluate residential exposure to surface soil or residential beach surface sediment COPCs. IC dripline and discrete samples were not utilized in the HHRA. Sample counts are listed in Table 2-12. The <250 µm composite beach surface sediment samples were also used to estimate uptake by waterfowl in Appendix 1. QA/QC samples are described in detail in TAI (2016b, 2017a) and are shown in Table 2-13. Lab replicates and sample results that were rejected ("R" qualified) were not utilized in the HHRA.

Sample processing at the laboratory is described in detail in TAI (2017a). Samples were analyzed for percent solids and TAL metals (except mercury). At 40 DUs having non-dripline IC samples with a lead or arsenic concentration ≥100 or 20 mg/kg, respectively, samples were submitted for IVBA of arsenic and lead analysis. Laboratory analyses, QA/QC procedures, and analytical results are detailed in the DSR (TAI, 2017a).

Six DUs sampled in 2016 were subject to removal actions in 2017-2018, along with two additional DUs (203-O3 and 203-O4) removed in 2018 (see Appendices 6 and 7). These DUs, shown in Table 2-3, are evaluated in this HHRA using the maximum COPC concentrations in backfill based on data received on June 20, 2019.

2.6.3 Outdoor Air

Outdoor air data were considered a moderate data need in the HHRA work plan (SRC, 2009). A review of current and historical air data from air monitoring is found in CH2MHill (2015). Starting in January 1994, Teck operated an outdoor air monitoring station at Sheep Creek near Northport (Figure 2-12). Every 6 days, a composite air sample was collected and analyzed for arsenic, cadmium, lead, and zinc. Particulate matter (PM_{10}) levels were measured as well. These data were collected through 2009 (for more information see ECY, 2017a and ECY, 2017b). Because the Trail smelter implemented a process change in the lead smelting operation in 1997 (installation of a KIVCET smelter and other operational improvements) that lowered emissions (Teck, 2017), data collected from 2002 through 2009 at the Sheep Creek monitoring station were used in this HHRA (Table 2-14). EPA evaluated air data measured at the Sheep Creek monitoring station near Northport and determined that concentrations in air today are lower than those measured at the Sheep Creek station from 1994 to 2009 because of the operational improvements at the Teck facility (Figure 2-13; EPA, 2018b; Appendix 8). Based on aerial emissions reported to the Canadian National Pollutant Release Inventory, COPC concentrations in air emissions have been decreasing over time. For example, arsenic emissions ranged from 1,089 to 13,608 kg per year from 1994 to 2001, and from 662 to 2,572 kg per year from 2002 to 2009 (Canadian National Pollutant Release Inventory Data Search)[22]. Additionally, ECY estimated air concentrations south of the CA border using data from a series of Teck-managed monitoring stations in the Columba River valley from Trail to Columbia Gardens, CA. Based on the most recent available data (2014), estimated PM10 COPC concentrations at Northport are similar to (cadmium) or lower than (arsenic, lead) the EPCs used in this risk assessment (ECY, 2017a).

The 2002-2009 Sheep Creek data were downloaded from the TAI database (http://teck-ucr.exponent.com) and used in this HHRA to evaluate all receptor populations (including the CHIRU and STI populations in Appendices 1 and 2). No field or lab replicates were present in the database; thus, no data reduction was performed on this data set.

2.6.4 Aquatic Biota and Terrestrial Plants

Biota evaluated in the HHRA with Site-specific data include fish, macroinvertebrates (mussels and crayfish), and aquatic and terrestrial plants. Each is discussed separately below. While macroinvertebrate and plant data are summarized here, they were evaluated in Appendix 1 and not discussed in the remainder of this document.

2.6.4.1 Fish

Fish tissue data were collected from the Site in several sampling events. The four sources of data included in this HHRA are the Phase I 2005 Fish Tissue Study, the 2009 Fish Tissue Study, the 2016 Hatchery White Sturgeon Study, and the 2018 Northern Pike Study, each of which is described separately below.[23]

[22] https://pollution-waste.canada.ca/national-release-inventory/archives/index.cfm?do=facility_history&lang=En&opt_npri_id=0000003802&opt_report_year=2017.
[23] Common names for fish in this document were capitalized following the American Fisheries Society guidelines, Section 2.15; https://fisheries.org/docs/pub_stylefl.pdf and https://fisheries.org/docs/pub_style10.pdf.

Phase I 2005 Fish Tissue Study

EPA collected fish from the UCR in September and October 2005 as part of Phase I of the CERCLA RI/FS for the Site (CH2MHill, 2005a, 2005b, 2006, 2007). This study was designed to gather data to support HHRA and ERA at the Site, and to provide information related to the issuance of an updated fish advisory in Lake Roosevelt (CH2MHill, 2005a). Samples were collected from upper, middle, and lower reaches of the UCR, defined based on physical characteristics of the river and historical contaminant distribution. Two distinct Fish Sample Collection Areas (FSCAs) were located in each reach; five of these were co-located with sediment sampling areas (Figure 2-14). Five fish species were targeted for sampling: Walleye (*Sander vitreus*), Rainbow Trout (*Oncorhynchus mykiss*), Lake Whitefish (*Coregonus clupeaformis*), Largescale Sucker (*Catostomus macrocheilus*), and Burbot (*Lota lota*). In the northernmost FSCA, Mountain Whitefish (*Prosopium williamsoni*) was collected instead of Lake Whitefish because of habitat limitations.

Five composite samples of each species (each consisting of 3-5 individual fish of similar size) were targeted for collection and analysis from each FSCA. Whole-body samples were analyzed for all species. At one FSCA per reach (FSCAs 1, 3, and 6), skin-on fillets and remainder were analyzed separately for walleye and Rainbow Trout. To avoid overestimating metals in Largescale Sucker (a bottom feeder), gut (esophagus, stomach, and intestines) and gut contents were removed and analyzed separately from each individual fish in 2 randomly selected composite samples from FSCA 1, and from 1 randomly selected composite sample in FSCAs 3 and 6. Table 2-15 summarizes the fish tissue composite samples submitted for chemical analysis by species and FSCA. Fish samples were analyzed for PCB Aroclors, all 209 PCB congeners, dioxins and furans (tetra- through octa-chlorinated dibenzo-p-dioxins and chlorinated dibenzofurans), TAL metals, arsenic speciation, percent lipids, and percent moisture (Table 2-16). Data were reported on a wet-weight basis.

For the purposes of HHRA, fillets from fish longer than 30 cm were considered relevant for the evaluation of consumption by humans. Therefore, the skin-on fillet data for Rainbow Trout and Walleye collected in this sampling effort were used in the HHRA. Data were reduced using methods detailed in Section 2.7.2.

2009 Fish Tissue Study

To supplement the 2005 fish tissue data in support of both the HHRA and BERA for the Site, TAI collected several species and size classes of fish in September and October 2009, from the six FSCAs in the UCR sampled in 2005 (TAI, 2009a, 2013a). The FSCAs, shown in Figure 2-14, are within 6 approximate UCR river reaches defined as follows:

- Reach 1 (U.S. – Canada border at RM 745 to RM 730)
- Reach 2 (RM 730 to RM 712)
- Reach 3 (RM 712 to RM 700)
- Reach 4 (RM 700 to RM 640)
- Reach 5 (RM 640 to RM 617)
- Reach 6 (RM 617 to Grand Coulee Dam near RM 596)

Size classes of fish sampled included less than or equal to (≤) 15 cm, >15 to ≤30 cm, and >30 cm, with the smaller fish intended to fill a data gap for the BERA. Six single-species composite samples (each with a minimum of 5 fish >30 cm in length) were targeted for the following species: Walleye, Burbot, Smallmouth Bass (*Micropterus dolomieu*), Rainbow Trout, kokanee (*Oncorhynchus nerka*), Lake Whitefish, and Largescale Sucker. Mountain Whitefish and Longnose Sucker (*Catostomus catostomus*) were collected as surrogates for Lake Whitefish and Largescale Sucker, respectively, when necessary. Table 2-17 contains a summary of the >30 cm fish collected during the 2009 study.

Analyses of larger fish (>30 cm) were conducted on skin-on fillet and remainder (head, viscera, fins, skeleton, and muscle excluding fillets) samples. Gut contents of Largescale and Longnose Sucker (benthivorous fish) were removed from the "remainder" samples prior to analysis. Fillets of fish >30 cm in length were analyzed for TAL metals plus total mercury[24], uranium and metalloids; dioxins/furans; PCB congeners; PBDEs; PAHs; pesticides; SVOCs; percent moisture; and percent lipids. A subset of fillet samples from large fish was analyzed for inorganic arsenic (n = 98). Subsamples of individual Smallmouth Bass fillets were also analyzed for total mercury prior to compositing.

As stated above, fillets of fish longer than 30 cm are considered most relevant for the evaluation of consumption by humans. Therefore, the skin-on fillet data for Burbot, kokanee, Lake Whitefish, Mountain Whitefish, Largescale Sucker, Longnose Sucker, Rainbow Trout, Smallmouth Bass, and Walleye were used in the HHRA. Data were reduced using methods detailed in TAI (2019b) as described in Section 2.7.2.

2016 Hatchery White Sturgeon Study

In 2016, hatchery White Sturgeon (*Acipenser transmontanus*) were collected from the UCR as part of a population stock assessment and targeted removal effort by the Lake Roosevelt Fishery Co-Managers and the Upper Columbia White Sturgeon Recovery Initiative (SRC, 2016). White Sturgeon are a traditional tribal food source and common sport fish in the UCR but were not sampled in the 2009 fish tissue study because they were not part of the legal UCR fishery at that time. The 2016 White Sturgeon data were intended for use in the HHRA and to support WDOH in their review of the potential need for a UCR White Sturgeon fish advisory.

Fish were collected in August and September of 2016 from UCR Reaches 2 through 4 (RM 723 to RM 684), from the U.S. – Canada border downstream to Inchelium/Gifford (Figure 2-15). White Sturgeon were divided into three size classes, with 24 fish per size class: 50-97 cm,

[24] Mercury was analyzed using EPA Method 7471B/EPA Method 1631e, which only measures total mercury. In fish, methylmercury and inorganic mercury are absorbed by the gut; methylmercury is then transferred to blood and distributed to other body tissues (Ribeiro et al., 1999), ultimately accumulating in skeletal muscle tissue. As a result, most mercury in the muscle of fish is methylmercury (Wiener et al., 2003). Chumchal et al. (2011) collected invertebrates and vertebrates from a freshwater lake, including six fish species, and analyzed for total mercury and methylmercury. The percentage of mercury in muscle tissue present as methylmercury ranged from 84 to 100%. As part of the Environmental Monitoring and Assessment Program (EMAP) sampling program, 10 species were collected from 14 estuaries in southern Florida (Kannan et al., 1998). Total mercury and methylmercury were measured in fish muscle tissue; on average, methylmercury contributed 83% of the total mercury tissue concentration (range 62-100%). Because the majority of mercury present in fish fillets is methylmercury, risk calculations were done assuming mercury was present in fish tissue as methylmercury even though only total mercury was analyzed in UCR fish tissue.

98-137 cm, and 138-160 cm fork length. Within each size class, the 24 fish were filleted with skin removed and sorted into 3 composite samples using a stratified random approach described in Appendix B of Windward (2017a). The nine skinless fillet composite samples were analyzed for TAL metals/metalloids (including mercury), inorganic arsenic, dioxins/furans, total PCBs, PCB congeners, total PBDEs, and PBDE 47, PBDE 99, PBDE 153, and PBDE 209.

Because fillet data from fish >30 cm are considered most relevant for the evaluation of consumption by humans, all nine White Sturgeon composite samples were used in the HHRA. Data were reduced as described in Section 2.7.2, including conversion of dry weight (dw) fish tissue data to wet weight (ww) concentrations.

2018 Northern Pike Study

Northern Pike (*Esox lucius*) were collected from the UCR in July, 2018 (TAI, 2018). Northern Pike were not sampled in 2009 because they had not yet expanded their range into the UCR. These fish were not detected in the UCR until 2011. Since then, Northern Pike have become the top predator in Lake Roosevelt and have rapidly increased in abundance, negatively impacting both native and hatchery prey fish (Lee and King, 2015, 2016; Lake Roosevelt Fisheries Co-managers 2018). Although Smallmouth Bass and Walleye were collected in 2009, they are not appropriate surrogates for Northern Pike as Northern Pike are voracious predators with piscivory beginning in earlier life stages. Smallmouth Bass and Walleye feed on aquatic invertebrates for a much longer period of time (Walrath, 2013). As a result, Northern Pike have a much faster growth trajectory and larger terminal size, which may result in differences in COPC bioaccumulation. Because Northern Pike are a non-native invasive species, the CCT offer a $10 bounty per head incentive for anglers to remove them from the UCR. This may increase consumption of Northern Pike by anglers. The 2018 Northern Pike data were intended for use in the HHRA and to support WDOH in their review of the potential need for a UCR Northern Pike fish advisory.

Northern Pike tissue samples were collected by the Lake Roosevelt Fisheries Co-Managers July 24 through 26, 2018 as part of the Northern Pike suppression effort. Fish were captured in the area between Gifford and Northport, Washington using gill nets (Figure 2-16; CH2MHill, 2018a). Northern Pike were separated into two size classes: 30 fish 300 to 449 mm total length, and 30 fish >450 mm total length. Within each size class, fish were filleted with skin removed and sorted into six composites per size class. A stratified random compositing approach based on size class was used: individual fillets from each size class were randomly assigned to one of six composite samples for that size bin. The twelve skinless fillet composite samples were submitted for analysis of TAL metals, inorganic arsenic, mercury, percent moisture, and percent lipids.

Because fillet data from fish >30 cm are considered most relevant for the evaluation of consumption by humans, all twelve Northern Pike composite samples were used in the HHRA. Data were reduced as described in Section 2.7.2, including conversion of dw fish tissue data to ww concentrations.

2.6.4.2 Macroinvertebrates (Mussels and crayfish)

TAI collected macroinvertebrates during two sampling events in the spring and fall of 2016 to support both the BERA and HHRA. Mussels and crayfish were collected from sampling areas within six river reaches of the Site and two reference areas (the Sanpoil River and Buffalo Lake;

Figure 2-17)[25] (Windward, 2017b). In each sampling area, six composite samples from different locations within each reach and from each reference area were targeted for both mussels and crayfish, with a target of at least five organisms per composite (Exponent et al., 2016). The spring sampling event was timed to coincide with the annual reservoir drawdown period, when water levels were expected to be lowest and mussels most accessible. However, water levels were higher than expected and sufficient numbers of crayfish and mussels were not obtained. Clams were collected from one river reach in spring 2016 where the target sample numbers for mussels had not been met and several clam beds were located. A second sampling event was held in early fall to complete the sampling effort. Target sample numbers were met for crayfish and mussels from all sampling areas, except only one crayfish was collected from Sampling Area 1 and no mussels were collected from Buffalo Lake. Mussel samples were collected by divers in Site Sampling Areas 1, 3, 4, 5, and 6 in order to meet target sample numbers.

Six composite mussel samples were collected from each Site reach, and from the Sanpoil River reference area. No mussels or clams were found in the Buffalo Lake reference area. Mussels were identified to the lowest practical level; species collected were *Anodonta sp*. and the western pearlshell mussel (*Margaritifera falcata*). Western pearlshell mussels were only found and collected from the Sanpoil River reference location. One clam sample (*Corbicula sp.*) was collected from Reach 6 in the spring sampling event; this sample was not utilized in this HHRA. For mussels, the soft tissue of the mussel (including any liquid inside the shell) was collected and submitted for analysis. The QAPP (Exponent et al., 2016) stated that six composite samples would be collected from different locations within each sampling area; however, samplers were not able to locate six different mussel beds in all sampling areas. Mussel samples collected from the same mussel bed were considered independent composite samples when calculating the EPCs for mussel tissue. Mussel beds sampled and composites are shown in Table 2-18. Table 2-19 summarizes the mussel samples collected by species and by reach.

Two crayfish species were collected: the native signal crayfish (*Pacifastacus leniusculus*) and the non-native northern/virile crayfish (*Orconectes virilis*). The whole body minus the carapace and stomach[26] and the stomach/carapace were submitted separately for analysis for crayfish collected from Areas 2, 5, 6, Buffalo Lake, and the Sanpoil River. Whole body crayfish collected in Areas 3 and 4 were submitted for analysis. Six composite crayfish samples were collected from Reaches 2 to 6, and from the Sanpoil River and Buffalo Lake. Only one crayfish was collected by hand from Reach 1. This specimen was immobile, appeared to have a hole in its carapace, and was not retained for analysis. Most of the crayfish composites contained crayfish that were collected from multiple locations; no locations were over-represented within a composite sample. As per the QAPP, the crayfish composites were considered representative of different locations within each sampling area. Table 2-19 summarizes the crayfish samples collected by species and by reach.

Samples collected from Areas 2, 5, 6, Buffalo Lake and the Sanpoil River were analyzed for TAL metals, methylmercury, and total and inorganic arsenic (by EPA methods 6020A and 1632A; analytical results from these two methods are reported separately), PCB congeners, and

[25] The lower, teardrop-shaped area of the Sanpoil River Reference Sampling Area on Figure 2-16 was excluded from sampling due to it being within the UCR Site area.
[26] Whole body minus the carapace and stomach leaves the head, claws, abdomen, tail, and shell around the claws and tail.

dioxins/furans. Samples collected from Areas 1, 3 and 4 were analyzed for TAL metals and percent solids. Table 2-20 summarizes the macroinvertebrate composite samples submitted for chemical analysis by reach.

Data from all areas of the Site are being utilized in the BERA. Sampling Reaches 5 and 6 were identified as potential local source areas for mussels and crayfish that may be consumed by people based on data provided in the *UCR Tribal Consumption and Resource Use Survey Data Summary Report* (CCT Tribal Survey) (Westat Inc., 2012) and anecdotal information provided to EPA by a representative of the Spokane Tribe (Knudson, 2015). Reach 2 was also targeted for evaluation in the HHRA because sediment TOC concentrations were >1%. Because similar tissue was collected for mussel samples from all reaches, mussel data from all six reaches were evaluated in Appendix 1. Crayfish data (whole body minus the carapace and stomach samples) from Reaches 2, 5, and 6 were evaluated in Appendix 1. Limited information is available on locations where people may collect shellfish for consumption, and the *Recreational Consumption and Resource Use Survey for the UCR Site HHRA and RI/FS* (RecUse Survey) (Industrial Economics, Inc. [IEc], 2012) did not include questions on collection of shellfish. Therefore, the shellfish data were evaluated on a Site-overall basis (data from Reaches 2, 5 and 6 for crayfish, data from Reaches 1 through 6 for mussels).

For mussels, there were 36 composite samples available for on-Site reaches, and 6 composite samples available for the reference areas. There were 18 crayfish samples from on-Site reaches, and 12 composite samples from the reference areas.

Field replicates were not collected during the two sampling events. Samples identified in the data file as field splits were determined to be laboratory replicates based on the sample handling description in the QAPP. For laboratory replicates, only the parent sample was utilized in the EPC calculations as described in the revised DMP (TAI, 2019b). Shellfish tissue data needed to be expressed in terms of ww for evaluation in Appendix 1 and were reported on a ww basis by the laboratory.

2.6.4.3 Wild/Cultural Terrestrial and Aquatic Plants

Collection of terrestrial and aquatic plants at the Site took place in 2018. Members of the CCT consume and otherwise use (e.g., for weaving) terrestrial and aquatic plants harvested from the vicinity of the Site. Plants were collected from CCT tribal allotments in the study area to characterize the concentrations of metals in the tissues of wild upland plants used for food or cultural purposes. Data collection efforts focused on obtaining information used in the exposure assessments for receptors who ingest, mouth, or otherwise utilize plants from the study area. Plant species and parts collected during the plant study are summarized in Table 2-21.

TAI collected plant tissue and co-located soil samples during three sampling events in 2018: April 24 through May 2 (Spring), June 18 through 20 (June), and August 20 through 28 (August) (TAI, 2019a). Plant sample locations are shown in Figure 2-18. Plant tissues targeted for collection were determined based on their expected stage of growth in each season, typical CCT collection times (Fraser, 2017), and field observations during reconnaissance events. Plants were collected from three CCT tribal allotments sampled in the 2014 Residential Soil Study (CH2MHill, 2016a) that had bioavailability-adjusted soil lead concentrations >700 mg/kg, and from CCT tribal allotments with lower soil lead concentrations measured during the 2014 and

2016 residential soil studies. Willows, a plant of cultural significance, were not found on the CCT tribal allotments surveyed during the August 2017 field reconnaissance (AECOM, 2017), so willows were collected from additional sampling areas located along the UCR.

A total of six plant tissue and co-located soil samples were targeted for collection from the anticipated high and low lead sample areas for each plant species and tissue type. Plant tissue samples were collected from an individual plant or, if necessary to meet sample mass requirements for the analytical laboratory, from two or more adjacent individual plants forming a composite sample of sufficient sample mass. A co-located surface soil sample for an individual plant tissue sample was collected from beneath the sampled plant (0-3 inches bgs). This depth interval was selected because it is heavily used by plant root hairs, and it is consistent with prior soil sampling depth for the CCT tribal allotments sampled in the 2014 and 2016 Residential Soil Studies and for the ADAs sampled in the 2014 Upland Soil Study. If the plant tissue sample was collected as a composite of multiple individual plants, the co-located surface soil sample was similarly a composite made up of subsampled surface soil collected from beneath each of the individual plants included in the composite tissue sample. This sample was not mass-weighted to be representative of the mass of plant tissue collected from each individual plant. Plants and soil samples were submitted for analysis of TAL metals (except calcium, magnesium, potassium and sodium). Mercury was analyzed in a subset of samples (stem and leaf tissue) based on studies demonstrating mercury is highest in these tissues (Li et al., 2017).

Chemistry data for plant parts of interest were used in Appendix 1 to evaluate the potential for COPC uptake into plants and subsequent exposure of receptor populations who harvest and consume or otherwise utilize those plants. The co-located soil sample results were utilized in the tissue uptake models to estimate muscle concentrations of COPCs in upland birds and game (mammals); the average soil concentration was used as the soil EPC. More information can be found in Appendix 1. Data were reduced as described in Section 2.7.2, including conversion of dw plant tissue data to ww concentrations.

2.7 Data Management

This section describes data compilation and data reduction methods used in this HHRA; methods are in accordance with the Final DMP Amendment No. 1 (TAI, 2019b).

2.7.1 Data Compilation

Data produced by the investigations described above were downloaded from the TAI database (http://teck-ucr.exponent.com/) and compiled into a UCR project-specific SAS® database. Data validation reports and technical memorandums summarizing split sample results were also available in the TAI database.

2.7.2 Data Reduction Methods

As part of regular data management operations, data from each of the investigations were re-formatted to standardize chemical nomenclature and concentration units across studies for each medium. Because the purpose of the HHRA was to evaluate exposures for the UCR, samples collected from rivers, tributaries, and upland areas outside of the UCR Site were excluded, with

the exception of reference areas sampled for crayfish and mussels. In addition, samples collected for the purposes of assessing QC in the field (e.g., field splits, field/trip blanks) and in the analytical laboratory (e.g., laboratory duplicates, matrix spikes) were excluded.

In this HHRA, IC soil samples for upland ADAs, relict floodplains, and residential soil DUs; sediment for residential beaches; and public beach soil and sediment collected in 2015 were treated as individual samples, as described in Section 2.6. Although the DMP states that field duplicate samples should be averaged (TAI, 2019b), replicate IC samples are not collected the same way or for the same purpose as field duplicate samples. Field duplicates are co-located samples collected at the same location to evaluate field sampling precision. IC replicate samples are collected within the same DU, ADA, UDU, SDU, or RFDA as the original IC sample, but at a different starting point. Each IC sample replicate provides an estimate of the mean concentration of COPCs within a DU, ADA, RFDA, SDU, or UDU (Section 7.2, ITRC, 2012); therefore, the IC samples were treated as individual samples and not averaged for use in non-lead risk calculations. IC samples were reduced to a single sample result per the DMP (TAI, 2019b) for use in the COI screens (Section 3.2).

For analysis of surface water, public beach sediment collected in 2009-2011, plant tissue, and fish tissue, field duplicate/triplicate samples were combined with primary field samples to yield one value to represent the sample per the DMP (TAI, 2019b). If concentrations in all samples at a location (primary and field duplicates/replicates) were detected, then the average concentration of the samples was used to represent that sample and that sample was considered a detect. If concentrations in all samples at a location (primary and field duplicates/replicates) were non-detects, the lowest detection limit across the samples was used to represent the detection limit for that sample, and the sample was considered non-detect. If there was a mixture of detected and not detected results among the samples (primary and field duplicates/triplicates), then the average of the detected results was used to represent that sample and the sample was considered a detect. Replicates were not collected for macroinvertebrate or outdoor air samples.

In the project database, analytical results included two types of data qualifiers (laboratory and validation)[27]. If either of these data qualifiers was identified as "R," the result was ranked as rejected and was excluded. If either of these data qualifiers was identified as "U," "UJ," or "EMPC,"[28] the result was ranked as non-detect. For "EMPC"-flagged results, there was no value in the "detection limit" field. For these results, the value in the "meas_value" field was used as the detection limit. For the 2005 fish and sediment data, the database does not include a definition of the value that is used for non-detects. Except for Aroclor results in fish (discussed below), calculations of total PCBs, calculations of toxic equivalents (TEQ), and calculation of

[27] The 2005 fish and sediment data had three qualifier fields: laboratory, project, and validation. The project qualifier field used the value in the validator qualifier field if it was populated; otherwise, it used the value in the laboratory qualifier field.

[28] U = analyte was analyzed for but not detected above the level of the reported sample quantitation limit; UJ = analyte was analyzed for but not detected; the reported quantitation limit is approximate and may be inaccurate or imprecise; EMPC = estimated maximum possible concentration. (Chromatographic peaks are present in the expected retention time window; however, the peaks do not meet all of the conditions required for a positive identification. The detection limit represents the EMPC if the compound was present.) https://www.epa.gov/sites/production/files/2017-01/documents/national_functional_guidelines_for_inorganic_superfund_methods_data_review_01302017.pdf.

total PBDE, all results ranked as non-detect were evaluated in ProUCL (EPA, 2015d) using Regression on Order Statistics (ROS). When calculating estimates of TEQ from dioxin/furan and PCB congeners, non-detects were evaluated at one-half the reported sample detection limit.[29] When calculating estimates of total PCBs, two values were calculated: the sum of detected non-dioxin-like congeners and the sum of detected individual Aroclors. If a sample was analyzed for both PCB congeners and Aroclors, the maximum of the two calculated values was used as the total PCB result (the other value was excluded). Aroclors were non-detects in all samples except for two; both of those samples were analyzed for both Aroclors and congeners, and the total PCB concentration calculated using the congener data set was higher.

The value reported in the "meas_value" field was used as the sample detection limit for non-detects. For values flagged as non-detected values, the measurement value field in the Site database was either the Method Detection Limit or Method Reporting Limit, depending on requirements of the study-specific QAPPs. The value reported in the "meas_value" field was the "detection limit" field for all non-detects except for those flagged as "EMPC."

2.7.2.1 Fish and Plant Tissue Data

Tissue data need to be expressed in terms of ww for use in HHRA. In the 2009 fish data set, some metal results were reported in dw. For samples with a reported fraction of solids result, that percent was used to calculate the ww concentration from the dw concentration using the following formula:

$$\text{Concentration}_{ww} = \text{Concentration}_{dw} \times \text{Fraction of solids}$$

For field duplicate or triplicate samples without a fraction of solids result, the parent sample's fraction of solids result was used to convert dw to ww concentrations. For samples with no fraction of solids result across field duplicate, triplicate, or primary sample, the average fraction of solids (by sample type and species) was used to calculate the concentration expressed as ww.

In the 2016 fish tissue (White Sturgeon), 2018 fish tissue (Northern Pike), and 2018 plant tissue data sets, all metals results were reported as dw concentrations. Each sample was also analyzed for the fraction of solids; therefore, the above equation, with sample-specific fraction of solids result, was used to convert dw tissue concentrations to ww concentrations for those data.

2.7.2.2 Total PCB Concentrations and TEQ Estimation

There are 209 PCB congeners. Of the 209 congeners, twelve congeners are classified as "dioxin-like." There are potential risks associated with possible enhancement of these dioxin-like PCB congeners (toxicologically related to 2,3,7,8-tetrachlorodibenzo-para-dioxin [TCDD]), whereby congener-based analysis can be utilized to ensure that overall PCB risks are not underestimated (EPA, 1996a).

[29] The revised DMP indicates that ProUCL should be used to estimate EPCs for the risk assessments, and that ProUCL includes methods for estimating EPCs with data sets that include non-detects. To calculate total TEQ using congener data, a simpler substitution method of one-half the sample detection limit was used for non-detects in this HHRA.

Because dioxin and furan congeners and dioxin-like PCBs all act by the same mechanism as 2,3,7,8-TCDD, data for the dioxin and furan congeners and dioxin-like PCBs were converted to a TCDD TEQ by computing the sum across congeners of the product of congener-specific concentration and relative Toxicity Equivalence Factor (TEF):

$$TEQ = \sum (C_i \times TEF_i)$$

TEFs were based on EPA (2010a). Separate TEQ values were calculated for dioxins and furans and dioxin-like PCBs assuming that one-half the detection limit for concentrations qualified as non-detects. TEQ values were also calculated using a value of zero for concentrations qualified as non-detects but were not utilized in risk calculations. A calculated TEQ concentration is flagged as a non-detect only if no individual congener was detected in that sample.

Total PCB concentrations were calculated two ways. One was calculated by summing across all detected non-dioxin-like PCB congeners. PCBs generally occur as a mixture of congeners. The second was calculated by summing across Aroclors, which are commercial mixtures of PCB congeners that contain many of the individual congeners in varying ratios. When Aroclors are released into the environment, the original congener composition of the PCB mixture changes because of differential fate and transport processes (EPA, 1996a). Chemical analyses of environmental samples often report PCB concentrations in terms of the Aroclor mixture(s) they most closely resemble. Analysis of PCBs as Aroclors by gas chromatography involves a comparison of the chromatogram of peaks to diagnostic patterns for different commercial Aroclor mixtures (i.e., 1242, 1254, 1260, etc.). The analyst selects the mixture that best represents the observed sample chromatogram when reporting concentrations for an environmental medium (EPA Method 8082A). In some instances, when the observed sample chromatogram has peak characteristics that could be represented by either of two different mixtures (e.g., 1254 and 1260), results may be reported for both; however, these results should be interpreted as alternate estimates of one concentration, not two independent estimates that should be summed. Therefore, total PCB concentrations for each sample (based on Aroclor data) were estimated as follows:

- If a single Aroclor mixture was reported as detect, the total PCB concentration was equal to the detected Aroclor concentration.

- If more than one Aroclor mixture was reported as detect, the total PCB concentration was equal to the sum of the detected Aroclor concentrations.

- If all Aroclor mixtures were reported as non-detect, the total PCB concentration was non-detect and reported as less than the minimum Aroclor detection limit for the sample.[30]

[30] Fish were only analyzed for PCB Aroclors in the 2005 sampling event, and all fish sampled analyzed for Aroclors had at least one detected Aroclor.

3 EXPOSURE ASSESSMENT

Exposure is the process by which receptors (representing hypothetical populations) contact chemicals in the environment. Receptors can be exposed to chemicals in soil, sediment, water, air, or food, and these exposures can occur through several pathways: ingestion, dermal contact, and inhalation. This section summarizes the environmental media and COPCs at the Site, identifies complete human exposure pathways, and describes the methods used to quantify exposure from each pathway.

3.1 Conceptual Site Model

Figure 3-1 presents a Conceptual Site Model (CSM) that summarizes the current understanding of how chemical contaminants that have been released to the environment at the Site might result in exposure of receptors. Figure 5-1 of the HHRA Work Plan (SRC, 2009) details potential sources and fate and transport mechanisms for human exposure. This has been refined in Figure 3-1 to illustrate pathways that can be quantitatively evaluated. As noted in Figure 3-1, sources of chemical contaminants to exposure areas at the UCR Site include the Teck smelter, the Northport smelter, potential related ambient air constituents, and potential smelter-influenced non-point source runoff. The primary environmental media to which humans may be exposed include surface water, beach sediment[31], and outdoor air. Other media evaluated in this Site-wide HHRA include soil, indoor air, indoor dust, and fish.

Two receptor populations included in the CSM are residents of the Colville Reservation and the Spokane Reservation. They are evaluated in Appendices 1 and 2 of this HHRA, respectively. The following Site-related environmental media and their exposure pathways were evaluated exclusively in Appendix 1 and are not discussed in the remainder of this report: macroinvertebrates (mussels and crayfish), amphibians/reptiles, waterfowl, wild/cultural plants, upland birds, and wild game (mammals). Sweat lodge air was not quantitatively evaluated in this HHRA or in Appendices 1 and 2 and is expected to make a negligible contribution to risk (see Appendix 3).

3.1.1 Exposure Areas

An exposure area (also referred to as an exposure unit or exposure point) is an area where a receptor population may be exposed to one or more environmental media. Within the exposure area, contact with each medium is assumed to be random when considered on the time scale of the exposure scenario being evaluated (usually many years). Selection of the bounds of an exposure area is based mainly on consideration of the likely activity patterns of the exposed receptor populations. For example, most recreational visitors access the UCR and Lake Roosevelt from public access areas including campgrounds, swimming areas, boat launches, and marinas. Over the course of multiple years, it is suspected that most individuals will access the UCR Site at many different locations rather than always going to the same exact location. The HHRA Work Plan (SRC, 2009) identified the upper limit (UL) of the size of an exposure area for recreational activities as a river reach (described in Section 2.2.1.4 above), and the lower limit of an exposure area as a single beach. That definition of exposure area has evolved with the design

[31] In the CSM, "sediment" includes both beach sediment and beach soil.

of environmental sampling efforts for soil, outdoor air, surface water, sediment, and biota, and the analysis of the survey data.

The RecUse Survey (IEc, 2012) gathered information on resource use by recreational visitors to Lake Roosevelt, and the CCT Tribal Survey (Westat Inc., 2012) collected information on Local Area resource use by residents of the Colville Reservation. Merging the survey data with the data collected from the various environmental media (e.g., fish tissue, surface water and beach sediment, soil, and outdoor air) required consideration of the differences in the spatial scales the data represent. For example, the RecUse Survey may be used to estimate the number of beach trips per year for three regions of the UCR (upper, middle, and lower) while the surface water, fish, and macroinvertebrate data were collected by river reach, as described in Section 2.6. Sediment data were collected on a beach-specific basis.

Table 3-1 presents the exposure areas defined for evaluation in this risk assessment. These are described in the following subsections by medium.

3.1.1.1 Sediment

The public beach sediment study was designed to allow for exposures in the HHRA to be calculated on a beach-specific basis (TAI, 2009b). Recreational visitors to public beaches were assumed to receive all of their exposure to surface sediment by frequenting the same beach. Public beach sediment was evaluated on a beach-by-beach basis, with each beach representing an exposure area and using the same value for exposure frequency for all beaches. For Bossburg Flat Beach and Evans Campground Beach, the SDUs sampled in 2015 (TAI, 2016a) were additional exposure areas.

For subsurface sediment, only data from SDUs sampled in 2015 at Bossburg Flat Beach and Evans Campground Beach were sieved to a grain size appropriate for evaluating human health exposures. As such, the exposure areas for subsurface sediment are only the 2015 SDUs from Bossburg Flat Beach, Evans Campground Beach, and the former cable ferry landing location (F-01). The size of the exposure area was assumed to be the size of the SDU sampled.

Residential beaches were sampled as part of the 2014 and 2016 residential soil studies (Figures 3-2 and 3-3) to assess potential risks to existing residents from exposure to metals in the sediments of beach areas on their properties. The size of the exposure area was assumed to be the size of the DU sampled.

3.1.1.2 Surface Water

The exposure area for surface water was identified as UCR Reaches 1 through 6. The rationale for combining the surface water data to estimate one EPC for the Site was based on the number of disturbed, unfiltered samples that were available for each reach of the UCR, as discussed in Section 2.6.1. Surface water samples were collected from one transect per river reach. Each reach of the UCR includes 12 to 59 miles of surface water, making a statistical comparison among the reaches tenuous. Additionally, the differences in the numbers of beach trips per year, and the time spent swimming and wading in water more than waist deep during beach trips, are not substantially different among the three regions of Lake Roosevelt (“upper,” “middle,” and “lower”; Figure 3-4; SRC, 2019a). Therefore, a single exposure area for surface water (i.e.,

incidental ingestion of and dermal contact with surface water while swimming) was used (Section 3.5.2.2).

3.1.1.3 Fish

The measured concentrations of COPCs in fish tissue collected from all six river reaches were used to calculate EPCs; the exposure area for fish was Reaches 1-6. The rationale for combining the fish tissue data across reaches, by species, was an assumption that some of the fish species have home ranges that span more than one river reach, resulting in exposures throughout the river. Fish risks were estimated for individual species. Concentrations in individual species sampled, except for sucker (*Catostomus* sp.), did not vary greatly by river reach. The fish species with the highest lead concentration was sucker; the Reach 1-6 EPC for sucker is the Kaplan Meier (KM) mean of 0.135 mg/kg ww (Table 3-29). Lead in sucker was highest in fish collected in Reach 1 (0.393 mg/kg ww) and Reach 2 (0.183 mg/kg ww) and ranged from 0.0642 to 0.0993 in sucker collected from Reaches 3 to 6. Because sucker were not reported as being frequently consumed by anglers surveyed in the RecUse Survey or the CCT Tribal Survey, the EPC for sucker was Reaches 1-6 as well.

3.1.1.4 Soil

As described in Section 2.6, soil samples were collected in 2014 from residential properties in the northernmost reaches of the Columbia River valley (north of the town of Northport, Washington to the U.S. – Canada border; Figure 2-10) to assess potential risks to existing residents from exposure to metals in surface soils. Based on the results of the 2014 soil study, additional residential properties located along the UCR from the U.S. – Canada border to approximately the intersection of Williams Lake Road and Highway 25 on the east side of the UCR were sampled in 2016 (Figure 2-11). Residential soil sampling efforts focused on locations on a property where there was a high potential for exposure to surface soil by residents, especially children. These locations were identified based on property-specific interviews with residents.

Soils in upland ADAs were sampled in the northernmost 100 mi^2 of the Site, extending south from the U.S. – Canada border, as part of the 2014 upland soil study for use in HHRA (Figure 2-8). These ADAs are located on privately owned land, U.S Forest Service or Bureau of Land Management land, or land managed by the Washington Department of Natural Resources. They are designated residential, undeveloped, forest land, or government land, with the majority having the land use code "designated forest land."[32]

Relict floodplains were sampled in 2014 as described in Section 2.6 to assess historical fluvial deposition of sediment and potential risks to recreational visitors who may frequent those areas to access the UCR. The size of the exposure area for this medium was assumed to be the size of the RFDA sampled. Relict floodplain samples were treated as beach soil in this risk assessment. For Bossburg Flat Beach, UDUs sampled in 2015 (TAI, 2016a) were additional beach soil exposure areas.

[32] http://propertysearch.trueautomation.com/PropertyAccess/Property.aspx?cid=0.

Current and future residents exposed to soil at DUs and ADAs, recreators exposed to relict floodplain soil at RFDAs and public beach soil at UDUs, and outdoor workers exposed to public beach soil at UDUs were assumed to receive all of their exposure to COPCs in soil in each DU, ADA, RFDA, or UDU sampled. Therefore, the size of the exposure area for soil was assumed to be the size of the DU (individual DUs on residential properties), ADA, RFDA, or UDU sampled.

3.1.1.5 Outdoor Air

Outdoor air samples were collected at one location near Northport, Washington, by Teck (see Figure 2-12; ECY, 2017a)[33]. Data used in this HHRA were collected from 2002 to 2009. Air concentrations based on a single location may not be spatially representative of other areas throughout the Site. In the absence of other data, however, the exposure area for outdoor air was identified as the entire Site.

3.1.1.6 Indoor Air

No indoor air samples were collected; indoor air EPCs were based on outdoor air data. As such, the exposure area for indoor air was identified as the entire Site.

3.1.1.7 Indoor Dust

No indoor dust samples were collected; indoor dust EPCs were based on outdoor residential or upland soil data. As such, the exposure area for ingestion of indoor dust was based on the COPC concentration for each DU on a residential property or each ADA on a potential future residential property.

3.1.2 Exposed Populations

Risks from exposure to COPCs were evaluated for hypothetical populations that represent subsets of hypothetical populations who live near the UCR, visit or work on its beaches, or eat fish, shellfish, game, or cultural/wild plants harvested from the area. These hypothetical populations are referred to in the report as "receptors." Receptor populations are constructed with exposure parameters (e.g., intakes of selected media, exposure frequencies and durations) to represent typical (most likely) exposures, referred to as "central tendency exposures" (CTE), or greater than typical exposures, referred to as "reasonable maximum exposures" (RME). Receptor populations are hypothetical because they are restricted to exposures to selected media and defined media intakes; therefore, they may not represent any single individual or real population, whose members engage in a broader diversity of activities and intensities (duration, frequency) than assumed in the receptor population. For example, the outdoor worker receptor is assumed to be exposed exclusively during worker activities. However, workers may live in the area and may recreate at the UCR. The recreational visitor receptor is assumed to be exposed exclusively during intermittent visits to the Site. However, some recreators may also be local residents.

Five receptors (hypothetical populations) were evaluated in this risk assessment:

[33] ECY's Air Quality Program also collected data from Northport, Washington, from 1992 to 1998.

- *Residents* who contact soil in their yards and dust in their homes, who breathe air, who may eat fish from the UCR, and who may contact surface water and sediment if they have a beach on their property;
- *Outdoor workers* who contact surface and subsurface sediment or soil at public beaches, who contact UCR surface water, and who breathe air;[34]
- *Recreational visitors* who use the UCR for beach day trips, boating, camping, swimming, and fishing and thereby contacting surface sediment and beach soil at public beaches and relict floodplains, contact UCR surface water, breathe air, and may eat fish harvested from the UCR;[35]
- *Colville High Intensity Resource Users* (CHIRU), members of the CCT who have contact with soil, beach sediment, and UCR surface water, who breathe air, and who may eat fish, mussels, crayfish, amphibians/reptiles, waterfowl, wild/cultural plants, upland birds, and wild game (mammals) harvested from the Site; and
- *Spokane Tribe of Indians* (STI) members who have contact with soil, beach sediment, and UCR surface water, who breathe air, and who may eat fish harvested from the UCR.

EPA (1989) recommends evaluating exposure and risk to individuals using intakes that are near the upper end of the range (e.g., the 95th percentile), and "average," near the central portion of the range. These exposure scenarios are referred to as the RME scenario when the intake parameters are from the upper end of the range, and the CTE scenario when the average values are used. This section of the HHRA discusses residents, outdoor workers, and recreational visitors. The HHRA for the CHIRU population is presented in Appendix 1, and the HHRA for the STI population is presented in Appendix 2.

3.1.2.1 Residents

The residential receptor population consists of people who reside in communities near the UCR now (at locations sampled during the 2014 and 2016 residential soil studies) or may do so in the future (at locations sampled during the 2014 upland soil study). The UCR area includes several towns and communities outside of the Colville and Spokane reservations that are adjacent to or near the river. Demographic profiles based on the 2010 United States census are available for some of the larger communities (U.S. Census Bureau, 2020). The population of Northport in 2010 was 295 residents, consisting of 139 households and 76 families. The median age was 48.5 years old; 21% of residents were under the age of 18. The population of Marcus was 183 residents in 2010, with 49 families comprising 76 households; 18% of residents were under the age of 18 years. Kettle Falls had a population of 1,595 living in 676 households, according to the 2010 census. Of these, 26.6% were under the age of 18 years. The population of Inchelium in 2010 was 409 residents, 27.4% of whom were under the age of 18 years. Coulee Dam had a

[34] Workers in the area may also be exposed in upland areas away from the river (e.g., in the forest or agricultural industries). As discussed in Section 3.1.2.2, because upland areas are evaluated for residential land use with higher exposure assumptions than occupational exposure, the residential exposure evaluation is protective of the upland outdoor worker population; a separate evaluation is not warranted.

[35] The recreational visitor population also encompasses visitors from completely outside the UCR and Lake Roosevelt community and corridor.

population of 1,098 residents in 2010, divided among 301 families and 459 households. Of the 1,098 residents, 24.8% were under 18 years of age.

Because exposures for children and adults can be different, the residential receptor population consists of adults and children (assumed to be <6 years old). For some COPCs (e.g., lead) and exposure media (e.g., soil), young children represent the most sensitive and most exposed receptor population and risks estimated for this age group will not underestimate risks for older children. Residents may have direct contact with surface soil in their yards over a long period of time (26 years, the assumed average residence time at the Site); they may also be exposed to COPCs while breathing outdoor air on their properties, as well as exposure to air and dust inside their homes. Current and potential future residential use was defined by soil datasets: "current" residents were assumed to be potentially exposed to soil from DUs sampled during the 2014 and 2016 residential soil studies, and "future" residents were assumed to be potentially exposed to soil from ADAs sampled during the 2014 upland soil study. Residents who own beaches that were sampled as part of the 2014 and 2016 residential soil studies may also have direct contact with sediment and surface water on their beaches over a long period of time (assumed to be 26 years).

Residents are likely to eat fish from the UCR, given their proximity to the river and the strong possibility that the local resident population overlaps to some degree with the recreational visitor population. For this evaluation, residents with and without beaches were assumed to consume fish having a tissue concentration representative of each individual fish species sampled. Residents may also eat cultivated terrestrial plants; however, risk management decisions for residential soil that are protective of full-time residential soil exposures are expected to be protective of gardening exposures in those soils. EPA determined that this is an acceptable approach for gardens, where direct soil ingestion is the source of the majority of lead risk (EPA, 2014b). Garden soils tend to have lower lead concentrations which may result from cultivated gardens including soil amendments which dilute lead in soil or decrease lead bioavailability (e.g., Brown and Chaney, 2016). The most sensitive receptor for lead is the young child, who is not likely to be an avid gardener. Because soils were evaluated DU-by-DU, including garden DUs (i.e., assuming that a young child is a full-time resident of a garden DU) is more protective than evaluating a gardener receptor population. As such, that exposure pathway, while complete, was not evaluated.

3.1.2.2 Outdoor Workers

Many types of work could potentially result in exposures to Site-related contaminants, which will vary with the location and type of work performed. Because it is assumed that an outdoor worker is likely to be more exposed to Site-related chemical contamination than an indoor worker, the outdoor worker was selected as the worker population evaluated in this HHRA. This receptor was assumed to work full-time at campgrounds and boat launches along the river, engaging in activities similar to those conducted by a park ranger or boat dock worker. Work activities are assumed to vary widely, ranging from occasional excavation activities in warmer months, such as repair of dock pilings or as part of maintenance activities at recreational facilities, to activities that involve less intensive sediment and beach soil exposure. These

outdoor workers may be exposed to public beach surface[36] and subsurface sediment and beach soil, UCR surface water, and outdoor air now or in the future, for a long period of time (around 25 years). Exposures to the outdoor worker receptor are assumed to occur exclusively during work activities.

The outdoor worker receptor population defined for evaluation in this HHRA is likely more exposed to Site-related chemical contamination than either workers who work full-time with minimal sediment/beach soil exposure, or workers whose activities focus on sediment/beach soil-intensive activities but only work part-time. Workers in the area may also work full-time in upland areas away from the river (e.g., in the forest or agricultural industries). Upland areas are evaluated for residential land use in this HHRA; the potential future residential exposure evaluation is protective of the upland outdoor worker population and a separate evaluation for upland workers is not warranted.

3.1.2.3 Recreational Visitors

At the UCR Site, there are numerous campgrounds, public access beaches, boat launches, and picnic areas which are frequented by recreational visitors who engage in a variety of activities, including camping, swimming, fishing, hunting, boating, and hiking. Recreational visitors may be exposed to COPCs in surface water, surface sediment, beach surface soil in RFDAs and UDUs, and outdoor air while engaging in these activities at the UCR. For this HHRA, recreational visitors were also assumed to fish; swim during trips to the beach, boat, and camp; and spend time on UCR public beaches and relict floodplains. Relevant Site-specific exposure parameters for these pathways were derived from the RecUse Survey.

Recreational visitors may also be residents or outdoor workers; however, different risk scenarios may not be combined, as the scenarios are independent. Risks of a combined residential, occupational, and/or recreational exposure were not evaluated in this HHRA. Adult and child (aged 0-6 years) recreators may be exposed to contaminants in outdoor air by breathing outdoor air while recreating along the UCR. They may also be exposed to contaminants in surface sediment, beach surface soil, and surface water on day trips to beaches (including swimming) or relict floodplains, to surface sediment, beach surface soil, and surface water while swimming on boating and camping trips, and they may also be exposed to contaminants by consuming fish caught from the UCR. It is assumed that young children represent the most sensitive and most highly exposed child receptor population and that evaluation of this age group will be protective of older children who may also recreate in the area.

3.1.3 Exposure Pathways

As described above, several types of media may be contaminated at the Site, including sediment, surface water, air, soil, and biota. Figure 3-1 presents the likely routes by which residential, recreational visitor, and outdoor worker populations might contact contaminants in the environment. As shown, receptors may be exposed to contaminants in primary, secondary, or tertiary environmental media by ingestion, inhalation, and dermal contact. Primary, secondary, and tertiary refer to how many steps the exposure medium is from the source of contamination.

[36] For lead, surface sediment exposure was not assessed for outdoor workers at public beaches because decisions based on the child recreator scenario are protective of the outdoor worker population.

"Primary" refers to the original source (e.g., the discharge point) of a chemical constituent, while "secondary" and "tertiary" sources are environmental media (abiotic or biotic) that receive chemical inputs from a primary (or secondary) source. For example, air is a primary source of contaminants from a smokestack, while soil is a secondary source (contaminants are first in the air, then deposited on soil). The terms do not convey any prioritization of importance, but rather refer to their transport in the CSM. Only exposure pathways considered complete (known with certainty to occur) and for which data were available were carried through the remainder of this HHRA.

3.1.3.1 Incidental Ingestion of Surface Soil

Even though few people intentionally ingest soil, residents who have direct contact with surface soils at the Site ingest small amounts that adhere to their hands during outdoor activities. In addition, children, especially those under 6 years of age, ingest more soil because of frequent hand-to-mouth or object-to-mouth behaviors. Incidental ingestion of soil is often one of the more important routes of human exposure at a site.

3.1.3.2 Dermal Contact with Surface Soil

Residents who contact contaminated soils may have soil adhere to their skin. Uptake of metals across the skin into the body from contact with soil is considered a minor exposure pathway because of the relatively low tendency of metals to cross the skin. However, dermal exposure to soil is a complete exposure pathway and, therefore, is evaluated for COPCs for which there are estimates of dermal absorption parameters (e.g., permeability coefficients or absorption fractions), including arsenic and cadmium (EPA, 2001c).

Quantifying uptake from dermal exposure to lead is not recommended because of the uncertainty in assigning a dermal absorption fraction that would apply to the numerous inorganic forms of lead that are typically found in the environment (including water, soil, or sediment). Furthermore, uptake of lead across the skin from contact with soil is generally considered a minor exposure pathway relative to incidental ingestion (because of the relatively low tendency of metals to cross the skin even when contact does occur). Therefore, exposure to lead via dermal contact with soil was not evaluated quantitatively in this risk assessment.

3.1.3.3 Incidental Ingestion of Indoor Dust

Outdoor soil is potentially tracked into buildings (such as residences), leading to contamination of indoor dust and subsequent ingestion via hand-to-mouth activities. Residents may be exposed to COPCs in indoor dust via incidental ingestion.

3.1.3.4 Incidental Ingestion of Surface Sediment at Public Beaches or Residential Beaches

A child or adult recreational visitor may engage in activities such as wading, splashing, swimming, or general beach-going activities at public beaches adjacent to the UCR. A resident may similarly recreate on beaches located on his or her property. Outdoor workers may also be in contact with sediment through occupational activities. Although it is not expected that any of these receptor populations intentionally ingest sediment, recreational, and occupational activities can lead to the incidental ingestion of small amounts of surface sediment.

3.1.3.5 Dermal Contact with Surface Sediment at Public Beaches or Residential Beaches

Skin contact with surface sediment can occur while engaged in recreational activities such as wading, splashing, swimming, or other activities on beaches. Receptor populations who can experience dermal contact with sediments include recreational visitors to the Site, residents who own property adjacent to the UCR (e.g., residential beaches) or outdoor workers. Uptake of metals across the skin from contact with sediment is generally considered a minor exposure pathway because of the relatively low tendency of metals to cross the skin. Lead was not assessed for the dermal pathway (as discussed in Section 3.1.3.2). However, dermal exposure to surface sediment is a complete exposure pathway and, therefore, was evaluated for non-lead COPCs with dermal absorption data (EPA, 2001c).

3.1.3.6 Incidental Ingestion of Surface Soil at Public Beaches and Relict Floodplains

A child or adult recreational visitor may engage in activities such as wading, splashing, swimming, or general beach-going activities at public beaches or on relict floodplains adjacent to the UCR. Outdoor workers may also be in contact with beach soil through occupational activities at public beach UDUs. Although it is not expected that any of these receptor populations intentionally ingest soil, recreational and outdoor worker activities can lead to the incidental ingestion of small amounts of beach surface soil at relict floodplains and upland areas of public beaches.

3.1.3.7 Dermal Contact with Surface Soil at Public Beaches and Relict Floodplains

Skin contact with surface soil can occur while engaged in recreational activities such as wading, splashing, swimming, or other activities on beaches or relict floodplains adjacent to the UCR. Receptor populations who can experience dermal contact with soils include recreational visitors to the Site and outdoor workers (at UDUs). Uptake of metals across the skin from contact with soil is generally considered a minor exposure pathway because of the relatively low tendency of metals to cross the skin. Lead was not assessed for the dermal pathway (as discussed in Section 3.1.3.2). However, dermal exposures to surface soil are complete exposure pathways and, therefore, were evaluated for non-lead COPCs with dermal absorption data (EPA, 2001c).

3.1.3.8 Incidental Ingestion of Public Beach Subsurface Soil and Subsurface Sediment

Outdoor workers may be in contact with subsurface soil or sediment at public beaches during occasional occupational activities such as digging holes, trenches, and footings, which can lead to the incidental ingestion of small amounts of soil or sediment. Subsurface soil data for the <149 µm fraction are only available for the 2015 Bossburg Flat beach SDUs; quantitative risk calculations could only be done using subsurface data from that sampling event. Subsurface sediment data for the <250 µm fraction are only available for the 2015 Bossburg Flat and Evans Campground beach SDUs and the former cable ferry landing sample (F-01); quantitative risk calculations could only be done using subsurface data from that sampling event.

Subsurface sediment core samples were collected at public beaches where discrete surface samples were collected during the 2009-2011 sampling event (see Section 2.6.2.2). The core samples were only sieved to the <2mm particle size fraction. The subsurface samples were

collected from 0-15, 15-30, and 30-45 cm depth intervals with soil cores.[37] Table 3-2 compares the mean concentrations of COPCs in the 0-15 cm depth interval (core sediment) to the concentrations in the entire 0-45 cm depth interval on a Site-wide comparison (comparisons for each beach are provided in Appendix 9). Table 3-2 shows that the Site-wide concentrations of COPCs in the subsurface sediment are similar to surface sediment concentrations. Thus, risk estimates based on Site-wide surface sediment exposure are unlikely to appreciably underestimate risks from exposure to subsurface sediment.

3.1.3.9 Dermal Contact with Public Beach Subsurface Soil and Subsurface Sediment

Outdoor workers engaged in occupational activities at beaches may be exposed to subsurface soil or sediment by skin contact. As noted previously, uptake of metals across the skin from contact with subsurface soil or sediment is generally considered a minor exposure pathway because of the relatively low tendency of metals to cross the skin (consequently, dermal exposure was not assessed for lead, as discussed in Section 3.1.3.2). However, dermal exposure to subsurface soil or sediment is a complete exposure pathway and, therefore, was evaluated for non-lead contaminants for which there are estimates of dermal absorption parameters (e.g., permeability coefficients or absorption fractions). For most public beaches, subsurface sediment data are only available for the <2 mm particle size (see previous section for further discussion); quantitative risk calculations could only be done using subsurface data from the 2015 Bossburg Flat and Evans Campground sampling event.

3.1.3.10 Incidental Ingestion of UCR Surface Water

A child or adult recreational visitor, or a resident with a beach on his or her property, may engage in activities such as wading, splashing, swimming, general beach-going, or fishing at public/private beaches along the UCR. Outdoor workers may also be in contact with UCR surface water at public beaches along the UCR. Although it is not expected that these receptors intentionally ingest surface water, such activities can lead to the incidental ingestion of small amounts of surface water.

3.1.3.11 Dermal Contact with UCR Surface Water

Skin contact with surface water can occur while engaged in recreational activities such as wading, splashing, swimming, or other activities on beaches, as well as occupational activities. Receptor populations who can experience dermal contact with surface water include recreational visitors to the Site, residents who own beaches, and outdoor workers. As with dermal contact with sediment and soil, uptake of metals across the skin from contact with surface water is generally considered a minor exposure pathway because of the relatively low tendency of metals to cross the skin (dermal exposure was not assessed for lead, as discussed in Section 3.1.3.2). However, dermal contact with surface water is a complete exposure pathway and, therefore, was evaluated for non-lead COPCs for which there are estimates of dermal absorption parameters (e.g., permeability coefficients or absorption fractions).

[37] IC samples were also collected in 2015 from the subsurface at SDUs located at Bossburg Flats Beach and Evans Campground Beach.

3.1.3.12 Ingestion of Groundwater

In the HHRA Work Plan (SRC, 2009), ingestion of groundwater was considered a potentially complete exposure pathway. Further evaluation of water quality monitoring records for all public water systems (including well-sourced systems) in the northern portion of the Site resulted in a conclusion that groundwater/well water is an incomplete exposure pathway (CH2MHill, 2018b). Although groundwater/well water may be utilized by local residents, there is not a compete pathway from Site-related contaminants to groundwater. Detailed information regarding this evaluation is found in Appendix 10. Therefore, this pathway was not evaluated in this HHRA.

3.1.3.13 Inhalation of Airborne Particulates in Outdoor Air

The Teck smelter in Trail, B.C. continues to operate and release aerial emissions. Although Teck implemented a process change in the lead smelting operation that lowered emissions in the 1990s, arsenic, cadmium, lead, and zinc were detected in air samples collected at a station near Northport (Figure 2-12) from 1999 through 2009; as noted above, data collected from 2002 through 2009 were utilized in this HHRA. More recent air monitoring data from the Site are not available but may be lower than data used in this assessment[38] (Figure 2-13). In addition, whenever contaminated soils are exposed at the surface, fine-grained particles of contaminated surface soil may become suspended in air by wind or human disturbance and inhaled. In cases where the soil is disturbed only by wind or walking, it is assumed that the amount of particulate material inhaled from air is generally quite small compared to the amount that is typically assumed for incidental ingestion. Residents, recreational visitors, and outdoor workers engaging in activities outdoors at the Site may inhale COPCs in outdoor air.

3.1.3.14 Inhalation of Airborne Particulates in Indoor Air

As described in the previous section, COPCs in soil may become suspended in air by wind or human disturbance. Outdoor air may be transferred indoors via open windows and/or doors. In the absence of Site-specific data, concentrations of COPCs in air inside residences were assumed equal to concentrations in outdoor air. For residents, time spent inside the home and time spent outdoors have been estimated on a nationwide basis, as described in the EPA Exposure Factors Handbook (EPA, 2011a).

3.1.3.15 Consumption of Fish

Adult and child recreational visitors and residents with and without beaches on their property consuming locally-caught fish may ingest COPCs that accumulate in edible portions (e.g., fillets) of fish from the UCR.

3.1.3.16 Inhalation of Sweat Lodge Air

Residents who participate in sweat lodge activities may contact COPCs in UCR surface water that is heated by contact with heated rocks in the sweat lodges. Because non-volatile metals will not vaporize at sweat lodge temperatures, intake of metals will be from ingestion of inhaled water droplets rather than from absorption from the respiratory tract. Ingestion of COPCs in surface water spray in a sweat lodge is a negligible contributor to risks, relative to ingestion of

[38] For additional air monitoring data from Trail since 2009, see http://www.thep.ca/pages/airquality and https://pollution-waste.canada.ca/national-release-inventory/archives/index.cfm?do=facility_history&lang=En&opt_npri_id=0000003802&opt_report_year=2017.

unfiltered disturbed surface water from direct contact during other water activities such as swimming and wading. As such, this exposure pathway, though complete, was not quantitatively evaluated (see Appendix 3 for more detailed discussion).

3.2 Selection of Chemicals of Potential Concern (COPCs)

The HHRA work plan for the UCR Site (SRC, 2009) presented a list of initial COIs developed using information about known and potential sources of contamination and data obtained during investigations and monitoring events near or at the Site. The COI list included metals and metalloids, pesticides, SVOCs, PAHs, PCBs, dioxins/furans, PBDEs, and radionuclides associated with the uranium-238 decay chain. COIs were evaluated in a risk-based screen to identify COPCs described below. The list of COIs analyzed differed by each medium depending on the Site-specific data collected during RI sampling events.

Typically, only a few contaminants at a site are responsible for most of the human health risk because of the concentrations present, the toxicity of the contaminants, and their behavior in environmental media (e.g., fate and transport, mobility, bioaccumulation potential). At large complex sites such as the UCR, a key step in data evaluation is reducing the number of COIs for each medium to a smaller number of COPCs. This allows the HHRA to focus on the contaminants that are likely to be driving risk. Guidance for selecting COPCs is provided in the EPA's *Risk Assessment Guidance for Superfund (RAGS) Part A* (EPA, 1989), as well as the *Soil Screening Guidance: User's Guide* (EPA, 1996c) and *Technical Background Document* (EPA, 1996b). These selection criteria generally include the frequency with which a COI is detected, whether it is considered an essential human nutrient, its concentration relative to background (non-site-related) concentrations, and its concentration relative to a toxic concentration (through comparison to risk-based screening levels [RBSLs]). Comparison of COI concentrations to background concentrations was not conducted as part of the selection of COPCs for this Site. Information on background for inorganic COPCs in soil is presented in Section 6 of this HHRA.

In the absence of policy that incorporates findings from current scientific literature that supports a specific target blood lead concentration (PbB), lead is considered a COPC in exposure media where it is found at concentrations that are above the detection limit. Lead was retained as a COPC for all environmental media.

3.2.1 COPC Screening Process

Specific methods for screening non-lead COIs to determine COPCs in each medium are described in the following subsections, but follow the general steps listed below:

1. Calculate the frequency of detection of each COI in the medium. Retain any COI that is detected at a frequency of ≥5%.
2. Remove essential human nutrients that have no toxicity values (i.e., calcium, magnesium, potassium and sodium).
3. Calculate medium-specific RBSLs for each COI based on the exposure scenario that results in the highest exposure; remove any COI with a maximum detected concentration that is less than the RBSL for that medium.

The presence of COIs above levels of detection may vary spatially, because of source locations and release rates, fate and transport mechanisms, and other local conditions. Therefore, in addition to Site-wide detection frequencies, detection frequencies were also calculated by river reach (for beach sediment, surface water and fish tissue), and by species (for fish tissue). If a COI was detected at a frequency of ≥5% within a reach or within a species, that COI was added to the Site overall COPC list for that medium. River reach- and species-specific COPC lists were not developed.

3.2.2 Calculating Risk-Based Screening Levels (RBSLs)

RBSLs were calculated following the Regional Screening Level (RSL) approach described by EPA on its "*Regional Screening Levels (RSLs) for Chemical Contaminants at Superfund Sites*" screening level/preliminary remediation goal (PRG) website.[39] This website (referred to herein as the "RSL Calculator" [EPA, 2019a]) provides current toxicity values, default exposure parameters, and equations for calculating RBSLs. These screening levels are not clean-up levels, but rather are intended to determine whether measured levels of COIs warrant further investigation at a site.

RBSLs are generally based on full-time residential exposure, because exposure parameters for residents typically yield higher exposures than for other receptor populations. However, at the UCR Site, other receptors may have greater exposure to various media than predicted by standard residential exposure parameters, depending on the exposure scenario (e.g., receptors evaluated using parameters derived from the CCT Tribal Survey). To determine the RBSL to be used to screen COIs for each medium, intake for each receptor population potentially exposed to the medium (based on the CSM) was estimated as shown in Table 3-3. The exposure parameters associated with the receptor population with the greatest potential exposure ("Intake for Screening" in Table 3-3) were entered using the "site-specific" function of the RSL Calculator to determine the RBSL for that medium. The COI screen was conducted for the residential, recreational, outdoor worker, and CCT receptor populations (the "CCT receptor population" refers to receptors evaluated using exposure parameters derived from the CCT Tribal Survey).

Risk assessment is an iterative process, and the first draft of this HHRA was prepared in 2017-2018. At that time, the residential receptor population was evaluated as two separate populations: one using standard residential exposure parameters, and one using exposure parameters from the CCT Tribal Survey to represent and be protective of CCT residents living on tribal allotments in the vicinity of the Site (the "CCT Receptor" shown in Table 3-3). These are the populations, along with recreational visitors and outdoor workers, that were used when deriving RBSLs to use in the COI screen. Since that time, evaluation of the CCT and STI populations has evolved (including modifications to intake rates that could impact the results of the COI screen); however, the COI screen and resulting list of COPCs were not re-calculated for each subsequent draft of the HHRA. As a result, the fish daily consumption rates (DCRs) used to evaluate the CHIRU receptor in Appendix 1 and the STI population in Appendix 2 are not the DCRs used in the COI screen to derive RBSLs for use in determining COPCs. It is unlikely that updating the COI screen using the intake rates that are currently presented in this HHRA would substantially change the outcome of the assessment.

[39] http://www.epa.gov/risk/regional-screening-levels-rsls.

RBSLs were calculated using an excess cancer risk of 10^{-6} and a hazard quotient (HQ) of 0.1. Current toxicity values for COIs are included in the RSL Calculator.[40] Based on the antimony, mercury, and vanadium species expected to exist in Site soils, the RSL Calculator toxicity values for metallic antimony, mercury,[41] and vanadium and compounds (respectively) were selected for these analytes (Diamond and Thayer, 2011a, 2011b, 2012). In the absence of speciation information for nickel and thallium, the available toxicity values for compounds containing these elements were evaluated and the following were selected: for nickel, the toxicity values for nickel refinery dust were used,[42] and for thallium, the toxicity values for soluble thallium salts were used. For chromium, toxicity values for both tri- and hexavalent species were retained, and screening was performed using the toxicity values for hexavalent chromium (Cr(VI)).[43] For COIs with both a carcinogenic and noncarcinogenic RBSL, the lower of the two values was used for screening. If a chronic toxicity value for a COI was not listed in the RSL Calculator, the following sources were consulted for updated information since the last RSL Calculator update, according to Office of Solid Waste and Emergency Response (OSWER) Directive 9285.7-53 (EPA, 2003d) for the hierarchy of toxicity values:

Tier 1:

- EPA's Integrated Risk Information System (IRIS)[44]

Tier 2:

- EPA's Provisional Peer-Reviewed Toxicity Values for Superfund (PPRTVs)[45]

Tier 3:

- California EPA[46]
- Agency for Toxic Substances and Disease Registry (ATSDR) Minimal Risk Levels (MRLs)[47]
- EPA Health Effects Assessment Summary Tables (HEAST)[48]

As Tier 3 allows for other sources of toxicity values not enumerated in EPA (2003d), the following sources were also consulted for chronic toxicity values:

[40] The 2013 Agency for Toxic Substances and Disease Registry (ATSDR) intermediate-duration oral minimal risk level (MRL) for uranium is recommended for use as the chronic oral reference dose (RfD) by EPA (2016b, 2019a).
[41] The toxicity value for methylmercury was used in the RBSL calculation for tissue. For soil, sediment, and surface water, the toxicity value for mercuric chloride was used (SRC, 2015a).
[42] For nickel, the toxicity values for nickel refinery dust were used for solid media. For surface water, nickel refinery dust is not an appropriate toxicity value, as the dissolved fraction of the metal in surface water is most relevant. For this medium, the toxicity value for nickel soluble salts was used.
[43] These toxicity values, and those for the other COIs, are discussed in more detail in Section 4.
[44] https://cfpub.epa.gov/ncea/iris2/atoz.cfm.
[45] https://hhpprtv.ornl.gov/quickview/pprtv.php.
[46] http://www.oehha.ca.gov/risk/chemicalDB//index.asp.
[47] http://www.atsdr.cdc.gov/mrls.html.
[48] https://cfpub.epa.gov/ncea/risk/recordisplay.cfm?deid=2877.

- Health Canada[49]
- World Health Organization (WHO) Environmental Health Criteria[50]
- Texas Commission on Environmental Quality[51]

As discussed in the HHRA work plan (SRC, 2009), COIs that do not have a Tier 1, 2, or 3 chronic toxicity value from the sources listed above, or that do not have an appropriate surrogate toxicity value from a chemical similar in composition as specified in the work plan, were removed from quantitative consideration as a COPC. These are discussed qualitatively in Section 6.

For COI screening, concentrations of dioxins, furans, and dioxin-like PCB congeners were converted to TEQs for comparison with the RBSL for TCDD. Concentrations of detected non-dioxin-like congeners or detected Aroclors were summed for comparison with the RBSL for total PCBs. If a sample was analyzed for both PCB congeners and Aroclors, the maximum of the two summed values was used as the total PCB result. PBDE congeners that lack toxicity values were summed in any exposure medium in which they were detected and included in the discussion of chemicals without toxicity values in the uncertainty section (Section 6). Specific screening methods for exposure media are discussed below.

3.2.3 Surface Water Chemical of Interest (COI) Screening

Surface water is a primary medium at the Site with potential contamination from COIs. Surface water data were collected in 3 sampling rounds from multiple transects in the UCR in 2009 and 2010 as described in the QAPP (TAI, 2009c) and summarized in Exponent (2013b). The maximum detected COI concentrations measured in unfiltered (total), disturbed surface water samples from UCR Reaches 1 through 6 were compared to RBSLs. Results were used to evaluate incidental ingestion of and dermal contact with surface water while swimming and wading, and during other activities near the shore. The following approach was used to select COPCs for surface water:

1. Calculate the frequency of detection of each COI in disturbed (total) surface water for Reaches 1-6 and reach-by-reach; retain COIs that are detected at a frequency of ≥5% for all reaches combined or reach-by-reach.[52]
2. Remove essential human nutrients that have no toxicity values (i.e., calcium, magnesium, potassium and sodium).

[49] https://www.canada.ca/en/health-canada/services/environmental-workplace-health/reports-publications.html.
[50] http://www.who.int/ipcs/publications/ehc/en/.
[51] https://www.tceq.texas.gov/toxicology.
[52] For surface water, the frequency of detection for each analyte is the number of valid samples with concentrations above the analytical detection limit across all rounds of sampling divided by the total number of samples submitted for laboratory analysis that had valid results. This applies to detection frequencies that were calculated for Reaches 1-6 and on a reach-by-reach basis.

3. Calculate RBSLs for each non-lead COI with the RSL Calculator using the residential scenario with exposure parameters derived from the CCT Tribal Survey (Table 3-3). Compare maximum detected concentrations for each non-lead COI to RBSLs.
4. Remove any COI whose maximum detected concentration is less than the RBSL for surface water.

The RSL Calculator (with Site-specific exposure parameters) was used to calculate the RBSL for incidental ingestion and dermal contact with surface water for adult and child residents using Site-specific exposure parameters. The RBSL used in the COI screen was the lower of the cancer or non-cancer RBSL for that substance. The surface water COI screen is shown in Table 3-4.[53] If the maximum detected concentration of a COI with a detection frequency >5% exceeded the RBSL, that COI was identified as a COPC. Surface water COPCs that have toxicity values and had measured concentrations that exceeded RBSLs and were evaluated quantitatively in this HHRA are:

- Aluminum
- Antimony
- Arsenic
- Barium
- Beryllium
- Chromium
- Cobalt
- Iron
- Lead
- Manganese
- Thallium
- Uranium
- Vanadium

3.2.4 Beach Sediment and Soil COI Screening

Sediment is a primary medium at the Site with potential contamination from COIs in solid materials (e.g., slag) discharged to the river. Public beach sediment (and beach soil) data were collected from 15 beaches in 2005,[54] 7 beaches in 2009 and 2010, 26 beaches in 2011 (TAI, 2009b, 2010b, 2010c, 2013b), and 2 beaches in 2015 (TAI, 2016a); some beaches were sampled more than once. These data are summarized in EPA (2006b, 2006c, and 2006d) and TAI (2014a, 2016a, and 2017b). Sediment data were also collected from beaches on residential properties in 2014 and 2016 (SRC, 2014a; TAI, 2016b) and are summarized in CH2MHill (2016a) and TAI

[53] COIs that were never detected in surface water are not listed in Table 3-4.
[54] http://www.ucr-rifs.com/assets/Docs/EPA-Beach-Screening-Results/Draft-UCR-Beach-Screen-RA-082806.pdf.

(2017a). Finally, relict floodplain data were collected as part of the 2014 upland soil study as described in TAI (2015a); in this risk assessment, these samples were evaluated as soil samples.

Sediment and beach soil data were screened for COPCs by data set as follows: 1) public beach surface sediment (all beaches sampled from 2005 to 2015)[55], 2) public beach surface soil (Bossburg Flat Beach UDUs), 3) public beach subsurface sediment (all beaches sampled from 2009 to 2015), 4) public subsurface soil (Bossburg Flat Beach UDUs), 5) residential beach surface sediment, and 6) relict floodplain surface soil. Each of those screens is described separately below.

3.2.4.1 Public Beach Surface Sediment

The following approach was used to select COPCs for public beach surface sediment:

1. Calculate the frequency of detection of each COI in surface sediment on both a Site-wide and reach-by-reach basis; retain COIs that are detected at a frequency of ≥5% on a Site-wide or reach-by-reach basis.[56]
2. Remove essential human nutrients that have no toxicity values (i.e., calcium, magnesium, potassium and sodium).
3. Calculate RBSLs for each non-lead COI with the RSL Calculator using the Site-specific exposure parameters for the outdoor worker scenario for public beaches (Table 3-3). Compare maximum detected concentrations for each non-lead COI to public beach RBSLs.
4. Remove any COI whose maximum detected concentration in public beach surface sediment is less than the RBSL.

[55] For public beach sediment, data from 2005 bulk samples (0-15 cm) were used for beaches that were not sampled in 2009, 2010, or 2011. Also, 2005 dioxin/furan results were used for beaches that were resampled in 2009-2011 but not analyzed for dioxins/furans. During the 2009-2011 sampling event, core samples were collected, sieved to <2 mm, and analyzed for dioxins/furans, metals, PAHs, PBDEs, PCBs, pesticides/herbicides, and SVOCs. Section 2.7.2.2 above details methods for handling PCB data. Results from the 0-15 cm depth interval were used. Approximately five surface composite samples were also collected from each beach and analyzed for metals in the <2 mm grain size fraction (GSF). One composite sample from each beach also had additional metals analysis done for the <63, 63-125, and 125-250 µm GSFs so that results in the <250 µm GSF could be calculated. The calculated <250 µm results were flagged as detected if the <2 mm GSF sample was detected. Some samples were reanalyzed for metals concentrations in the <250 µm GSF in 2013. For these reanalyzed samples, the 2013 measured <250 µm result was used in lieu of the original calculated <250 µm result. The 2009-2011 metal results from core samples were not used, as the <250 µm results from the surface composite samples were preferred. In 2015, samples were collected from 10 SDUs located at the Bossburg Flat and Evans Campground beaches and analyzed for metals. Results from the composite surface samples (0-15 cm) that were sieved to <250 µm were included in the screen.

[56] The frequency of detection for each organic analyte is the number of valid samples with concentrations above the analytical detection limit across all years of sampling divided by the total number of organic samples in the data set that had valid results. The frequency of detection for each inorganic analyte is based on the number of valid samples with concentrations above the analytical detection limit from 2009-2015 plus the number of valid samples with concentrations above the analytical detection limit from 2005 divided by the total number of inorganic samples in the data set that had valid results. This applies to detection frequencies that were calculated both for Reaches 1-6 and reach-by-reach.

The RSL Calculator (with Site-specific exposure parameters as described above) was used to calculate the RBSL for incidental ingestion and dermal contact with public beach surface sediment. The RBSL used in the COI screen was the lower of the cancer or non-cancer RBSL. The public beach surface sediment COI screen is shown in Table 3-5.[57] Public beach surface sediment COPCs that have toxicity values and had measured concentrations that exceeded RBSLs and were evaluated quantitatively are:

- Antimony
- Arsenic
- Chromium
- Iron
- Lead

3.2.4.2 Public Beach Surface Soil

The following approach was used to select COPCs for public beach surface soil:

1. Calculate the Site-wide frequency of detection of each COI in surface soil; retain COIs that are detected at a frequency of ≥5%.[58]
2. Remove essential human nutrients that have no toxicity values (i.e., calcium, magnesium, potassium and sodium).
3. Calculate RBSLs for each non-lead COI with the RSL Calculator using the Site-specific exposure parameters for the outdoor worker scenario for public beaches (Table 3-3). Compare maximum detected concentrations for each non-lead COI to public beach RBSLs.
4. Remove any COI whose maximum detected concentration in public beach surface soil is less than the RBSL.

The RSL Calculator (with Site-specific exposure parameters as described above) was used to calculate the RBSL for incidental ingestion and dermal contact with public beach surface soil. The RBSL used in the COI screen was the lower of the cancer or non-cancer RBSL. The public beach surface sediment COI screen is shown in Table 3-6. Public beach surface soil COPCs that have toxicity values and had measured concentrations that exceeded RBSLs and were evaluated quantitatively are:

- Arsenic
- Chromium
- Lead

[57] COIs that were never detected in public beach surface sediment are not listed in Table 3-5.

[58] For public beach soil, the frequency of detection for each inorganic analyte is based on the number of valid samples with concentrations above the analytical detection limit divided by the total number of inorganic samples in the data set that had valid results.

3.2.4.3 Public Beach Subsurface Sediment

Outdoor workers may be exposed to subsurface sediment at public beaches during occasional occupational activities (i.e., digging holes, trenches, footings, etc.; Figure 3-1). Subsurface sediment data collected in 2009-2011 (<2 mm size fraction) and 2015 (<250 µm size fraction) were used to conduct an additional COI screen for outdoor workers.

The following approach was used to select COPCs for public beach subsurface sediment:

1. Calculate the frequency of detection of each COI in subsurface sediment on both a Site-wide and reach-by-reach basis; retain COIs that are detected at a frequency of ≥5% on a Site-wide or reach-by-reach basis.[59]
2. Remove essential human nutrients that have no toxicity values (i.e., calcium, magnesium, potassium and sodium).
3. Calculate RBSLs for each non-lead COI with the RSL Calculator using the Site-specific exposure parameters for the outdoor worker scenario for public beaches (Table 3-3). Compare maximum detected concentrations for each non-lead COI to public beach RBSLs.
4. Remove any COI whose maximum detected concentration in public beach subsurface sediment is less than the RBSL.

The RSL Calculator (with Site-specific exposure parameters as described above) was used to calculate the RBSL for incidental ingestion and dermal contact with public beach subsurface sediment. The RBSL used in the COI screen was the lower of the cancer or non-cancer RBSL. The public beach subsurface sediment COI screen is shown in Table 3-7.[60] Public beach subsurface sediment COPCs that have toxicity values and had measured concentrations that exceeded RBSLs and were evaluated quantitatively are:

- Antimony
- Arsenic
- Chromium
- Cobalt
- Iron

[59] During the 2009-2011 sampling event, core samples (0-15, 15-45, and 45-75 cm) were collected, sieved to <2 mm, and analyzed for dioxins/furans, metals, PAHs, PBDEs, PCBs, pesticides/herbicides, and SVOCs. Results from the 0-15 and 15-45 cm depth intervals were used in the subsurface sediment screen. In 2015, samples were collected from 10 SDUs located at the Bossburg Flat and Evans Campground beaches and analyzed for metals. Results from core samples (0-15, 15-30, and 30-45 cm) that were sieved to <250 µm were used in the subsurface sediment screen. For each core, the maximum concentration across depth intervals (above 45 cm) was used as the result for that core in the subsurface sediment screen. Only one result per core, per analyte was used in the screen. The frequency of detection for each analyte is the number of cores with valid result concentrations above the analytical detection limit across all years of sampling divided by the total number of cores in the data set that had valid results. This applies to detection frequencies that were calculated for both Reaches 1-6 and reach-by-reach.

[60] COIs that were never detected in public beach subsurface sediment are not listed in Table 3-7.

- Lead
- Manganese

3.2.4.4 Public Beach Subsurface Soil

Outdoor workers may also be exposed to subsurface soil at public beaches during occasional occupational activities (i.e., digging holes, trenches, footings, etc.). Subsurface soil data collected from Bossburg Flat Beach UDUs in 2015 were used to conduct an additional COI screen for outdoor workers.

The following approach was used to select COPCs for public beach subsurface soil:

1. Calculate the Site-wide frequency of detection of each COI in subsurface soil; retain COIs that are detected at a frequency of ≥5%.[61]
2. Remove essential human nutrients that have no toxicity values (i.e., calcium, magnesium, potassium and sodium).
3. Calculate RBSLs for each non-lead COI with the RSL Calculator using the Site-specific exposure parameters for the outdoor worker scenario for public beaches (Table 3-3). Compare maximum detected concentrations for each non-lead COI to public beach RBSLs.
4. Remove any COI whose maximum detected concentration in public beach subsurface soil is less than the RBSL.

The RSL Calculator (with Site-specific exposure parameters as described above) was used to calculate the RBSL for incidental ingestion and dermal contact with public beach subsurface soil. The RBSL used in the COI screen was the lower of the cancer or non-cancer RBSL. The public beach subsurface soil COI screen is shown in Table 3-8. Public beach subsurface soil COPCs that have toxicity values and had measured concentrations that exceeded RBSLs and were evaluated quantitatively are:

- Arsenic
- Chromium
- Lead

[61] During the 2015 sampling event, core samples (0-15 and 15-45 cm) that were sieved to <250 µm were used in the subsurface soil screen. For each core, the maximum concentration across depth intervals (above 45 cm) was used as the result for that core in the subsurface soil screen. Only one result per core, per analyte was used in the screen. The frequency of detection for each analyte is the number of cores with valid result concentrations above the analytical detection limit divided by the total number of cores in the data set that had valid results.

3.2.4.5 Residential Beach Surface Sediment

The following approach was used to select COPCs for residential beach sediment:

1. Calculate the frequency of detection of each COI in residential beach sediment; retain COIs that are detected at a frequency of ≥5%.[62]
2. Remove essential human nutrients that have no toxicity values (i.e., calcium, magnesium, potassium and sodium).
3. Calculate RBSLs for each non-lead COI with the RSL Calculator using the Site-specific exposure parameters for the resident child scenario (Table 3-3). Compare maximum detected concentrations for each non-lead COI to RBSLs.
4. Remove any COI whose maximum detected concentration in residential beach sediment is less than the RBSL.

The RSL Calculator (with Site-specific exposure parameters) was used to calculate the RBSL for incidental ingestion and dermal contact with sediment. The RBSL used in the COI screen was the lower of the cancer or non-cancer RBSL. The residential beach surface sediment COI screen is shown in Table 3-9. Residential beach sediment COPCs that have toxicity values and had measured concentrations that exceeded RBSLs and were evaluated quantitatively are:

- Antimony
- Arsenic
- Chromium
- Cobalt
- Iron
- Lead
- Manganese
- Thallium

3.2.4.6 Relict Floodplain Surface Soil

The following approach was used to select COPCs for surface soil in relict floodplains:

1. Calculate the frequency of detection of each COI in relict floodplain surface soil; retain COIs that are detected at a frequency of ≥5%.[63]

[62] Residential beach samples were collected from within the 0-15 cm depth interval at 22 DUs (including one CCT tribal allotment beach). Samples were sieved to <250 µm and were analyzed for metals. The frequency of detection for each analyte is the number of valid samples with concentrations above the analytical detection limit divided by the total number of samples submitted for laboratory analysis that had valid results.

[63] Relict floodplain samples were collected from 0-7.5 cm depth interval at 16 RFDAs. Samples were sieved to <149 µm and <2 mm and were analyzed for metals. Only the <149 µm results were used in the screen. The frequency of detection for each analyte is the number of valid samples with concentrations above the analytical detection limit divided by the total number of samples submitted for laboratory analysis that had valid results.

2. Remove essential human nutrients that have no toxicity values (i.e., calcium, magnesium, potassium and sodium).
3. Calculate RBSLs for each non-lead COI with the RSL Calculator using the residential scenario with exposure parameters for the recreational visitor (Table 3-3). Compare maximum detected concentrations for each non-lead COI to relict floodplain RBSLs.
4. Remove any COI whose maximum detected concentration in relict floodplain surface soil is less than the RBSL.

The RSL Calculator (with Site-specific exposure parameters as described above) was used to calculate the RBSL for incidental ingestion and dermal contact with relict floodplain surface soil. For adult and child residents those Site-specific exposure parameters were used in the Calculator. The RBSL used in the COI screen was the lower of the cancer or non-cancer RBSL. The relict floodplain surface soil COI screen is shown in Table 3-10. Relict floodplain surface soil COPCs that have toxicity values and had measured concentrations that exceeded RBSLs and were evaluated quantitatively are:

- Arsenic
- Chromium
- Lead

3.2.5 Outdoor Air COI Screening

Outdoor air is a primary medium at the Site, potentially receiving COIs from current mining and smelting operations and ambient air (Figure 3-1). Outdoor air data collected from the Sheep Creek monitoring station from January 2002 through February 2009 (CH2MHill, 2015)[64] were used to screen COIs and identify COPCs using the following approach:

1. Calculate the frequency of detection of each COI in outdoor air; retain COIs that are detected at a frequency of ≥5%.[65]
2. Calculate RBSLs for each non-lead COI with the RSL Calculator using the residential scenario with exposure parameters derived from the CCT Tribal Survey (Table 3-3). Compare maximum detected concentrations for non-lead COIs to RBSLs.
3. Remove any COI whose maximum detected concentration is less than the RBSL for outdoor air.

The RSL Calculator (with Site-specific exposure parameters as described above) was used to calculate the RBSL for inhalation of outdoor air. The RBSL used in the COI screen was the lower of the cancer or non-cancer RBSL. The outdoor air COI screen is shown in Table 3-11.

[64] ECY has predicted average air concentrations near Northport for February 2009 through December 2014 using a regression model (ECY, 2017a, 2017b). These predicted data were not used in the COI screen, since they do not differ substantially from the measured data.

[65] For outdoor air, the frequency of detection for each analyte is the number of valid samples with concentrations above the analytical detection limit across all rounds of sampling divided by the total number of samples submitted for laboratory analysis that had valid results across all years.

Outdoor air COPCs that have toxicity values and had measured concentrations that exceeded RBSLs and were evaluated quantitatively are:

- Arsenic
- Cadmium
- Lead

3.2.6 COI Screening for Secondary and Tertiary Media

Secondary and tertiary media at the Site include soil, fish and macroinvertebrates, terrestrial (wild and cultivated) and aquatic plants, sweat lodge air, indoor dust and air, birds, mammals, reptiles and amphibians (Figure 3-1). Some of these media have been sampled and analyzed for COI concentrations. These include soil (sampled in 2014 and 2016), cultural (wild) terrestrial and aquatic plants (sampled in 2018), and fish and macroinvertebrates (sampled in 2005, 2009, 2016, and 2018). COI screening for crayfish, mussels, amphibians/reptiles, waterfowl, plants, upland birds, and wild game (mammals) is detailed in Appendix 1. While sweat lodge air is a secondary medium at the Site, it was not quantitatively evaluated as an exposure pathway separate from the incidental ingestion of UCR surface water (Appendix 3). Methods for screening COIs for secondary and tertiary media are described below.

3.2.6.1 Soil COI Screening

Soil is a secondary medium at the Site, receiving input from airborne contaminants that deposit on surface soil (Figure 3-1). Surface soil data were collected in 3 sampling efforts in 2014 through 2016 (TAI, 2014b, 2016b; SRC, 2014a, HDR et al., 2015a and 2015b). Data from these soil studies have been summarized in TAI (2015a), CH2MHill (2016a), TAI (2016a), and TAI (2017a). The TAI (2015a) soil data ("Upland Soil Study data") that were used for the COI screen were collected from 142 large, undeveloped ADAs near the UCR. The 2014 and 2016 soil data described in CH2MHill (2016a) and TAI (2017a) ("Residential Soil Study Data") were collected from residential properties (including CCT tribal allotments). The 28 residential soil DUs subject to TCRA/VRA removal actions that occurred from 2015 through 2018 were included in the COI screen using the maximum backfill concentration of each COI, rather than the initial concentration sampled in 2014 or 2016 (Table 3-12).[66] COIs were screened separately for the Residential, Upland, and Bossburg Flat Soil Study data.

COPCs for soil were selected as follows:

1. Calculate the frequency of detection of each COI in soil; retain COIs that are detected at a frequency of ≥5%. This was done separately for the Residential Soil Study data and the Upland Soil Study data.[67]

[66] This DU count includes 2 DUs (203-O3 and 203-O4) that were on one of the properties sampled in the 2016 residential soil study, but that were sampled in 2018 rather than in 2016. Information concerning TCRA/VRA removals is current through June 20, 2019.

[67] For upland soil, while results for both the <149 µm and <2 mm GSFs were reported, only results from the <149 µm GSF were used here. The frequency of detection for each analyte is the number of valid samples with

2. Remove essential human nutrients that have no toxicity values (i.e., calcium, magnesium, potassium and sodium).
3. Calculate RBSLs for each non-lead COI with the RSL Calculator using the Site-specific exposure parameters for the resident child scenario (Table 3-3). Compare maximum detected concentrations for each non-lead COI to RBSLs.
4. Remove any COI whose maximum detected concentration in Residential or Upland soil is less than the RBSL.

The RSL Calculator (with Site-specific exposure parameters) was used to calculate the RBSL for incidental ingestion and dermal contact with soil. The RBSL used in the COI screen was the lower of the cancer or non-cancer RBSL. The residential and upland soil COI screens are shown in Tables 3-13 and 3-14.

Residential soil COPCs that have toxicity values and had measured concentrations that exceeded RBSLs and were evaluated quantitatively are:

- Aluminum
- Antimony
- Arsenic
- Cadmium
- Chromium
- Cobalt
- Iron
- Lead
- Manganese
- Nickel
- Thallium
- Vanadium

Upland soil COPCs that have toxicity values and had measured concentrations that exceeded RBSLs and were evaluated quantitatively are:

- Aluminum
- Antimony

concentrations above the analytical detection limit divided by the total number of samples submitted for laboratory analysis that had valid results. For residential soil, the frequency of detection for each analyte is the number of valid samples with concentrations above the analytical detection limit divided by the total number of samples submitted for laboratory analysis that had valid results across both years.

- Arsenic
- Cadmium
- Chromium
- Cobalt
- Iron
- Lead
- Manganese
- Thallium
- Vanadium

3.2.6.2 Fish COI Screening

Fish are secondary media at the Site, contacting contaminants in surface water and sediment (Figure 3-1). Fish data collected in 2005 were available at the time the HHRA work plan (SRC, 2009) was written. To supplement those data, additional fish were collected in 2009 from six reaches of the UCR (TAI, 2013a), hatchery White Sturgeon were collected from four UCR reaches in 2016 (CH2MHill, 2016b), and Northern Pike were collected in 2018 from the area between Gifford and Northport, Washington (CH2MHill, 2018a). For the fish COI screen, data for fillets of fish larger than 30 cm from all sampling locations were compiled. This includes skin-on fillets of Rainbow Trout, Walleye, Burbot, Smallmouth Bass, kokanee, whitefish, and Largescale Sucker sampled in 2005 and 2009, skinless fillets of White Sturgeon sampled in 2016, and skinless fillets of Northern Pike sampled in 2018. These data were evaluated as follows:

1. Calculate the frequency of detection of each COI in fish tissue on a Site-wide, reach-by-reach, or species-by-species basis; retain COIs that are detected at a frequency of ≥5% on a Site-wide, reach-by-reach, or species-by-species basis.[68]
2. Remove essential human nutrients that have no toxicity values (i.e., calcium, magnesium, potassium and sodium).
3. Select RBSLs for each non-lead COI using the residential scenario with exposure parameters derived from CCT Tribal Survey (Table 3-3). Compare maximum detected concentrations for each non-lead COI to RBSLs.
4. Remove any COI whose maximum detected concentration is less than the RBSL.

The RSL Calculator (with Site-specific exposure parameters) was used to calculate the RBSL. For the adult resident, those Site-specific exposure parameters were used in the Calculator. The RBSL used in the COI screen was the lower of the cancer or non-cancer RBSL. The fish COI

[68] For fish, the frequency of detection for each analyte is the number of valid samples with concentrations above the analytical detection limit across all years of sampling and species of fish divided by the total number of valid samples submitted for laboratory analysis across all years of sampling and species of fish. This applies to detection frequencies that were calculated for Reaches 1-6, and on a reach-by-reach and species-by-species basis.

screen is shown in Table 3-15. Fish COPCs that have toxicity values and had measured concentrations that exceeded RBSLs and were evaluated quantitatively are:

- 4,4'-DDE
- Aluminum
- Antimony
- Inorganic arsenic[69]
- Bis(2-ethylhexyl)phthalate
- Chromium
- Dieldrin
- Heptachlor epoxide
- Hexachlorobenzene
- Lead
- Methylmercury[70]
- PBDE congener 47
- Selenium
- TEQ (dioxin-like PCBs and dioxins/furans)
- Thallium
- Total PCBs
- Zirconium

3.2.6.3 Sweat Lodge Air COI Screening

Sweat lodge air is a secondary medium at the Site that may be impacted by contaminants in surface water if they are vaporized or aerosolized when the water is poured over heated rocks in the sweat lodge (Figure 3-1). However, non-volatile metals will not vaporize at sweat lodge temperatures. Therefore, for the sweat lodge exposure scenario, intake is assumed to occur via ingestion of dissolved contaminants in surface water that is sprayed from contact with heated

[69] See Section 4.5 for discussion of evaluation of arsenic/inorganic arsenic in fish.

[70] Mercury was analyzed using EPA Method 1631e, which only measures total mercury. In fish, methylmercury and inorganic mercury are absorbed by the gut; methylmercury is then transferred to blood and distributed to other body tissues (Ribeiro et al., 1999), ultimately accumulating in skeletal muscle tissue. As a result, most mercury in the muscle of fish is methylmercury (Wiener et al., 2003). Chumchal et al. (2011) collected invertebrates and vertebrates from a freshwater lake, including six fish species, and analyzed for total mercury and methylmercury. The percentage of mercury in muscle tissue present as methylmercury ranged from 84 to 100%. As part of the EMAP sampling program, 10 species were collected from 14 estuaries in southern Florida (Kannan et al., 1998). Total mercury and methylmercury were measured in fish muscle tissue; on average, methylmercury contributed 83% of the total mercury tissue concentration (range 62-100%). Because the majority of mercury present in fish fillets is methylmercury, and the toxicity value is for methylmercury, mercury is referred to as "methylmercury" in this report.

rocks inside the sweat lodge. Relative to ingestion of UCR surface water during in-water activities, ingestion of dissolved COPCs in surface water spray in sweat lodges is a negligible contributor to risk and was not quantitatively evaluated in this HHRA (see Appendix 3).

3.2.6.4 Indoor Air COI Screening

In the absence of Site-specific data, outdoor air COPCs were the COPCs for indoor air. Outdoor/indoor air COPCs that have toxicity values and had measured concentrations that exceeded RBSLs are:

- Arsenic
- Cadmium
- Lead

3.2.6.5 Indoor Dust COI Screening

In the absence of Site-specific data, indoor dust EPCs for non-lead COPCs were derived from the outdoor residential soil concentration from each DU or upland soil concentration from each ADA using the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) default adjustment factor (mass fraction of soil in indoor dust [MSD]) of 0.7 (EPA, 1994b, 1994c).

COPCs identified for residential soil were the COPCs identified for indoor dust on residential DUs:

- Aluminum
- Antimony
- Arsenic
- Cadmium
- Chromium
- Cobalt
- Iron
- Lead
- Manganese
- Nickel
- Thallium
- Vanadium

COPCs identified for upland soil were the COPCs identified for indoor dust on upland ADAs:

- Aluminum

- Antimony
- Arsenic
- Cadmium
- Chromium
- Cobalt
- Iron
- Lead
- Manganese
- Thallium
- Vanadium

3.3 Exposure Point Concentrations (EPCs)

An EPC is an estimate of the concentration of a COPC in a site medium, averaged over the area to which a receptor is exposed (i.e., exposure area). EPA (1992) recommends that the 95% upper confidence limit of the arithmetic mean (95UCL) be used as the EPC for quantifying exposure and risk to individuals under both the RME and the CTE scenarios (EPA, 1992). The approach to calculate a 95UCL depends on the number of data points available, the shape of the data distribution, and the degree of censoring (i.e., samples below the detection limit) (EPA, 2002e). EPA has developed ProUCL software (version 5.1) to estimate 95UCL values (EPA, 2015d). ProUCL calculates 95UCLs for a data set using several different strategies and recommends a 95UCL based on the properties of the data set. A minimum of five discrete samples or three samples collected using IC sampling and two distinct detected values are required to calculate a 95UCL in ProUCL. When ProUCL provided more than one "recommended" 95UCL to use (e.g., Chebyshev or Bootstrap), the higher recommended value was selected as the EPC. Appendix 12 details the approach used in this HHRA to derive the EPC for an exposure area when only one IC soil sample was available.

3.3.1 EPCs for Lead

Both the IEUBK model and the Adult Lead Methodology (ALM) require the EPC for lead to be an arithmetic mean value (see Section 3.4 for more information). For residential soil (and residential beach sediment), upland soil, relict floodplains, and public beach sediment collected using IC sampling, the EPC for lead was the arithmetic mean across triplicate samples (when available) or the single sample result for each DU, ADA, SDU, UDU, or RFDA (all results were detects for lead). For media where there were non-detect values for lead (e.g., surface water and outdoor air), the EPC was the mean estimated using the KM method. Helsel (2005) recommends using the KM method for estimating the mean when the percentage of non-detect values is less than 50%, regardless of sample size. For fish, the lead EPC was the arithmetic mean, with non-detects (ND) = ½ detection limit.[71]

[71] The estimate of the mean by the ProUCL KM method is within 0.01 mg/kg of the arithmetic mean with non-detects = ½ the detection limit.

The IEUBK model requires the user to input a soil concentration for the child's residence regardless of the medium being assessed. For the residential beach sediment evaluation, residential soil lead EPCs were calculated for the house DU(s) (or the nearest appropriate DU or DUs) on the property. In some exposure scenarios, a house DU soil lead concentration was not available to input into the model (for example, public beaches, relict floodplains, UDUs, and residential beaches on properties without a house DU). If no house DU was sampled on the property, and for public beaches and relict floodplains, the soil lead concentration for the child's residence was calculated as follows. Table 3-16 illustrates the distribution of mean soil lead concentrations adjusted for bioavailability from the 2014 and 2016 residential soil studies. To derive the concentrations shown in Table 3-16, each DU was assigned to either the 2014 or 2016 residential soil study area using the approach described in Appendix 11 (i.e., "2014" is defined as being within the 2014 study area boundary, regardless of whether it was sampled in 2014 or 2016, and "2016" is defined as being outside the 2014 study area boundary). Additionally, the 28 DUs subject to TCRA/VRA removal actions were included using the maximum concentration in backfill, which was 12 ppm lead for DUs subject to removal actions in 2015 and 19 ppm lead for DUs removed in 2017-2018. Those concentrations were then adjusted for bioavailability as described in Appendix 11. For evaluation of lead exposure in this HHRA where a house DU soil concentration was not available, a surrogate value was selected as described in Appendix 13 as the assumed soil lead concentration for the residence to be used in the IEUBK model.

3.3.2 EPCs for Non-Lead COPCs

The 95UCL was selected as the EPC for all non-lead COPCs. For all media except residential and upland soil, 95UCLs were calculated using ProUCL when data requirements were met. When ProUCL provided more than one "recommended" 95UCL to use (e.g., Chebyshev or Bootstrap), the higher recommended value was selected as the EPC. For residential and upland soil exposure areas where 3 IC replicate samples were collected, a 95UCL based on the t-statistic was calculated (ProUCL was not used because of the size of the data sets). For exposure areas where only a single IC sample was collected, the IC sample concentration was multiplied by a confidence factor (CF) that was based on an analysis of 95UCL/mean ratios for DUs at which more than one IC sample was collected to generate an EPC (Appendix 12).

3.3.2.1 EPCs for Polychlorinated Dibenzodioxin (PCDD), Polychlorinated Furan (PCDF) and Dioxin-Like Polychlorinated Biphenyl (PCB) Congeners

PCDD, PCDF, and dioxin-like-PCB congeners are all believed to act by the same toxic mechanism as TCDD. Data for the PCDD, PCDF, and dioxin-like-PCB congeners were converted to a TCDD TEQ as described in Section 2.7.2. EPC values were computed for TEQs using half of the detection limit for non-detect congeners (ND = ½ detection limit).

3.3.3 Surface Water EPCs

Surface water EPCs, shown in Table 3-17, were calculated for the UCR (inclusive of Reaches 1-6) as discussed in Section 3.1.1 using data from unfiltered (total), disturbed surface water samples. In this HHRA, only samples designated as unfiltered were considered useable surface water data for the following reasons. Human exposures to surface water may occur under a variety of exposure scenarios. In most instances, on-Site exposures are expected to occur primarily in shallower water near beach areas (e.g., recreational visitors during wading/playing

activities, outdoor workers during occupational activities) which may be directly influenced by sediment disturbances resulting from these activities. Recreational visitors may also be exposed to surface water further away from beach areas (e.g., during swimming). The unfiltered disturbed surface water samples collected in shallow water near beach areas were used to evaluate exposure during all recreational activities in this HHRA, as this is what we would expect people to ingest. The EPCs from the disturbed shallow samples are likely to overestimate exposure from swimming in deeper water where disturbance of the underlying sediment would not be expected. The EPCs calculated for unfiltered, undisturbed surface water samples were always lower than for unfiltered, disturbed samples.

The unfiltered disturbed surface water samples were used to evaluate exposure to surface water during recreational or occupational activities. A site-overall EPC (sample size [n] = 42) was calculated for COPCs to evaluate exposure to surface water during these activities. For arsenic, two EPCs were calculated: RBA-adjusted arsenic concentration data were used to calculate EPCs to evaluate surface water ingestion exposure (adjusted using methods described in Section 4.6; EPA [2020]); and unadjusted arsenic data were used to calculate EPCs to evaluate dermal exposure to surface water. For lead, the EPC was calculated using the RBA-adjusted lead concentration (Section 4.6) (EPA, 2020).

3.3.4 Public Beach Surface and Subsurface Sediment EPCs

For beaches sampled from 2009-2011, approximately 5 surface sediment (0-15 cm) composite samples were collected from each beach and analyzed for metals in the <2 mm grain size fraction (GSF). One composite sample from each beach also had additional metals analysis done for the <63, 63-125, and 125-250 µm GSFs that were used to derive a <250 µm result for every sample collected from each beach. The calculated <250 µm results were flagged as detected if the <2 mm GSF sample was detected. Some archived samples from the 2009-2011 event were sieved to <250 µm and reanalyzed for arsenic and lead in 2013. For these reanalyzed samples, the 2013 measured <250 µm result was used in lieu of the original calculated <250 µm result (note that only lead was analyzed for IVBA in 2013, so arsenic results from 2013 were adjusted for RBA using the calculated <250 µm results from the 2009-2011 event). A 95UCL was calculated for each beach using the data set that combined the calculated <250 µm results from 2009-2011 and the measured <250 µm results from 2013 and used as the EPC for each beach in non-lead analyses. For lead analyses, the average RBA-adjusted concentration was calculated for each beach using the data set that combined the calculated <250 µm results from 2009-2011 and the measured <250 µm results from 2013.

For beaches sampled in 2015, surface sediment EPCs were calculated using the Incremental Composite Sampling (ICS) data from the 0 to 15 cm depth interval (<250 µm results). ProUCL was used to generate a 95UCL for beach SDUs that had 3 IC samples. The EPCs for SDUs with one IC sample were estimated by multiplying the single result by a CF (see Appendix 12) to generate an EPC. Public beach surface sediment EPCs, calculated from surface sediment samples, are shown in Table 3-18.

EPCs for subsurface sediment at Bossburg Flat and Evans Campground Beach (sampled in 2015) were generated in ProUCL using data from core samples that were collected at three depth

intervals (0-15, 15-30, and 30-45 cm) and sieved to <250 µm (Table 3-19).[72] For each core, a single subsurface (0-45 cm) sample result was derived by averaging concentrations across the three depth intervals for each COPC.

For arsenic, two EPCs were calculated: RBA-adjusted arsenic concentration data were used to calculate EPCs to evaluate incidental sediment ingestion exposure (using methods described in Section 4.6; EPA [2020]); and unadjusted arsenic data were used to calculate EPCs to evaluate dermal exposure to sediment. For lead, the EPC was calculated using the RBA-adjusted lead concentration (Section 4.6) (EPA, 2020).

3.3.5 Residential Beach Sediment EPCs

EPCs were calculated using data from the IC surface sediment samples for each residential beach DU. Three IC samples were collected from all residential beach DUs in 2014, and from two of the residential beach DUs sampled in 2016. A 95UCL was calculated for DUs that had 3 IC samples (see Table 3-20). The EPCs for DUs with one IC sample were estimated by multiplying the single result by a CF (see Appendix 12) to generate an EPC.

As described in Section 3.3.4, two EPCs were calculated for arsenic; one to estimate risk via dermal exposure and one to estimate risk via incidental ingestion. EPCs for the residential beach DUs are presented in Table 3-20.

3.3.6 Relict Floodplain Soil EPCs

Three to nine RFDAs were sampled on each relict floodplain, as described in Section 2.6.2.5. EPCs for relict floodplains were calculated for each RFDA. ProUCL was used to generate a 95UCL for RFDAs that had 3 IC samples. The EPCs for RFDAs with one IC sample were estimated by multiplying the single result by a CF (see Appendix 12) to generate an EPC.

As described in Section 3.3.4, two EPCs were calculated for arsenic; one to estimate risk via dermal exposure and one to estimate risk via incidental ingestion. EPCs for the RFDAs are presented in Table 3-21.

3.3.7 Bossburg Flat Beach Surface and Subsurface Soil EPCs

Beach surface soil EPCs were calculated using the ICS data from the 0-15 cm depth interval (<149 µm results). ProUCL was used to generate a 95UCL for the one UDU that had three IC samples collected (UDU-04). The EPCs for all other UDUs with only one IC sample were estimated by multiplying the single result by a CF (see Appendix 12) to generate an EPC. Bossburg Flat beach surface soil EPCs, calculated from surface soil samples, are shown in Table 3-22.

EPCs for subsurface soil at Bossburg UDUs were generated in ProUCL using data from core samples that were collected at three depth intervals (0-15, 15-30, and 30-45 cm) and sieved to <149 µm (Table 3-23). For each core, a single subsurface (0-45 cm) sample result was derived by averaging concentrations across the three depth intervals for each COPC.

[72] EPCs were calculated for subsurface sediment at Bossburg Flat and Evans Campground Beach only. EPCs were not calculated for subsurface sediment for any of the other public beaches due to a lack of subsurface sample data sieved to the appropriate grain size for HHRA.

For arsenic, two EPCs were estimated: RBA-adjusted arsenic concentration data were used to calculate EPCs to evaluate incidental soil ingestion exposure (using methods described in Section 4.6; EPA [2020]); and unadjusted arsenic data were used to estimate EPCs to evaluate dermal exposure to soil. For lead, the EPC was calculated using the RBA-adjusted lead concentration (Section 4.6) (EPA, 2020).

3.3.8 Outdoor Air EPCs

Outdoor air data collected at an air monitoring station at Sheep Creek near Northport from 2002 through 2009 were used to calculate the outdoor air EPCs. The outdoor air EPCs were calculated on a Site-wide basis. The 95UCL was selected as the EPC for all non-lead COPCs. The EPCs for outdoor air are shown in Table 3-24.

3.3.9 Indoor Air EPCs

Because indoor air concentrations were not measured, the indoor air EPCs were set as equal to outdoor air EPCs and are shown in Table 3-24.

3.3.10 Residential and Upland Soil and Indoor Dust EPCs

Site-specific soil data collected by EPA in 2014 (CH2MHill, 2016a) and by TAI in 2014 and 2016 (TAI, 2015a, 2017a) were used to calculate the EPCs for residential and upland soil as well as indoor dust, as discussed in greater detail below. Summary statistics for the surface soil COPCs are shown in Table 3-25.

The ingestion rate for indoor dust and outdoor soil was partitioned according to the IEUBK model for children younger than 6 years old (55% indoor dust:45% outdoor soil; EPA, 1994b), and according to the *Exposure Factors Handbook* (EPA, 2017b) for older age groups. These partitions were applied to the outdoor residential and upland soil EPCs and the calculated indoor dust EPC (see Section 3.3.10.4) to generate a combined soil/dust EPC that was used to perform risk calculations for incidental ingestion. These combined soil/dust ingestion EPCs are shown in Tables 3-26 through 3-28.

3.3.10.1 Residential Soil for Non-Beach DUs

EPCs for residential soil, for all DUs except residential beaches and driplines, were calculated using data from the IC surface soil samples on a DU by DU basis. Triplicate IC samples were collected from most residential soil DUs in 2014, and from approximately 30% of the residential DUs sampled in 2016. For lead analyses, the EPC was the arithmetic mean across triplicate samples (when available) or the single IC sample result for each DU. For non-lead analyses, the EPC was the 95UCL calculated for triplicate IC samples (when available) or the single sample result multiplied by a CF (see Appendix 12) for each DU (see Table 3-25). For residential soil DUs subject to either a complete or partial TCRA or VRA (Table 2-3), the maximum measured COPC concentration in backfill used during that removal was used as the EPC for each COPC in risk calculations. DU 172-01 was excluded from risk calculations because of its former use as a small ore mill or ore stockpiling site (Roland, 2019).

For arsenic, one EPC was calculated using the RBA-adjusted arsenic concentration (Section 4.6; EPA [2020]), to evaluate incidental soil ingestion exposure. A separate EPC was calculated using the measured arsenic concentration to evaluate dermal exposure to soil. For lead, the EPC was calculated using the RBA-adjusted lead concentration (Section 4.6) to evaluate incidental soil ingestion exposure (EPA, 2020). Dermal exposure to soil was not evaluated for lead (Section 3.1.3.4). EPCs for the residential soil DUs are presented in Table 3-26.

3.3.10.2 Residential Soil/Sediment EPC for Resident with Beach DUs

For each residential beach DU, a residential soil concentration needed to be assigned to the resident using that beach. If a house DU(s) (or the nearest appropriate DU or DUs) was located on the property with a residential beach DU, analytical results for the "house" DU(s) were used. In some cases, a house DU was not sampled on the same property as the residential beach. In those cases, the residential soil EPCs for each non-lead COPC were calculated using the surrogate DUs listed in Table 3-27. The residential soil concentration was generated by averaging the EPC (95UCL or Result × CF) soil concentration for each residential soil DU paired with a beach. For 2 residential beach DUs, no other DUs were sampled on the property. In those cases, the mean concentration of each COPC for the Site was used as the soil EPC, similar to the description for the mean lead concentration for the Site described in Section 3.3.1 above. These concentrations are shown in Table 3-27.

For arsenic, one EPC was calculated using the RBA-adjusted arsenic concentrations (Section 4.6), to evaluate incidental soil ingestion exposure (EPA, 2020). A separate EPC was calculated using the measured arsenic concentrations to evaluate dermal exposure to soil.

3.3.10.3 Upland Soil

Using IC sampling, soil samples were collected from ADAs in upland areas likely impacted by aerial deposition of smelter particulates. EPCs for upland soil were calculated on an ADA by ADA basis. Triplicate IC samples were collected from approximately 10% of the ADAs sampled. For lead analyses, the EPC was the arithmetic mean across triplicate samples (when available) or the single sample result for each ADA. For non-lead analyses, the EPC was the 95UCL calculated for triplicate IC samples (when available) or the single sample result multiplied by a CF (see Appendix 12) for each ADA.

As was done for residential soil, one EPC was calculated using the RBA-adjusted arsenic concentration to evaluate incidental soil ingestion exposure. One EPC was calculated using the measured arsenic concentration to evaluate dermal exposure to soil. For lead, the EPC was calculated using the RBA-adjusted lead concentration (EPA, 2020). EPCs for the upland soil ADAs are presented in Table 3-28.

3.3.11 Soil Indoor Dust EPCs

Residential exposure to COPCs in soil includes indoor dust exposure for all DUs and ADAs, regardless of whether a house is present. In the absence of empirical data, indoor dust EPCs at each DU in the 2014 and 2016 Residential Soil Studies and ADA in the 2014 Upland Soil Study were derived from the outdoor soil COPC concentration at that DU or ADA by applying an adjustment factor of 0.7, such that Indoor Dust EPC = Outdoor Soil EPC × 0.7. The value 0.7 is the MSD term in the IEUBK model (EPA, 1994b), which represents the mass fraction

contribution of outdoor soil to indoor dust. The concentration in indoor dust was assumed to be 70% of the concentration in outdoor soil (accounting for a 30% contribution of non-soil sources to the indoor dust mass). This calculation also assumed that transport of soil-borne COPCs into the home is governed by transport of soil and, therefore, the 0.7 value can be applied to all COPCs.

3.3.12 Fish EPCs

The exposure area for estimating EPCs for fish consisted of the entire UCR (i.e., Reaches 1-6). EPCs were calculated for all COPCs in fish tissue utilizing the fillet data from all fish longer than 30 cm collected in UCR Reaches 1-6. EPCs were calculated by individual species. Fish tissue EPCs are shown by species in Table 3-29.

3.4 Evaluation of Exposures to Lead

The primary population of concern for risk from exposure to lead in this HHRA is children younger than age six. For the assessment of lead, EPA recommends the use of the IEUBK model to evaluate exposures of children to lead-contaminated media in a residential setting (EPA, 1994b, 1994c, 1998b; Vandenberg, 2020). The IEUBK model predicts the probability distribution of PbBs in a population of young children (12-72 month age range was used in this HHRA per EPA, 2017c) exposed to a user-specified set of environmental lead levels (EPA, 1994b, 1998b; Vandenberg, 2020). This model allows users to input data on the levels of lead in soil, dust, water, air, diet, and other (user-defined intake) for a specific location as well as data on the amounts of these media ingested or inhaled by a child living at that location. All of these inputs to the IEUBK model are central tendency point estimates (EPA, 1994b, 1994c). These point estimates are then integrated to calculate an estimate of the central tendency (the geometric mean) of the distribution of PbBs that might occur in a population of children exposed to the specified conditions. Assuming the distribution is lognormal and given (as input) an estimate of the variability in PbBs (this is specified by the geometric standard deviation [GSD]), the model calculates the expected distribution of PbBs for a population of similarly exposed children, as well as an estimate of the probability of PbB exceeding the target risk level specified by the user.

In addition to predicting PbBs and estimating the probability of PbB exceeding a target risk level (forward risk assessment), the IEUBK model can be used to calculate PRGs for an exposure unit (e.g., DUs or ADAs) for a target level of concern and risk probability (backward risk assessment). In the forward (risk calculation) mode the IEUBK model calculates the probability (P expressed as a percentile) that a hypothetical child or group of similarly exposed children will exceed a blood lead target for a user-defined set of exposures. In the forward mode, exposure information is entered in the IEUBK model and the resulting distribution of PbBs and the probability of exceeding a target PbB are compared with the risk benchmark selected (for example, no more than 5% probability of PbB exceeding the target PbB). In forward calculations, exposure pathways were evaluated against the selected risk benchmarks for 12-72 month-old children as described below.

EPA's Office of Research and Development (ORD) reviewed the health effects evidence for lead in the 2013 Integrated Science Assessment for Lead (ISA for Lead) and found that several studies have observed "clear evidence of cognitive function decrements (as measured by Full Scale intelligence quotient [IQ], academic performance, and executive function) in young

children (4-11 years old) with mean or group PbBs between 2 and 8 micrograms per deciliter (µg/dL) (measured at various lifestages and time periods)." In addition, the National Toxicology Program's (NTP, 2012) Monograph on Health Effects of Low-Level Lead found sufficient evidence of delayed puberty, reduced post-natal growth, and decreased hearing for children at PbBs below 10 µg/dL and adverse effects on academic achievement, IQ, other cognitive measures, attention-related behaviors, and problem behaviors at PbBs below 5 µg/dL (NTP, 2012).

For this HHRA, the risk evaluation benchmarks selected for evaluation of lead risk were 3, 5, or 8 µg/dL. Elevated lead risk was defined as >5% probability of exceeding each of these target PbBs: >5% probability of exceeding 3 µg/dL (referred to as "P3"), >5% probability of exceeding 5 µg/dL (referred to as "P5"), or >5% probability of exceeding 8 µg/dL (referred to as "P8").

At the low end of the target risk range (2 µg/dL), the risk target goal (of limiting exposure to lead such that children would have estimated risk of no more than 5% probability of exceeding 2 µg/dL as determined by the IEUBK model) is exceeded by the current default exposures in the IEUBK model. Currently, assumed exposure to lead in other (not Site-related) media (drinking water, air, diet) results in a baseline PbB of approximately 2.3 µg/dL, which means that even if the soil lead concentrations were zero (0 ppm), use of the current default parameters in the IEUBK model will result in more than 5% probability that PbB will be >2 µg/dL. The estimated Site background soil lead concentration is around 35 ppm (Appendix 4). P3 was selected as a lead risk benchmark to evaluate the low end of the risk range of child PbBs associated with adverse health effects. For this HHRA, the results for P3, P5, and P8 were assessed quantitatively.

The IEUBK model (V1.1, build 11 with inputs shown in Table 3-30 below) was used to derive PRGs for residential soil lead exposures for each of these risk benchmarks as follows:

- Utilizing the IEUBK model, a 5% probability of exceeding a blood lead level of 3 µg/dL was associated with a concentration of approximately 50 ppm lead in soil.
- Utilizing the IEUBK model, a 5% probability of exceeding a blood lead level of 5 µg/dL was associated with a concentration of approximately 200 ppm lead in soil.
- Utilizing the IEUBK model, a 5% probability of exceeding a blood lead level of 8 µg/dL was associated with a concentration of approximately 400 ppm lead in soil.

The PRGs associated with each of these risk benchmarks is based on an assumed default RBA of 60% for lead. Site-specific IVBA information was used to derive RBA-adjusted lead concentrations, which allows direct comparison with these PRGs. At some locations, exposure of children to Site media is unlikely, while adult exposure may occur (e.g., exposure to deeper horizons of soil or sediment because of occupational activities). When adults are exposed to media where lead is a COPC (and there is no exposure to young children), the ALM is recommended (EPA, 2003b). The ALM is designed to assess non-residential (i.e., workplace or commercial) exposure to lead-contaminated soil by adults. The receptor evaluated in the ALM is the fetus (the most sensitive endpoint being the developing nervous system); risk estimates based on the ALM are considered to be protective of other adverse health effects in adults related to lead exposure.

This Site-wide HHRA calculated lead risk for the following receptors, exposure pathways, and media:

- Children (residents) who incidentally ingest soil at residential DUs and upland ADAs;
- Children (residents) who incidentally ingest beach sediment at residential beaches;
- Children (recreational visitors) who incidentally ingest beach sediment at public beaches;
- Children (recreational visitors) who incidentally ingest UCR surface water while swimming and wading in shallow water near public beaches during beach visits, and while boating or camping;
- Children (recreational visitors) who incidentally ingest soil near Bossburg Flat Beach;
- Children (recreational visitors) who incidentally ingest soil at relict floodplains;
- Children (recreational visitors and residents) who consume fish from the UCR;
- Adults (outdoor workers) who incidentally ingest subsurface beach sediment or subsurface beach soil while working at public beaches;
- Lead in air was included in the above exposures as appropriate. Lead in indoor air and indoor dust were based on measured concentrations of lead in outdoor air and outdoor soil.

In general, the IEUBK model predicts that lead intakes of at least 1 µg/day (at 30% absolute bioavailability [ABA]) are needed to decrease the soil PRG by approximately 20 ppm for a target risk goal of 5% exceeding a PbB of 5 µg/dL (P5). For example, the PRG decreases from 200 ppm to 180 ppm when lead intake increases 1 µg/day (i.e., the alternate exposure pathway in the IEUBK model). In this Site-wide HHRA, exposures that were predicted to contribute <1 µg Pb/day were classified as minimal.

3.4.1 Exposure Parameters in the Integrated Exposure Uptake Biokinetic Model (IEUBK)

The IEUBK model default parameter values were used when representative Site-specific information was not available. These defaults were modified to incorporate Site-specific information from the UCR Site when appropriate. Site-specific exposure parameters are described later in this section. Tables 3-30 and 3-31 present the input parameters used for evaluating lead exposures to children using the IEUBK model for selected pathways at the UCR Site. Table 3-30 shows IEUBK model values with defaults as recommended by EPA and the Site-specific information. Table 3-31 provides the values for the age-specific parameters for the IEUBK model.

3.4.2 Exposure Parameters in the Adult Lead Methodology (ALM)

The default variable values in the ALM (EPA, 2003b; updated to include National Health and Nutrition Examination Survey [NHANES] information as shown on the Technical Review Workgroup [TRW] Lead Committee website[73]) were used when representative Site-specific

[73] See https://www.epa.gov/superfund/lead-superfund-sites-software-and-users-manuals#update.

information was not available. Site-specific exposure parameters are described later in this section.

3.4.3 Site-Specific Information for Exposure Pathways

3.4.3.1 Current Child Resident Exposed to Lead in Residential Soil and Fish from the UCR

Exposure to lead in residential soil and fish harvested from the UCR was assessed on a DU by DU basis for current child residents using the IEUBK model. The exposure pathway for this medium was incidental ingestion of lead in soil and dust by current full-time resident children. To allow for potential future land use changes at residential properties, a residential exposure scenario was assumed to assess risk for all DUs located on residential properties, regardless of the currently reported land use. The EPCs for current residential soil lead concentration were the arithmetic mean values for the ICs of each DU sampled in the 2014 and 2016 residential soil studies if triplicate IC samples were collected, or the sample result for each DU where a single IC sample was collected (aside from CCT tribal allotments, driplines, residential beaches, and one DU that was previously used as a potential ore stockpile) (see Section 3.1.1.4). Generally, soil samples were collected from the surface where direct contact by young children is most frequent: house DUs were sampled at 0-2.5 cm depth interval and sieved to collect the <149 µm particle size fraction (CH2MHill, 2016a; TAI, 2017a). For non-house DUs, the EPCs for lead were based on sampling depths that vary depending on land use; this ranged from 0-2.5 cm interval for some DUs (e.g., children's play areas) to 0-30 cm interval for other DUs (e.g., gardens). The RBA for lead in the residential soil DUs was based on the results from IVBA assessments, as described in Section 4.6 (EPA, 2020). Dust lead concentration was calculated using the MSD result from the RBA-adjusted soil lead EPC (the MSD approach included the contribution from Site-specific outdoor air lead as described in Table 3-30). In addition to lead in current residential soil, resident children may be exposed to lead in the edible portion of fish (i.e., fillets) harvested from the UCR. The EPC for fish was the mean (arithmetic mean or KM mean) concentration for each fish species measured in fillets from all fish that were at least 30 cm long from Reaches 1-6 (CH2MHill, 2007; Exponent, 2013a; Windward, 2017a, 2018). To evaluate whether variability in fish tissue concentrations between species is potentially important and to inform the public, the lead concentrations for specific fish species are presented in Table 3-29.

3.4.3.2 Future Child Resident Exposed to Lead in Residential Soil and Fish from the UCR

Exposure to lead in residential soil and fish harvested from the UCR was assessed on an ADA by ADA basis for potential future child residents using the IEUBK model. The exposure pathway for this medium was incidental ingestion of lead in soil and dust by future full-time resident children, with the assumption that these areas may be used for future residential development. The EPCs for potential future residential soil lead concentration were the arithmetic means of triplicate IC samples collected, or the sample result for each ADA where a single IC sample was collected (see Section 3.3.10 for more information) of the lead concentrations of all samples collected at each ADA for soil fraction <149 µm in size (TAI, 2015a). ADAs were sampled at 0-8 cm depth interval. The RBA for lead in the ADA was based on the results from IVBA assessments, as described by TAI (2015a). In the event that no IVBA sample was collected for a given ADA, the RBA was the average of all other ADA RBA results (see Section 4.6) (EPA, 2020). Dust lead concentration was calculated as the MSD-based result from the RBA-adjusted soil lead EPC (the MSD approach included the contribution from outdoor air lead). Outdoor air

lead concentration was based on Site-specific information as described in Table 3-30. In addition to lead in future residential soil, future resident children may be exposed to lead in the edible portion of fish (i.e., fillets) harvested from the UCR. The EPC for fish was the mean (arithmetic mean or KM mean) concentration for each fish species measured in fillets from all fish that were at least 30 cm long from Reaches 1-6 (CH2MHill, 2007; Exponent, 2013a; Windward, 2017a, 2018). To evaluate whether variability in fish tissue concentration between species is potentially important and to inform the public, the lead concentrations for specific fish species are presented in Table 3-29.

3.4.3.3 Current Child Exposure to Lead at Residential Beaches (2014 and 2016 Soil Studies) and in Fish from the UCR

Exposure to lead in sediment at residential beaches was assessed at each beach DU on residential properties. The exposure pathway was incidental ingestion of sediment by children during beach recreation. For this HHRA, exposure at residential beaches used the time-weighting approach recommended by EPA (2003a) based on exposure frequency information to apportion exposure to the beach sediment (2 days/week) and residence (5 days/week). The EPC for lead concentration in residential beach sediment was the average (see Appendix 13 for more information) of the lead concentrations of all IC samples collected at each residential beach DU for sediment fraction <250 µm in size if triplicate IC samples were collected, or the sample result for each beach where a single IC sample was collected (CH2MHill, 2016a; TAI, 2017a). The residential soil EPC for lead was the average of the lead concentration in the house DU (or the nearest appropriate DU) containing the residential beach DU (see Appendix 13). The sediment samples were collected from the depth interval that was thought to be most likely for exposure for children spending time at beach DUs. The RBA for lead in the sediment at residential beach DUs was based on the results from IVBA assessments as described in Section 4.6 (EPA, 2020).

Table 3-16 gives the mean RBA-adjusted lead concentrations in soil from the 2014 and 2016 residential soil studies, excluding driplines and a DU used historically as a small ore mill or ore stockpiling site (Roland, 2019) sampled in 2016. For residential soil DUs subject to either a complete or partial TCRA or VRA (Table 2-3), the maximum measured lead concentration in backfill used during that removal was used as the lead concentration for those DUs. This information was included in the derivation of the mean concentrations shown in Table 3-16. These results were used to derive 129 ppm as the assumed residential soil lead concentration for exposure scenarios where a house DU soil lead concentration was not available for time weighting calculations (for example, incidental ingestion of beach sediment at public beaches, incidental ingestion of beach sediment at residential beaches at properties without a house DU or other surrogate DU on the property).

Exposure to lead in suspended sediment while swimming and wading in shallow surface water (TAI, 2009c) by residents with beaches on their property is likely to be greater than exposure to lead that is dissolved in surface water or sediment suspended in undisturbed surface water while swimming in deep water[74]. The exposure pathway in this HHRA was incidental ingestion of water by children while swimming and wading in shallow water during beach visits, camping trips, or while boating. The EPC for lead was based on the average concentration (see Section

[74] The concentration of lead in disturbed surface water is much greater than the concentration in non-disturbed surface water (3.15 vs. 0.205 µg/L, respectively). Both results are the mean from ProUCL using the KM method.

3.3.1 for more information) of lead in disturbed, unfiltered surface water from samples collected near public beaches (Exponent, 2013b). Because the lead in this medium would primarily be derived from sediment, the RBA for sediment (averaged across all public beaches) was used in the calculation.

Outdoor air lead concentration was based on Site-specific information as described in Table 3-30. In addition to lead in future residential soil, resident children may be exposed to lead in the edible portion of fish (i.e., fillets) harvested from the UCR. The EPC for fish was the mean (arithmetic mean or KM mean) concentration for each fish species measured in fillets from all fish that were at least 30 cm long from Reaches 1-6 (CH2MHill, 2007; Exponent, 2013a; Windward, 2017a). To evaluate whether variability in fish tissue concentration between species is potentially important and to inform the public, the lead concentrations for specific fish species are presented in Table 3-29.

3.4.3.4 Child Exposure to Lead from Incidental Ingestion of Surface Solid Media (Sediment or Soil) and Surface Water While Recreating at Public Beaches, Bossburg Flat Beach and Evans Campground UDUs, and Relict Floodplains during Beach Day Trips, Boating Trips, or Camping and from Consuming Fish from the UCR

Exposure to lead in solid media (sediment or beach soil) while recreating at beaches was assessed at all public beaches, Bossburg Flat Beach and Evans Campground Beach UDUs, and Relict Floodplains. The exposure pathway was incidental ingestion of surface sediment or soil by children while recreating at these areas (adult outdoor worker exposure to deeper horizons of soil or sediment during beach maintenance work is addressed in Section 3.4.3.5).

Public beaches were sampled in 2009-2011 and 2015 (see Table 5-8 for the list of beaches assessed). The EPC for lead concentration in public beach sediment was the average lead concentration (beaches sampled in 2009-2011; see Section 3.3.1 for more information) from all samples collected at each public beach DU for sediment in the <250 µm size fraction (TAI, 2014a). The RBA for lead in the sediment at public beach DUs was based on the results from IVBA assessments (see Section 4.6) (EPA, 2020).

For Bossburg Flats Beach and Evans Campground Beach there are solid media samples using the <250 µm fraction (SDU beach sediment samples) and <149 µm fraction (UDU soil samples) (TAI, 2016a). These samples were collected in 2015 from the depth interval that was thought to be most appropriate for exposure (i.e., most likely to be contacted by recreating children): beach DUs and UDUs were sampled at 0-15 cm depth interval. The EPC for lead concentration in Bossburg Flats and Evans Campground Beach samples collected in 2015 was the average lead concentration if triplicate IC samples were collected, or the sample result for each beach where a single IC sample was collected (TAI, 2016a). See Section 2.6.2.4 for more information. The RBA for lead in the soil and sediment samples from Bossburg Flats Beach and Evans Campground Beach was based on the results from IVBA assessments (see Section 4.6) (EPA, 2020).

Exposure to lead in soil at relict floodplains was assessed at all relict floodplains that were sampled in 2014 (see Table 3-21 for the list of sixteen RFDAs sampled at relict floodplains). The exposure pathway was incidental ingestion of relict floodplain soil by children while recreating.

The EPC for lead concentration in relict floodplain soil was the average lead concentration from all IC samples collected at each RFDA for soil in the <149 µm size fraction. Note that only 4 of the RFDAs had replicates collected, so the EPC was the mean value for those; the other 12 RFDAs just had a single sample collected that was used as the EPC (see Section 3.3.1 for more information). The relict floodplain soil samples were collected from the depth interval that was thought to be most likely for exposure: RFDAs were sampled at 0-7.5 cm depth interval. The RBA for lead in the soil at RFDAs was based on the results from IVBA assessments (TAI, 2015a). In the event that no IVBA sample was collected for a given RFDA, the RBA was the average of all other RFDA RBA results (see Section 4.6) (EPA, 2020).

For recreational visitor children, exposure to lead in suspended sediment while swimming and wading in shallow surface water (TAI, 2009c) during beach trips, boat trips, or camping trips is likely to be greater than exposure to lead that is dissolved in surface water or sediment suspended in undisturbed surface water while swimming in deep water[75]. The exposure pathway in this HHRA was incidental ingestion of water by children while swimming and wading in shallow water during beach visits, camping trips, or while boating. The EPC for lead was based on the average concentration (see Section 3.3.1 for more information) of lead in disturbed, unfiltered surface water from samples collected near public beaches (Exponent, 2013b). Because the lead in this medium would primarily be derived from sediment, the RBA for sediment (averaged across all public beaches) was used in the calculation (see Section 4.6) (EPA, 2020).

Table 3-16 gives the mean RBA-adjusted lead concentrations in soil from the 2014 and 2016 residential soil studies, excluding driplines, and (for lead) a DU sampled in 2016 that was historically used as a small ore mill or ore stockpiling site (Roland, 2019).[76] These results were used to derive 129 ppm as the assumed house soil lead concentration to be used for exposure scenarios where a house soil lead concentration was not available for time weighting calculations (such as all recreational beach exposures).

Outdoor air lead concentration was based on Site-specific information as described in Table 3-30. In addition to lead in future residential soil, resident children may be exposed to lead in the edible portion of fish (i.e., fillets) harvested from the UCR. The EPC for fish was the mean (arithmetic mean or KM mean) concentration for each fish species (because anglers may target specific species) measured in fillets from all fish that were at least 30 cm long from Reaches 1-6 (CH2MHill, 2007; Exponent, 2013a; Windward, 2017a, 2018).

3.4.3.5 Incidental Ingestion of Public Beach Sediment and Soil at Depth by Outdoor Workers

For outdoor workers, the exposure pathway was incidental ingestion of deep sediment and soil while working at a beach. The only beaches that have EPC data for the soil horizon of interest (i.e., 0-45 cm) for this exposure pathway at public beaches are Bossburg Flats and Evans Campground (TAI, 2016a). The EPC for lead concentration in deep sediment and soil at public beaches was the arithmetic mean lead concentrations from core samples collected either at SDUs for sediment in the <250 µm size fraction (including sample F-01) or at UDUs for soil in the <149 µm size fraction at Bossburg Flats and Evans Campground. The sediment and soil samples

[75] The concentration of lead in disturbed surface water is much greater than the concentration in non-disturbed surface water (3.15 vs. 0.205 µg/L, respectively). Both results are the mean from ProUCL using the KM method.
[76] Field replicates were averaged to produce a single result for each DU.

were collected from the depth interval that was thought to be most relevant to assess occupational exposure for beach maintenance work: 0-45 cm depth interval. The RBA for lead in the sediment and soil at public beach SDUs and UDUs, respectively, was based on the results from IVBA assessments (see Section 4.6) (EPA, 2020).

3.4.3.6 Inhalation of Outdoor and Indoor Air

Lead concentrations in outdoor air, indoor air, and indoor dust were included in the integrated assessment of risks to residents and recreators. The outdoor air lead concentration used as an input to the IEUBK was the KM mean measured concentration calculated by ProUCL from the 2002-2009 Sheep Creek monitoring data (CH2MHill, 2015). This air EPC was used as an input to the IEUBK model for all exposure scenarios assessed. Air exposure is not assessed discretely in the ALM; however, air lead exposure is reflected in the baseline PbB in the ALM.

3.5 Evaluation of Exposures to Non-Lead COPCs

3.5.1 Basic Equations

3.5.1.1 Ingestion Exposures

The amount of a chemical which is ingested is referred to as "intake" or "dose." For non-lead chemicals, exposure is quantified using an equation of the following general form:

$$DI = C \cdot (IR / BW) \cdot (EF \cdot ED / AT) \cdot RBA$$

where:

DI = Daily intake of chemical (mg of chemical per kg of body weight [BW] per day).

C = Concentration of the chemical in the contaminated environmental medium (soil, sediment, water, food) to which receptor is exposed. The units are mg/L for water, and mg/kg for solid media.

IR = Intake rate of the contaminated environmental medium. The units are L/day for aqueous media, and kg/day for solid media.

BW = Body weight of the receptor (kg).

EF = Exposure frequency (days/year). How often receptor is likely to be exposed to the contaminated medium over the course of a typical year.

ED = Exposure duration (years). How long receptor is likely to be exposed to the contaminated medium during his or her lifetime.

AT = Averaging time (days). The number of days over which the average dose is calculated. Usually, two different ATs are considered:

"Chronic" exposure includes ATs on the scale of years (typically ranging from 7 to 70 years). This exposure duration is used when assessing non-cancer hazards.

"Lifetime" exposure employs an AT of 70 years. This exposure interval is selected when evaluating cancer risks.

RBA = Relative bioavailability

Note that the factors EF, ED, and AT combine to yield a factor between zero and 1. Values near 1.0 indicate that exposure occurs nearly continuously over the specified averaging period, while values near zero indicate that exposure occurs infrequently.

For mathematical convenience, the general equation for calculating dose can be written as:

$$DI = C \cdot HIF \cdot RBA$$

where:

HIF = Human Intake Factor. This term describes the average amount of an environmental medium contacted by the exposed receptor each day. The value of HIF is typically given by:

$$HIF = (IR / BW) \cdot (EF \cdot ED / AT)$$

The units of HIF are L/kg-day for aqueous media and kg/kg-day for solid media.

Because one or more exposure parameters (e.g., IRs, BW, and EF) may change as a function of age, exposure calculations for non-cancer hazards are performed separately for children and adults. However, for estimating excess cancer risks from exposure to a chemical, because the same individual may be exposed beginning as a child and extending into adulthood, exposure is calculated as the time-weighted average (TWA) lifetime exposure:

$$DI_{TWA} = C \cdot [(IR_c / BW_c) \cdot (EF_c \cdot ED_c / AT) + [(IR_a / BW_a) \cdot (EF_a \cdot ED_a / AT)]$$

where the subscripts "c" and "a" refer to child and adult receptors, respectively.

3.5.1.2 Dermal Exposures

Exposure to a chemical by the dermal pathway is based on the dose absorbed into the body rather than ingested or inhaled. The dose of a chemical which is absorbed across the skin is referred to as the dermally-absorbed dose (DAD), which is quantified using the following equation (EPA, 2004b):

$$DAD = DA_{event} \cdot EF \cdot ED \cdot EV \cdot SA / (BW \cdot AT)$$

where:

DAD = Dermally-absorbed dose (mg of chemical per kg of BW per day).

DA_{event} = Absorbed dose per event (mg of chemical per square cm of skin surface area per event). This is media-specific and is further described below.

EF = Exposure frequency (days/year). How often receptor is likely to be exposed to the contaminated medium over the course of a typical year.

ED = Exposure duration (years). How long receptor is likely to be exposed to the contaminated medium during their lifetime.

EV = Event frequency (events/day). The number of times per day receptor contacts a contaminant in soil.

SA = Surface area (square centimeters [cm^2]). The area of skin exposed to the contaminated media.

BW = Body weight of the exposed receptor (kg).

AT = Averaging time (days). The number of days over which the average dose is calculated.

For chemicals in soil or sediment, DA_{event} is estimated as follows:

$$DA_{event} = C \cdot CnF \cdot DAF \cdot ABS_d$$

where:

C = Chemical concentration in soil or sediment (mg of chemical per kg of soil or sediment).

CnF = Conversion factor (10^{-6} kg/mg).

DAF = Dermal adherence factor (mg of soil per square cm of skin surface area per event). This describes the amount of soil that adheres to the skin per unit of surface area.

ABS_d = Dermal absorption fraction (unitless). This value is chemical-specific and represents the contribution of absorption of a chemical across receptor's skin from soil to the systemic dose. Table 3-32 summarizes the dermal absorption fraction values for each COPC.

For chemicals in water, DA_{event} is estimated as follows:

$$DA_{event} = K_p \cdot C \cdot t_{event}$$

where:

K_p = Chemical-specific dermal permeability coefficient of compound in water (cm/hour). Table 3-33 summarizes the K_p values for each chemical.

C = Chemical concentration in water (mg of chemical per cubic cm of water).

t_{event} = Event duration (hours/event). This describes how long receptor is likely to be exposed to the contaminated medium per exposure event.

The equation for calculating DAD is:

Soil/Sediment: $DAD = C_{soil/sediment} \cdot ABS_d \cdot HIF_{soil/sediment}$
Water: $DAD = C_{water} \cdot K_p \cdot HIF_{water}$

where:

$HIF_{soil/sediment} = (SA \cdot DAF \cdot EF \cdot ED \cdot EV \cdot CnF) / (BW \cdot AT)$

$HIF_{water} = (SA \cdot EV \cdot EF \cdot ED \cdot t_{event}) / (BW \cdot AT)$

The units of HIF are kg/kg-day for soil and sediment, and cm^2-hour/kg-day for water.

As described above, cancer risks were calculated as the TWA for a lifetime of exposure. For non-cancer hazards, children and adult receptors were evaluated separately.

Dermal exposures were only evaluated for COPCs with appropriate ABS_d values (for soil/sediment exposures) or K_p values (for water exposures) (EPA, 2001c).

3.5.1.3 Inhalation Exposures

Inhalation exposures are evaluated in accordance with the inhalation dosimetry methodology presented in EPA's *Risk Assessment Guidance for Superfund (RAGS) Part F: Inhalation Risk Assessment* (EPA, 2009a). In accordance with EPA (2009a), the inhaled exposure concentration (EC) for chronic exposures is calculated as:

$EC = C \cdot (ET \cdot EF \cdot ED / AT)$

where:

EC = Exposure concentration ($\mu g/m^3$). This is the time-weighted concentration based on the characteristics of the exposure scenario being evaluated.

C = Concentration of the chemical in air ($\mu g/m^3$) to which the receptor is exposed.

ET = Exposure time (hours/day). How long receptor is likely to be exposed to the contaminated medium over the course of a typical day.

EF = Exposure frequency (days/year). How often receptor is likely to be exposed to the contaminated medium over the course of a typical year.

ED = Exposure duration (years). How long receptor is likely to be exposed to the contaminated medium during their lifetime.

AT = Averaging time (hours). Length of time over which the TWA concentration is calculated.

The equation for exposure concentration is:

$$EC = C \cdot TWF$$

where:

TWF = Time-Weighting Factor (unitless). The value of TWF is given by:

$$TWF = (ET \cdot EF \cdot ED / AT)$$

As described above, when the same individual may be exposed beginning as a child and extending into adulthood, exposure was calculated as the TWA lifetime exposure for evaluating cancer risks. For non-cancer hazards, children and adult receptors were evaluated separately.

3.5.2 Exposure Parameters

For every exposure scenario of potential concern, it is expected that there will be variability among individuals in the level of exposure at a specific location because of differences in intake rates, body weights, exposure frequencies, and exposure durations. Thus, there is normally a wide range of average daily intakes (ADIs) among different members of an exposed population. Because of this, all daily intake calculations must specify what part of the range of doses is being estimated. Typically, attention is focused on intakes that are near the upper end of the range (e.g., the 95^{th} percentile; RME), and on intakes that are "average" or are otherwise near the central portion of the range (CTE). Both RME and CTE scenarios were evaluated for receptors in this HHRA.

When selecting parameters for the RME scenario, the intake variables are selected such that the combination of the intake variables results in a "reasonable" maximum estimate of the daily intake (EPA, 1989). In other words, some inputs are set equal to mean values (e.g., body weight) and some inputs are set equal to upper bound values (e.g., ingestion rates, exposure frequency, and exposure duration), such that the resulting combination yields an estimate that is RME (EPA, 1989). When selecting parameters for the CTE scenario, the intake variables for a specific exposure pathway (e.g., body weight, ingestion rate, exposure frequency, exposure duration) are usually based on mean or median values, such that the CTE represents the "typical" or "average" exposure. As noted above, because exposure parameters (e.g., intake rates, body weight, and exposure frequency) may change as a function of age, values were selected separately for children (0-6 years) and adults (7+ years).

Tables 3-34 through 3-36 present summaries of HIF values for the RME and CTE exposure scenario, respectively (for ingestion and dermal pathways) and TWF values (for inhalation pathways) by receptor and exposure pathway. Detailed information on exposure parameters for each exposed population is provided below.

3.5.2.1 Residential and Outdoor Worker Exposure Parameters

The EPA has collected a wide variety of data and performed several studies to establish national default values for most residential and outdoor worker exposure parameters. For this HHRA, the primary sources of these parameters were:

- EPA, 1989: Risk assessment guidance for Superfund, Volume 1, Human health evaluation manual (Part A)
- EPA, 1994b: Guidance manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children
- EPA, 1996d: Recommendations of the technical review workgroup for lead for an interim approach to assessing risks associates with adult exposures to lead in soil
- EPA, 1998a: Ambient water quality criteria derivation methodology
- EPA, 2004b: Risk assessment guidance for Superfund, Volume 1. Human health evaluation manual (Part E – Dermal)
- EPA, 2011a: Exposure factors handbook
- EPA, 2014a: Human health evaluation manual, Supplemental guidance: Update of standard default exposure factors
- EPA, 2019a: Regional screening levels (RSLs) – generic tables

If default exposure parameters were not available, then professional judgement was used in selecting appropriate exposure parameter values. Tables 3-37 and 3-38 present the parameters that were used in this HHRA for the RME and CTE scenarios for outdoor worker and resident populations, respectively.

Residents, with and without beaches, were assumed to consume fish at a rate determined by Site-specific surveys. For both the RME and CTE scenarios, the most conservative 95th percentile fish consumption rate from either the RecUse or CCT Tribal Survey was selected for adults. For the RME, the fish consumption rate for adults was taken from the RecUse Survey (SRC, 2019a). The adult CTE fish consumption rate was taken from the CCT Tribal Survey (SRC, 2019b). The fish consumption rates used to evaluate fish consumption for the residential population also represent fish consumption for the members of the CCT population that are not high-intensity fish consumers (as evaluated in Appendix 1). Reliable fish consumption estimates could not be derived for children using the Site-specific survey data (SRC, 2019a, 2019b). The adult fish consumption estimates that were derived using survey data were multiplied by a ratio of children-to-adult dietary intake of 0.5 which was estimated using energy requirements for

children and adults based on regression models presented in Institute of Medicine (IOM, 2005) (see Appendix 14)[77].

The CTE daily fish consumption rates were estimated by Mountain Whisper Light (MWL) using a two-part regression model developed by the National Cancer Institute (NCI; MWL, 2017; Tooze et al., 2006) with data provided by the CCT Tribal Survey 24-hour dietary recall interviews (Westat Inc., 2012). The data included all freshwater finfish consumption reported on the 24-hour dietary recalls. Prior to estimating DCRs, the fish consumption data for each participant were reduced to daily consumption amounts (grams [g]/day) for each 24-hour dietary recall completed. After extensive testing of various NCI models, MWL did not include the food questionnaire (FQ) data in the NCI model estimates because the FQ data did not improve the estimates of daily fish consumption rates with the NCI model (MWL, 2017). The estimate of the RME DCR for fish is described in Section 3.5.2.2.

Residents with beaches were assumed to visit the beach on their property 32 days/year for the RME scenario, representing 2 days/week for 16 weeks of seasonable weather each year (Table 3-38). It was assumed that resident children join adults when spending time at their beach. For the CTE scenario, adult and child residents with beaches were assumed to visit their beach 20 days/year. The CTE EF for residential beaches is the RME EF for the number of day trips per year to public beaches (SRC, 2019a). The CTE and RME EF for residents with beaches on their property assume people with beaches on their property will visit the beaches more frequently than people who visit public beaches along the UCR.

3.5.2.2 Recreational Visitor Exposure Parameters

Exposure parameters for visitors to the UCR were estimated with data provided by the RecUse Survey (IEc, 2012, 2013a; SRC, 2019a) and professional judgement and are shown in Table 3-39.

Fish Consumption Rates

Daily fish consumption rates were estimated using data provided by the RecUse Survey fish consumption diaries and the survey questionnaire (IEC, 2013a; SRC, 2015b, 2019a). The estimates are for the population of recreational visitors who are fish consumers. The diary data were used for survey participants who provided three complete monthly diaries; otherwise, the questionnaire data were used. Prior to combining the questionnaire and diary data, the fish consumption data for each participant were reduced to an average DCR (g/day) for that participant. For the questionnaire data, the fish DCR was calculated as the total fish meals reported for the preceding 12 months, multiplied by the typical meal size (g), and then divided by 365 days. For the diary data, the fish DCR was calculated as the total amount (g) of fish ingested over the 3 monthly diaries, divided by 90 days. An analysis of the fish DCR did not support

[77] If a rate of consumption of a specific food could not be estimated for children from the RecUse or CCT Tribal Surveys (or other credible sources), consumption rates for children were calculated by apportioning the adult consumption rate based on estimated energy requirements for children and adults. The adjustment factor was as follows: child consumption = adult consumption × 0.5 (Appendix 14). This adjustment factor applies to the overall diets of children and adults, but may not accurately reflect child/adult consumption ratios for specific foods that are likely to be affected by differences in food preferences between children and adults.

adjusting the diary and/or questionnaire data to account for the difference in the lengths of time covered by the two survey instruments (i.e., 90 versus 365 days, respectively), or potential differences in recall error between the two survey instruments (SRC, 2019a). The mean (CTE) and 95th percentile (RME) DCR for fish were estimated using the SAS SurveyMeans[78] procedure.

Exposure Frequency

Exposure frequencies were estimated using the number of past trips to the UCR (Reaches 1-6) reported by the survey participants. Participants were asked to provide the number and location of past camping trips, boating trips and beach trips. The location of each beach and camping facility was determined using information provided by the survey DSR (IEc, 2013a) and based on further discussion with DOI (IEc, 2013b). Exposure frequencies were estimated separately for each of the three trip types and for each region of the UCR. The data available for each participant are as follows:

- Beach trips - number of beach trips reported during June-September of the year prior to the survey interview. Participants reported the number of trips for each beach they visited.
- Boating trips - number of boating trips taken during the "current" season (i.e., the season in which the survey interview took place: spring, summer, fall or winter) and the number of boating trips taken during the four seasons prior to the "current" season. Participants also reported the river reaches they visited during each visit.
- Camping trips – For each camping facility/location they camped at, participants provided the name of the camping facility/location, the number of nights spent camping at that location during the current season, and the number of nights spent camping at that location during the four seasons prior to the "current" season.

Exposure Time

Research indicates that people are not able to accurately recall times they typically spend engaging in recreational activities (IEc, 2013a). Therefore, participants were asked to provide the time they spent swimming (and wading) in water greater than waist deep during their current trip. Times spent swimming were estimated for adults and children for each of the three trip types (beach, boating and camping), as described in detail in SRC (2019a). As discussed in SRC (2019a), estimates of time spent swimming during beach trips were very precise for adults and children for all regions of the UCR combined. Few interviews captured swimming ET for children on boating and camping trips (n = 16 and n = 24, respectively). As such, swimming ET for those two types of trips were estimated by combining adult and child data. The number of interviews in the upper region was lower than expected, likely because of higher than average snowfall the previous winter, which flooded Black Sand Beach and necessitated a lower drawdown than usual, making some boat launches inaccessible for portions of the time. As a result, estimates of ET by lake region are considered less reliable than estimates of ET with lake regions combined. Therefore, separate estimates by lake region were not used to estimate risk.

[78] SAS|STAT and SAS|Graph Software Version 9.4 of the SAS System for Windows. Copyright (c) 2002–2012 by SAS Institute Inc.: Cary, NC, USA. All Rights Reserved; SAS|Enterprise Guide Version 7.13. Copyright (c) 2016 by SAS Institute Inc.: Cary, NC, USA. All Rights Reserved.

Table 3-39 presents estimates of the means and 95th percentiles for exposure frequencies and exposure times for beach, boating, and camping trips, along with other parameters for the CTE and RME scenarios, which were used to estimate potential risks to recreational visitors. Additional details on the RecUse data, data reductions, and the statistical methods that were used to estimate exposure parameters are provided in SRC (2019a).

4 TOXICITY ASSESSMENT

4.1 Overview

The toxicity assessment identifies potential adverse human health effects from a chemical, and how these adverse effects depend on exposure level, also known as the dose-response. In addition, the toxic effects of a chemical frequently depend on the route of exposure (oral, inhalation, dermal) and the duration of exposure (subchronic, chronic, or lifetime). Thus, a full description of the toxic effects of a chemical includes a listing of what adverse health effects the chemical may cause, and how the occurrence of these effects depends upon dose, route, and duration of exposure.

The toxicity assessment for lead is below in Section 4.2. The toxicity assessment of non-lead COPCs is usually divided into two parts: the first characterizes and quantifies the non-cancer effects of the chemical, while the second addresses the cancer effects of the chemical. This two-part approach, described in Sections 4.3 and 4.4, is employed because there may be major differences in the time-course of action and the shape of the dose-response curve for non-cancer and cancer effects.

4.2 Toxicity of Lead

Health effects associated with exposure to inorganic lead and compounds include, but are not limited to: neurotoxicity, developmental delays, hypertension, impaired hearing acuity, impaired hemoglobin synthesis, and male reproductive impairment. Lead is stored in the body, primarily in bone. Lead body burdens vary significantly. Thus, based on current knowledge of lead pharmacokinetics, and an apparent lack of a threshold effect (NTP, 2012), no toxicity values have been derived for lead.

Risks from lead are evaluated using a different approach than other chemicals. First, because lead is widespread in the environment, exposure can occur from many different sources. Thus, lead risks are based on all sources rather than just Site-related sources. Second, because epidemiological studies of lead exposures and resultant health effects in humans have not established a PbB below which adverse effects are not observed, lead exposures and risks are typically assessed by calculating the levels of lead that may occur in the blood among exposed populations and comparing these to PbB associated with health effects (for more information, see EPA, 1994b, 1998b). For convenience, the concentration of lead in blood is usually abbreviated "PbB," and is expressed in units of μg/dL.

4.2.1 Blood Lead Level of Concern

Health effects from elevated PbB are most sensitive for the developing nervous systems of young children or the fetus of pregnant women because: 1) young children typically have higher exposures (per unit body weight) to lead-contaminated media than adults, 2) young children typically have higher lead absorption rates than adults, and 3) young children and fetuses are generally more susceptible to effects of lead than are adults (NTP, 2012).

EPA's ORD reviewed the health effects evidence for lead in the 2013 ISA for Lead and found that several studies have observed "clear evidence of cognitive function decrements (as measured by Full Scale IQ, academic performance, and executive function) in young children (4-11 years old) with mean or group PbBs between 2 and 8 μg/dL (measured at various lifestages and time periods)." In addition, the NTP's (2012) Monograph on Health Effects of Low-Level Lead found sufficient evidence of delayed puberty, reduced post-natal growth, and decreased hearing for children at PbBs below 10 μg/dL and adverse effects on academic achievement, IQ, other cognitive measures, attention-related behaviors, and problem behaviors at PbBs below 5 μg/dL (NTP, 2012).[79]

Consistent with other EPA risk assessments, risks from exposure to lead (Pb) in Site media were evaluated and presented separately from risks from exposure to other COPCs. Risks from exposure to lead were assessed using the IEUBK Model for Lead in Children for residential and recreational exposures, and the ALM for the outdoor worker exposure pathway. Risk benchmarks are risk levels that EPA uses to distinguish risks that are a potential concern from risks that are below the level of concern. Recognizing the recent advances in lead toxicology (ATSDR, 2020), this risk assessment has evaluated a range of blood lead levels and the associated soil concentrations within which the risk management decision will most likely be made, from 3 to 8 μg/dL. For this HHRA, the risk evaluation benchmarks selected for elevated lead risk were defined as >5% probability of exceeding a PbB of 3, 5, or 8 μg/dL (referred to as "P3," "P5," and "P8," respectively). The IEUBK model cannot be used with a risk benchmark below P3 (such as P2) because the risk goal would be exceeded even if the soil lead concentration is 0 ppm due to dietary lead exposure. P3 was selected as a lead risk benchmark to quantitatively evaluate the low end of the risk range of child PbBs associated with adverse health effects and P8 was selected as a less protective benchmark.

The evaluation used a version of the IEUBK model (version 1.1, build 11) with updates that reflected EPA's Superfund TRW for Lead recommended changes to IEUBK version 1.1 input parameter default values, that were based on recent advances in scientific information, and will be incorporated in IEUBK (version 2). The differences between IEUBK (version 2) and IEUBK (version 1.1) with the updated input parameter values shown in Tables 3-30 and 3-31 are too small to meaningfully impact the results of the HHRA. An evaluation of the performance of IEUBK (version 2) found strong support for applications of the IEUBK (version 2) in CERCLA-related HHRAs (Vandenberg, 2020).

4.3 Non-Cancer Effects

All chemicals can cause adverse health effects if an individual is exposed at a sufficient dose. Alternatively, when the dose is sufficiently low, no adverse effect is observed. The key parameter is the threshold dose at which an adverse effect first becomes evident. Doses below the threshold are considered to be safe, while doses above the threshold may cause an adverse effect.

[79] The Centers for Disease Control and Prevention (CDC) recommendations for follow-up and case management based on child PbBs are available online at https://www.cdc.gov/nceh/lead/advisory/acclpp/actions-blls.htm.

The threshold dose is typically estimated from studies of humans and/or animals by finding the highest dose that does not produce an observable adverse effect, and the lowest dose which does produce an effect. These are referred to as the "no-observed-adverse-effect-level" (NOAEL) and the "lowest-observed-adverse-effect-level" (LOAEL), respectively. The threshold is presumed to lie between the NOAEL and the LOAEL. Alternatively, dose-response data for the critical effect may be modeled using EPA's Benchmark Dose Modeling Software to obtain the lower confidence limit on the estimate of the threshold dose (BMDL). Non-cancer hazard evaluations are not based directly on the threshold exposure level, but on a value referred to as the Reference Dose (RfD) for oral exposures or Reference Concentration (RfC) for inhalation exposures. The RfD and RfC are estimates (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

The RfD and RfC values are derived from a BMDL or NOAEL (or a LOAEL if a reliable NOAEL is not available) by dividing by an "uncertainty factor" (UF). Factors accounting for several sources of uncertainty (e.g., interspecies uncertainty [UF_A], intraspecies variability [UF_H], subchronic to chronic extrapolation [UF_S], LOAEL to NOAEL extrapolation [UF_L], etc.) are combined into a single UF that is applied to the RfD or RfC value. If the data are from studies in humans, and if the observations are considered to be very reliable, the UF may be as small as 1.0 (EPA, 2002f).[80] However, the UF is normally at least 10, and can be as high as 10,000 when toxicity data are limited or uncertain. The effect of dividing the BMDL, NOAEL, or LOAEL by a UF is to ensure that the RfD or RfC is not higher than the threshold level for adverse effects. Thus, there is always a "margin of safety" built into RfD and RfC values. Exposures higher than the RfD or RfC may carry some risk, but because of the margin of safety, an exposure above the RfD or RfC does not mean that an effect will necessarily occur.

4.4 Cancer Effects

For cancer effects, the toxicity assessment process has two components. The first is an evaluation of the weight of evidence (WOE) to determine if a COPC causes cancer in humans. Previously, this evaluation was performed by the EPA using the system summarized in Table 4-1 (EPA, 1986).

More recently, EPA has developed a revised classification system for characterizing the WOE for carcinogens (EPA, 2005c). However, this system has not yet been implemented for a number of chemicals, so the older classification scheme is still relevant.

For chemicals which are classified in Group A, B1, B2, or C, the second part of the toxicity assessment is to quantify the carcinogenic potency of the chemical. This is done by relating the number of cancers observed in animals or humans as the dose increases (dose-response). Typically, it is assumed that the dose response curve for cancer has no threshold, arising from the origin and increasing linearly until high doses are reached. Thus, the most convenient descriptor of cancer potency is the slope of the dose-response curve at low doses (where the slope is still

[80] For example, the intraspecies UF was reduced to 1 for nitrate based on very specific data regarding the vulnerability of infants and children within certain age ranges.

linear). This is referred to as the Slope Factor (SF), which has units of probability of cancer per unit dose.

Estimating the cancer SF is often complicated by the fact that observable increases in cancer incidence usually occur only at relatively high doses, frequently in the part of the dose-response curve that is no longer linear. Thus, it is necessary to use mathematical models to extrapolate from the observed high dose data to the desired (but unmeasurable) slope at low dose. To account for the uncertainty in this extrapolation process, EPA typically chooses to employ the upper 95UCL of the slope as the SF. That is, there is a 95% probability that the true cancer potency is lower than the value chosen for the SF. This approach ensures that there is a margin of safety in cancer as well as non-cancer hazard estimates. In this assessment, arsenic is a notable exception because its SF is a most likely or central tendency estimate, not an upper 95^{th} estimate.

For inhalation exposures, cancer risk is characterized by an Inhalation Unit Risk (IUR) value. This value represents the upper-bound excess lifetime cancer risk estimated to result from continuous lifetime exposure to a chemical at a concentration of 1 $\mu g/m^3$ in air.

4.5 Human Toxicity Values

Toxicity values (RfD, RfC, SF, and IUR values) that have been established by EPA are listed in an on-line database referred to as "IRIS" (Integrated Risk Information System) (EPA, 2015e). Other toxicity values are available as interim recommendations from EPA's Superfund Technical Assistance Center operated by the National Center for Environmental Assessment (NCEA). Selection of toxicity values (RfD, RfC, SF, and IUR values) for use in this risk assessment follows the hierarchy for use in HHRA at Superfund sites as described in Section 3.2.2 and EPA (2003d). A table of toxicity values derived following this hierarchy is maintained by EPA and is periodically updated by Oak Ridge National Laboratories (EPA, 2019a). This table is incorporated into the RSL table.

All toxicity values used in this assessment were taken from the May 2019 version of the RSL tables. Tables 4-2 through 4-5 show the toxicity values used for evaluation of human health risks from COPCs at this Site. Points to note regarding the data in these tables are listed below (see also the User's Guide to the RSL):

- Two oral RfD values are available for cadmium: food or water. The value for food was used to evaluate risks from exposure to sediment, soil, fish, macroinvertebrates, amphibians and reptiles, waterfowl, upland birds, wild game (mammals), and plants. The value for water was used to evaluate risks from exposure to surface water.
- Two oral RfD values are available for manganese: food and nonfood. The IRIS RfD (1.4×10^{-1} mg/kg-day) includes manganese from all sources, including diet. IRIS recommends subtracting the dietary contribution when evaluating non-food (e.g., drinking water or soil) exposures to manganese, leading to an RfD of 7.1×10^{-2} mg/kg-day for non-food items. IRIS further recommends using a modifying factor of 3 when calculating risks associated with non-food sources because of uncertainties, leading to an RfD of 2.4×10^{-2} mg/kg-day. This RfD based on non-food sources was used for sediment, soil, and surface water.

- The RfD and cancer SF for arsenic are based on the inorganic form. However, arsenic that accumulates in fish tissue is present mostly in a relatively non-toxic, organic form, usually as arsenobetaine (ATSDR, 2007). Numerous studies have measured the fraction of total arsenic in fish that exists as inorganic (toxic) arsenic in fish (e.g., Yost et al., 1998; Schoof et al., 1999; EPA, 2005d). Most measured values are below 10%, with a value of about 4% being typical (EPA, 2005d; Lorenzana et al., 2009). Inorganic arsenic was not detected in any fish fillet samples collected on-Site.[81] However, both inorganic and total arsenic were detected in whole body samples of some fish collected in 2005. Inorganic arsenic was detected in 3 of the 25 whole body fish submitted for analysis of both total and inorganic arsenic, and total arsenic was detected in all 25 whole body fish. When a mean inorganic arsenic concentration was calculated using the Reporting Limit as the concentration value for non-detects, the mean inorganic arsenic was 13.45 µg/kg ww, and the mean total arsenic in whole body fish was 246.4 µg/kg ww. This results in a Site-specific fraction of inorganic arsenic (compared to total arsenic) of 5.5% in whole body fish. For this assessment, if a fish tissue (fillet) sample was analyzed for inorganic arsenic, that result was used as the inorganic arsenic concentration in the EPC calculations. If a fish tissue (fillet) sample was not analyzed for inorganic arsenic, then the Site-specific fraction of 5.5% was applied to the total arsenic concentration and that result was used as the inorganic arsenic concentration in the EPC calculations.
- Data on metal speciation at the Site are not available. Toxicity values for metals were selected based on the most conservative value, unless it was not plausible for the medium. Details of the toxicity value selections for metals are as follows:
    - Chromium detected at the Site was assumed to be in the hexavalent form [Cr(VI)], the most toxic form of chromium. This is a conservative assumption and is likely to overestimate actual exposures.
    - For the purposes of this assessment, it was assumed that nickel in sediment, soil, fish, macroinvertebrates, amphibians, reptiles, waterfowl, upland birds, wild game (mammals), and plants at the Site is in the form of refinery dust. This is likely to overestimate actual exposures. For nickel in surface water (total or dissolved), the toxicity values for soluble nickel salts were used, as soluble forms are most likely to occur in an aqueous environment.
    - Thallium at the Site was assumed to exist in the form of soluble salts. The oral RfD for soluble thallium salts is as low or lower than RfDs for other forms of thallium. This is likely to overestimate actual exposures.
- Toxicity values are not available for o,p'-dichlorodiphenyldichloroethane (DDD) or o,p'-dichlorodiphenyltrichloroethane (DDT), but were available for the closely related compound p,p'-DDT; thus, toxicity values for p,p'-DDT were used for these compounds.
- alpha-Chlordane and gamma-chlordane are both constituents of technical grade chlordane, so the exposure concentrations for these compounds were summed and the

[81] The average % inorganic arsenic was also calculated using measured total arsenic in fillets and the method detection limit as the value for inorganic arsenic, as per the Arsenic Data Usability Report (SRC, 2012); the calculated % arsenic was 5.6%, similar to the measured value for whole body fish.

toxicity values for technical grade chlordane (Chemical Abstracts Service Registry Number [CASRN]: 57-74-9) were used to estimate associated risks.

- Toxicity values were not available for several other COPCs (see Table 4-6[82]). Risks associated with exposure to these COPCs are discussed in Section 6.0.

4.5.1 Toxicity Values for Dermal Exposures

Oral toxicity factors are expressed in terms of toxicity per unit dose of chemical ingested, rather than in terms of toxicity per unit amount of chemical absorbed. However, the equations for characterizing dermal contact with chemicals provide exposure values that are based on absorbed dose rather than ingested dose. Thus, oral RfD and SF values must be adjusted for use in evaluating dermal exposures:

$$\text{RfD(dermal)} = \text{RfD(oral)} \cdot \text{Oral absorption fraction}$$

$$\text{SF(dermal)} = \text{SF(oral)} / \text{Oral absorption fraction}$$

Table 4-7 lists the oral absorption efficiency for dermal fractions used to adjust oral toxicity values for use in assessing dermal exposure, as recommended by EPA (2004b). If chemical-specific absorption fractions are not available, a value of 1.0 was assumed, consistent with EPA (2004b) guidance.

4.5.2 Toxicity Values for 2,3,7,8-Tetrachlorodibenzo(p)dioxin (TCDD)-Like Congeners

Cancer risks and non-cancer hazards from exposure to dioxins, furans, and dioxin-like PCB congeners were assessed using the toxicity values for TCDD in conjunction with WHO (2005; cited as van den Berg et al., 2006) TEFs (Table 4-8), as recommended by EPA (2010a). The TEFs were used to convert EPCs for individual congeners to TEQ values, which were then summed to derive a total TEQ. The total TEQ was used as the EPC in the exposure assessment and combined with the toxicity values for TCDD to estimate risks.

4.5.3 Toxicity Values for Polycyclic Aromatic Hydrocarbons (PAHs)

Cancer risks from exposure to PAHs were assessed using the oral SF and IUR for benzo(a)pyrene in conjunction with the EPA (1993) relative potency factors (RPFs); see Table 4-9), as recommended by EPA (2018a). Each RPF is multiplied by the SF and unit risk for benzo(a)pyrene to derive a SF and unit risk for that PAH.

4.6 Adjustments for Relative Bioavailability (RBA)

An accurate assessment of human exposure to ingested chemicals requires knowledge of the amount of chemical absorbed from the gastrointestinal (GI) tract into the body from Site media compared to the amount of absorption that occurred in the toxicity studies used to derive the toxicity factors (EPA, 2007c). This ratio (amount absorbed from Site media compared to the amount absorbed in toxicity tests) is referred to as RBA.

[82] Only the common metals highlighted with "b" in Table 4-6 are applicable to this HHRA.

Accounting for RBA is particularly important for ingested metals (Goyer et al., 2004; Bradham et al., 2018; SRC, 2017). In general, metals in soil or sediment exist in the form of mineral particles that are not rapidly solubilized in GI fluids when ingested, while toxicity studies often utilize readily soluble forms of the test chemical. In the absence of data to the contrary, EPA (1989) recommends assuming equal bioavailability of a chemical in soil, diet, and water (i.e., RBA = 1.0). Data are limited or absent for most chemicals (aside from lead and arsenic). Therefore, RBA values were set to 100% for this HHRA for all COPCs except lead and arsenic. This is likely to overestimate actual exposures, as discussed in Section 6.3.1.3.

In the 2014 and 2016 Residential Soil Studies, arsenic and lead IVBA from soil was measured in a subset of DUs (SRC, 2014b; Ramboll Environ, 2016). For each DU with IVBA data, an RBA-adjusted soil lead concentration was calculated using the following equations (EPA, 2006e, 2009b, 2020):

$$\text{RBA} = (0.878 \times \text{IVBA} - 0.028) \quad \text{(Equation 1)}$$

$$\text{RBA-adjusted Pb concentration} = \text{RBA}/0.6 \times \text{measured Pb concentration} \quad \text{(Equation 2)}$$

where IVBA is in decimal format (i.e., not as a percentage), 0.6 is the default soil RBA in the IEUBK model (0.6 = 0.3/0.5), and the measured lead concentration is based on the IC soil sample result (or average, if replicate IC samples were collected).

Soil arsenic concentrations for the 2014 and 2016 residential soil data were adjusted for RBA using the following equations (EPA, 2017e, 2020):

$$\text{RBA} = (0.79 \times \text{IVBA} + 0.03) \quad \text{(Equation 3)}$$

$$\text{RBA-adjusted As concentration} = \text{RBA} \times \text{measured As concentration} \quad \text{(Equation 4)}$$

where IVBA is in decimal format (i.e., not as a percentage), and the measured arsenic concentration is based on the IC soil sample result.

The mean RBAs for arsenic and lead that were applied for residential soils not evaluated for IVBA are presented in Tables 4-10 and 4-11. The approach for adjusting arsenic and lead concentrations in DUs that were not evaluated for IVBA required categorizing the DUs as being located either within or outside the 2014 residential soil study area and assigning the corresponding mean RBA from Tables 4-10 and 4-11, as described in further detail in Appendix 11 (SRC, 2017).

Equations 1 through 4 were used to adjust arsenic and lead concentrations in Upland soil ADAs and RFDAs as well, for those ADAs and RFDAs having measured IVBA values. The mean RBA from those ADAs and RFDAs with measured IVBA was used to adjust arsenic and lead concentrations in soil samples from ADAs and RFDAs not having measured IVBA data (Table 4-12).

Public beach sediment data were also adjusted for RBA. EPCs for public beach sediment collected in 2009, 2010, and 2011 were first estimated for the <250 µm GSF using five 12-point

composite samples collected from the 0-6-inch depth interval from each beach. Each of the composite samples was sieved to four GSFs: <63 µm, 63-125 µm, 125-250 µm, and 250 µm-2 mm; the first three GSFs were used to estimate EPCs for the public beaches. One composite sample for each beach had all four GSFs analyzed for TAL metals and IVBA for arsenic and lead. The EPCs for the <250 µm GSF were calculated using a weighted average of the three GSFs that comprised the <250 µm GSF, where the weights were based on the mass of soil in each of the three GSFs. Likewise, the RBA-adjusted results for arsenic and lead in each sample were calculated as a weighted average for each of the three GSFs that comprised the <250 µm GSF. The measured lead and arsenic concentrations along with measured lead IVBA% in the <250 µm GSF provided by the reanalysis of beach sediment data (EPA, 2013a) were used for Bossburg Flat, Evans Campground, Flat Creek, Lyons Island, and Swimming Hole beaches rather than the estimates derived for them using the method described above. The calculated <250 µm arsenic IVBA% from the 2009-2011 results were applied to measured <250 µm arsenic concentrations from the 2013 reanalysis.

EPCs for the Bossburg Flat Beach and Evans Campground Beach SDUs (including location F-01) and UDUs sampled in 2015 were calculated by exposure area (TAI, 2016a). Equations 1 through 4 were used to adjust arsenic and lead concentrations in surface and subsurface sediment and beach soil samples using measured IVBA results for each sample.

Surface water concentrations of arsenic and lead were also adjusted for RBA using Equations 1 through 4 above. The mean RBA for arsenic (20.63%) and lead (44.42%) from across all public beach sediment samples was used to adjust arsenic and lead concentrations in surface water samples.

5 RISK CHARACTERIZATION

5.1 Lead Risk Calculations

5.1.1 Model Analyses for Child Exposure Scenarios

The IEUBK model (version 1.1, build 11) was used for all analyses involving lead exposure for children. This version of the IEUBK model was updated to include inputs recommended by the EPA TRW Lead Committee[83] as shown in Table 3-30.

Batch mode was used to assess risk for some exposure pathways using the IEUBK model. Because this assessment is prospective (i.e., not assessing actual children, but "typical" or hypothetical children 12-72 months old), the probability of exceeding a target PbB is based on the predicted average of the geometric mean blood lead result for children age 12-72 months. Instead of running the IEUBK model in batch mode 60 times (i.e., once for each age month) for each DU or ADA, the TRW Lead Committee recommends using the result from the 32-month-old child in batch mode to approximate the PbB average of the 12-72-month age range for children exposed to soil lead concentrations ranging from 100 to 800 ppm (TRW, 2017). That surrogate approach was used here. Appendix 5 presents the IEUBK batch mode output.

As discussed in Section 4.2.1, lead risk was quantitatively assessed in terms of whether the exposure exceeded the risk benchmark. For this HHRA, the selected benchmarks for quantitative risk evaluation were "does the exposure result in more than 5% probability of exceeding PbB of 3, 5, or 8 µg/dL;" P3, P5, and P8, respectively. For media other than residential soil, lead risk may also be presented in terms of how exposure to the medium contributes to overall risk given a selected concentration of lead in residential soil. When lead intake from the subject medium was <1 µg Pb/day, then that intake contributed minimally to lead risk (see Section 3.4).

Because the IEUBK model integrates exposures from all media, high lead exposures in media not associated with the specific pathway in question can result in benchmark exceedances even when the lead concentration(s) in media associated with the pathway in question is low to moderate. For example, for P5, residential soil lead concentrations near 200 ppm (the approximate soil lead PRG using the P5 risk benchmark) allow for little to no exposure to lead through other pathways.

The following sections present the results of the lead risk characterization by exposure scenario.

5.1.1.1 Current Child Resident[84] Exposed to Lead in Residential Soil with and without Fish Consumption

Methodology

This exposure pathway was assessed using the IEUBK model to calculate risk to current child residents from exposure to lead in Site air, residential DU soil/dust, and fish by species caught and consumed from the UCR. Model runs were done with and without fish consumption. Batch

[83] https://www.epa.gov/superfund/lead-superfund-sites-technical-assistance.

[84] While Appendix 1 evaluates the high-intensity resource users within the CCT population, the non-subsistence CCT population is represented by the residential population evaluated in this HHRA.

mode was used to assess risk for this pathway on a DU-by-DU basis using the IEUBK model (see Section 3.3.1 for more information). The receptor population includes all child residents. Arithmetic mean values for the ICs of each DU sampled in triplicate in the 2014 and 2016 residential soil studies or the sample result if only one IC sample was collected from a DU (aside from CCT tribal allotments, driplines, residential beaches, and 1 DU that was previously used as a potential ore stockpile) were used as the soil lead EPC for the batch mode calculation. The soil lead EPC was the RBA-adjusted lead result (see Section 4.6 and Appendix 11; EPA [2020]) to allow the default RBA for soil to be retained in the IEUBK model batch mode analysis. Dust lead concentration was calculated as the MSD-based result from the RBA-adjusted soil lead EPC (the MSD approach included the contribution from outdoor air lead). Outdoor air lead concentration was based on Site-specific information as described in Table 3-30. As discussed in Section 5.1.1, the results for the 32-month-old child were used as a surrogate for hypothetical children 12-72 months old in current residences.

Because fish consumption resulted in lead intake <1 µg/day (as shown in Table 5-1), risk for this receptor was assessed both with and without consumption of fish from the UCR. In general, risk from consuming fish from the UCR is low for lead for all species but sucker. Risk was calculated using lead intake from consuming fish as an alternate intake in the IEUBK model (using the individual fish species average lead concentration and the DCR for fish from the CCT Tribal Survey – 0.5 × the value used as the adult CTE DCR, Section 3.5.2.1 [SRC, 2019b; IOM, 2005; see Appendix 14]). The alternate intake of lead from fish consumption was calculated as follows: lead concentration in the fish species (µg Pb/g fish) × 4.2 g fish/day = lead intake for each fish species (µg Pb/day). Because lead in fish is dietary lead, a default absorption fraction of 50% was used in the IEUBK model for this alternate intake. The small amount of fish consumed (4.2 g/day) relative to all meat (which is 64.4 g/day for 1-2-year-old children, 79.9 g/day for 2-3-year-old children, and 95 g/day for 3-6-year-old children) was not considered as a significant source of uncertainty from double counting meat intake.

Results

Table 5-2 and Figures 5-1, 5-2, and 5-3 show the results of this pathway (not including fish results, which are presented in Appendix 5 and Table 5-1) for the lead benchmarks of P3, P5, and P8, respectively, for all 588 residential DUs. Of 588 residential DUs evaluated (not including residential beach DUs, CCT tribal allotments, driplines, or a DU that was used as a potential ore stockpile), 389 of the 588 residential DUs evaluated exceeded P3 (66%), 87 exceeded P5 (15%), and 12 exceeded P8 (2%) as shown in Table 5-2, Table 5-3, and Figures 5-1, 5-2, and 5-3. Because the exposure assumptions were for full-time residential use, application of this exposure scenario to DUs with less frequent use than residences or play areas may overestimate actual exposures.

As shown in Appendix 5, the consideration of UCR fish consumption changed the results at some DUs, suggesting that consuming fish from the UCR may pose a small (relative to lead intake from soil and sediment) additional lead risk to some residents. Consumption of sucker from the UCR would result in the highest exposure to lead (Table 5-1). As shown in Table 5-4, the concentration of lead in sucker was at least 5 times higher than other species.

5.1.1.2 Future Child Resident Exposed to Lead in Upland Soil with and without Fish Consumption

Methodology

This exposure pathway was assessed using the IEUBK model to calculate risk to potential future child residents from exposure to lead in Site air, upland ADA soil/dust, and fish (assessed by species separately) caught and consumed from the UCR. Model runs were done with and without fish consumption. Batch mode was used to assess risk for this pathway using the IEUBK model (see section 3.3.1 for more information) on an ADA-by-ADA basis. The receptor population includes all potential future child residents. Arithmetic mean values for the ADA IC soil results for each ADA sampled in triplicate, or the single result for ADAs where a single IC sample was collected, were used as the soil lead EPC for the batch mode calculation. This soil lead EPC was the RBA-adjusted lead result (see Section 4.6 and Appendix 11; EPA [2020]) to allow the default RBA for soil to be retained in the IEUBK model batch mode analysis. Dust lead concentration was the MSD-based result from the RBA-adjusted soil lead EPC for the same reason (the MSD approach included the contribution from outdoor air lead). The outdoor air lead concentration was based on Site-specific information as described in Table 3-30. As discussed in Section 5.1.1, the results for the 32-month-old child was used as a surrogate for hypothetical children 12-72 months old in potential future residences.

Because fish consumption resulted in lead intake <1 µg/day (as shown in Table 5-1), risk for this receptor was assessed both with and without consumption of fish from the UCR. Risk was calculated using lead intake from consuming fish as an alternate intake in the IEUBK model (using the individual fish species average lead concentration and the DCR for fish from the CCT Tribal Survey [SRC, 2019b; IOM, 2005; see Appendix 14]). The alternate intake of lead from fish consumption was calculated as follows: lead concentration in the fish species (µg Pb/g fish) × 4.2 g fish/day = lead intake for each fish species (µg Pb/day). Because lead in fish is dietary lead, an absorption fraction of 50% was used in the IEUBK model for this alternate intake. The small amount of fish consumed (4.2 g/day) relative to all meat (which is 64.4 g/day for 1-2-year-old children, 79.9 g/day for 2-3-year-old children, and 95 g/day for 3-6-year-old children) was not considered as a significant source of uncertainty from double counting meat intake.

Results

Table 5-5 and Figures 5-4, 5-5, and 5-6 show the results of this pathway (not including fish results, which are presented in Appendix 5 and Table 5-1) for the lead benchmarks of P3, P5, and P8, respectively. Of 142 upland ADAs evaluated, 139 ADAs exceeded P3 (98%), 68 ADAs exceeded P5 (48%), and 15 ADAs exceeded P8 (11%) as shown in Table 5-5 and Figures 5-4 through 5-6. Because the exposure assumptions were for full-time residential use, application to ADAs that are not currently residences may overestimate actual exposures, as other less frequent exposure scenarios may be expected to produce lower PbBs and estimates of P3, P5, and P8.

As shown in Appendix 5, the consideration of UCR fish consumption changed the results at some ADAs, suggesting that consuming fish from the UCR may pose a small (relative to lead intake from soil and sediment) additional lead risk to future residents. Consumption of sucker from the UCR would result in the highest exposure to lead (Table 5-1). As shown in Table 5-4, the concentration of lead in sucker was at least 5 time higher than other species.

5.1.1.3 Current Child Exposure to Lead at Residential Beaches with and without Fish Consumption

Methodology

Exposure of child residents to lead while spending time recreating at beaches on their property was evaluated assuming the child plays in sediment at the beach, swims in the UCR, and consumes fish from the UCR. This exposure pathway was analyzed using the IEUBK model (see Section 3.3.1 for more information) to assess risk from Site air, Site surface water, fish caught and consumed from the UCR, and residential soil and beach sediment which is represented in the IEUBK model by a time-weighted combination of residential house DU soil and residential beach DU sediment from the 2014 and 2016 residential soil studies.

The time weighting of residential soil and beach sediment intake was based on an assumed beach exposure of 2 days/week (adjusted from the RecUse Survey data for swimming during beach visits [5.9 days/year]; SRC, 2019a). As discussed in Section 6.2.1.9 and Appendix 15, this adjustment is unlikely to significantly impact predicted PbB from the results of the All Ages Lead Model (AALM) and is a health-protective assumption. Because residential beaches are close to the residence, track-in of sediment from the beach was assumed and the MSD-dust lead concentration was derived from the time-weighted soil and sediment concentration. The time-weighting approach recommended by EPA (2003a) is to time-weight the exposure concentrations from the various locations where exposure occurs to derive an input for soil to the IEUBK model. In this instance, the residential beach exposure was time-weighted with the associated house DU for that parcel or the "surrogate" house DU. Table 5-6[85] and Appendix 13 show the residential house DU or DUs that were used for time weighting (including when a surrogate was used because some residential beaches did not have a house DU on the property).

The soil value input in the IEUBK model, derived from beach sediment and house soil concentrations, was calculated as follows:

Weighted Lead Conc. = House DU conc. (5/7) + Beach DU conc. (2/7)

The time-weighted lead concentration value was then entered into the IEUBK model (the RBA adjusted lead concentrations were used for the calculations so that the default RBA in the IEUBK model could be retained) using batch mode. The additional ingestion due to increased adherence of sediment was not considered, because a larger particle size fraction was used for sediment (than the particle size fraction used for soil, <250 µm vs <149 µm, respectively) to reflect increased adherence of sediment as compared to soil (see Appendix 13).

The outdoor air lead concentration was based on Site-specific data as specified in Table 3-30. As discussed in Section 5.1.1, the results for the 32-month-old child were used as a surrogate for hypothetical children 12-72 months old for all potential future residences.

Incidental ingestion of surface water while swimming at a residential beach and consumption of fish from the UCR were added as an additional intake of lead. The average Site-wide concentration of lead in disturbed surface water was multiplied by the number of hours spent

[85] In Table 5-6, the row for Beach DU 411 is shaded; this is because it is a CCT tribal allotment and was evaluated in Appendix 1 only.

swimming per day and volume of water consumed while swimming per hour from the RecUse Survey (SRC, 2019a). This intake of lead from incidental ingestion of disturbed surface water while swimming was added to the intake of lead from consuming fish from the UCR.

Because fish consumption resulted in lead intake <1 µg/day (as shown in Table 5-1), risk for this receptor was assessed both with and without consumption of fish from the UCR. Risk was calculated using lead intake from consuming fish as an alternate intake in the IEUBK model (using the individual fish species average lead concentration and the DCR for fish from the CCT Tribal Survey [SRC, 2019b; IOM, 2005; see Appendix 14]). The alternate intake of lead from fish consumption was calculated as follows: lead concentration in the fish species (µg Pb/g fish) × 4.2 g fish/day = lead intake for each fish species (µg Pb/day). An absorption fraction of 50% was used for the additional intake of surface water while swimming and fish.

Because lead in fish is dietary lead, a default absorption fraction of 50% was used in the IEUBK model for this alternate intake. The small amount of fish consumed (4.2 g/day) relative to all meat (which is 64.4 g/day for 1-2-year-old children, 79.9 g/day for 2-3-year-old children, and 95 g/day for 3-6-year-old children) was not considered as a significant source of uncertainty from double counting meat intake. Because the volume of water incidentally consumed during swimming is small and occurs during an active event, it is unlikely to impact drinking water intake so double counting of water intake is not likely to be a significant source of uncertainty.

Results

Table 5-7 shows the results for this exposure pathway (not including fish results, which are presented in Appendix 5 and Table 5-1) for P3, P5, and P8 (Figure 5-7). As shown, incidental ingestion of sediment and surface water at residential beaches 2 days/week exceeded the P3 risk benchmark at 19 of 21 residential beaches. Incidental ingestion of sediment and surface water at residential beaches 2 days/week exceeded the P5 risk benchmark at 5 of 21 residential beaches. Incidental ingestion sediment and surface water at residential beaches 2 days/week did not exceed the P8 risk benchmark at any residential beaches. As shown in Appendix 5, the consideration of UCR fish intake did not change the results at any residential beaches because the contribution of lead from consuming local fish is small relative to the contribution from soil and sediment exposure. As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

5.1.1.4 Child Recreational Visitor Exposed to Lead during a Beach Day Trip to a Public Beach from Sediment and UCR Surface Water while Swimming with and without Consumption of UCR Fish

Methodology

The exposure pathway was analyzed using the IEUBK model to assess risk from Site air, Site surface water, and a time-weighted combination of an assumed residential house soil lead concentration of 129 ppm (see Table 3-16) and public beach DU sediment for solid media exposure. The time weighting of solid intake used an assumed beach exposure of 1 day/week (adjusted from the RecUse Survey data for swimming during beach day trips; SRC, 2019a). As discussed in Section 6, this adjustment is unlikely to significantly impact predicted PbB from the results of the AALM and is a health protective assumption. Because public beaches are not necessarily located close to the residence, track-in of sediment from the beach was not assumed

and the MSD-dust lead concentration was derived from the assumed residential soil lead concentration (129 ppm). Incidental ingestion of surface water while swimming at a public beach was added as an additional intake of water (where the average Site-wide concentration of lead in disturbed surface water was multiplied by the number of hours spent swimming per day during beach day trips [0.98 hours/day] from the RecUse Survey [SRC, 2019a] and volume of water consumed while swimming per hour [40 mL/hour] from EPA, 2019b). The EPC for surface water was the Site-wide disturbed surface water EPC (0.003151 µg Pb/mL). An absorption fraction of 50% was used for the additional intake of surface water while swimming. Because this volume is small and occurs during an active event, it is unlikely to impact drinking water intake so double counting of water intake is not likely to be an issue.

The results of the RecUse Survey (SRC, 2019a) for time spent recreating at public beaches (5.9 days/season) fails to meet the minima for exposure frequency and duration required by the IEUBK model (1 day/week for 13 consecutive weeks; EPA, 2003a). Rather than not assess this pathway, the HHRA assumed an exposure frequency of 13 days recreating over a 91-day exposure season. This adjustment from 5.9 exposure days/season based on the RecUse Survey (SRC, 2019a) to 13 exposure days/season to meet the IEUBK model minimal exposure requirements (EPA, 2003a) is more than a two-fold increase in the Site-specific exposure frequency and is closer to the RME than the CTE; however, this adjustment does not significantly impact predicted blood lead concentration (see Section 6 and Appendix 15 for more information).

Because fish consumption resulted in lead intake <1 µg/day for recreational visitors on beach day trips (as shown in Table 5-1), risk for this receptor was assessed both with and without consumption of fish from the UCR. Risk was calculated using lead intake from consuming fish as an alternate intake in the IEUBK model (added to the incidental ingestion of surface water while swimming intake). Because both of these exposures (lead in surface water and lead in fish) have the same absorption fraction (50%), this was easily accommodated. Lead exposure from consuming fish from the UCR was assessed by individual species, since anglers may target specific fish species. The intake was the EPC for fish multiplied by the DCR for fish from the RecUse Survey (SRC, 2019a). Because the DCR for fish is small (3.2 g fish/day) relative to all meat (which is 64.4 g/day for 1-2-year-old children, 79.9 g/day for 2-3-year-old children, and 95 g/day for 3-5-year-old children), it was not considered as a significant source of uncertainty from double counting meat intake.

Results, without Consumption of Fish

Table 5-8 and Figure 5-8 show the results for this exposure pathway for P3, P5, and P8. As shown, incidental ingestion of sediment and surface water while recreating at public beaches exceeded the P3 risk benchmark at all public beaches. Incidental ingestion of sediment and surface water while recreating at public beaches exceeded the P5 risk benchmark only at Bossburg Flat Beach (based on 2011 sampling and 2013 reanalysis). Incidental ingestion of sediment and surface water while recreating at public beaches did not exceed the P8 risk benchmark at any public beach.

Results, including Consumption of Fish

Results for this receptor that include consumption of fish from the UCR (by species) are presented in Appendix 5 and Table 5-1. These results are similar to the results for this pathway without consumption of fish from the UCR, suggesting that fish consumption is not a major contributor to overall lead risk to recreational visitors to public beaches who swim during beach day trips to public beaches. As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

5.1.1.5 Child Recreational Visitor Exposed to Lead during a Boating Trip at a Public Beach from Sediment and UCR Surface Water while Swimming with and without Consumption of UCR Fish

Methodology

The exposure pathway was analyzed using the IEUBK model to assess risk from Site air, Site surface water, and a time-weighted combination of an assumed residential house soil lead concentration of 129 ppm (see Table 3-16) and public beach DU sediment for solid media exposure. The time weighting of solid intake used an assumed beach exposure of 1 day/week (adjusted from the RecUse Survey data for swimming during boating trips; SRC, 2019a). As discussed in Section 6, this adjustment is unlikely to significantly impact predicted PbB from the results of the AALM and is a health protective assumption. Because public beaches are not necessarily located close to the residence, track-in of sediment from the beach was not assumed and the MSD-dust lead concentration was derived from the assumed residential soil lead concentration (129 ppm). Incidental ingestion of surface water while swimming at a public beach during a boating trip was added as an additional intake of water (where the average Site-wide concentration of lead in disturbed surface water was multiplied by the number of hours spent swimming per day during boating trips [0.79 hours/day] from the RecUse Survey [SRC, 2019a] and volume of water consumed while swimming per hour [40 mL/hour] from EPA, 2019b). The EPC for surface water was the Site-wide disturbed surface water EPC (0.003151 µg Pb/mL). An absorption fraction of 50% was used for the additional intake of surface water while swimming. Because this volume is small and occurs during an active event, it is unlikely to impact drinking water intake so double counting of water intake is not likely to be an issue.

The results of the RecUse Survey (SRC, 2019a) for time spent recreating at public beaches (5.9 days/season) fails to meet the minima for exposure frequency and duration required by the IEUBK model (1 day/week for 13 consecutive weeks; EPA, 2003a). Rather than not assess this pathway, the HHRA assumed an exposure frequency of 13 days recreating over a 91-day exposure season. This adjustment from 5.9 exposure days/season based on the RecUse Survey (SRC, 2019a) to 13 exposure days/season to meet the IEUBK model minimal exposure requirements (EPA, 2003a) is more than a two-fold increase in the Site-specific exposure frequency and is closer to the RME than the CTE; however, this adjustment does not significantly impact predicted blood lead concentration (see Section 6 and Appendix 15 for more information).

Because fish consumption resulted in lead intake <1 µg/day for recreational visitors during boating trips (as shown in Table 5-1), risk for this receptor was assessed both with and without consumption of fish from the UCR. Risk was calculated using lead intake from consuming fish as an alternate intake in the IEUBK model (added to the incidental ingestion of surface water while swimming intake). Because both of these exposures (lead in surface water and lead in fish)

have the same absorption fraction (50%), this was easily accommodated. Lead exposure from consuming fish from the UCR was assessed by individual species, since anglers may target specific fish species. The intake was the EPC for fish multiplied by the DCR for fish from the RecUse Survey (SRC, 2019a). Because the DCR for fish is small (3.2 g fish/day) relative to all meat (which is 64.4 g/day for 1-2-year-old children, 79.9 g/day for 2-3-year-old children, and 95 g/day for 3-6-year-old children), it was not considered as a significant source of uncertainty from double counting meat intake.

Results without Fish Consumption

Table 5-8 and Figure 5-8 show the results for this exposure pathway for P3, P5, and P8. As shown, incidental ingestion of sediment and surface water during a boating trip at public beaches exceeded the P3 risk benchmark at all public beaches. Incidental ingestion of sediment and surface water during a boating trip exceeded the P5 risk benchmark only at Bossburg Flat (based on 2011 sampling and 2013 reanalysis). Incidental ingestion of sediment and surface water during a boating trip did not exceed the P8 risk benchmark at any public beach.

Results including Fish Consumption

Results for this receptor that include consumption of fish from the UCR (by species) are presented in Appendix 5 and Table 5-1. These results are similar to the results for this pathway without consumption of fish from the UCR, suggesting that fish consumption is not a major contributor to overall lead risk to recreational visitors to public beaches who swim during boating trips to public beaches. As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

5.1.1.6 Child Recreational Visitor Exposed to Lead during a Camping Trip at a Public Beach from Sediment and UCR Surface Water with and without Consumption of UCR Fish

Methodology

The exposure pathway was analyzed using the IEUBK model to assess risk from Site air, Site surface water, and a time-weighted combination of an assumed residential house soil lead concentration of 129 ppm (see Table 3-16) and public beach DU sediment for solid media exposure. The time weighting of solid intake used an assumed beach exposure of 1 day/week (adjusted from the RecUse Survey data for swimming during camping trips; SRC, 2019a). As discussed in Section 6, this adjustment is unlikely to significantly impact predicted PbB from the results of the AALM and is a health protective assumption. Because public beaches are not necessarily located close to the residence, track-in of sediment from the beach was not assumed and the MSD-dust lead concentration was derived from the assumed residential soil lead concentration (129 ppm). Incidental ingestion of surface water while swimming at a public beach during a camping trip was added as an additional intake of water (where the average Site-wide concentration of lead in disturbed surface water was multiplied by the number of hours spent swimming per day during camping trips [1.8 hours/day] from the RecUse Survey [SRC, 2019a] and volume of water consumed while swimming per hour [40 mL/hour] from EPA, 2019b). The EPC for surface water was the Site-wide disturbed surface water EPC (0.003151 µg Pb/mL). An absorption fraction of 50% was used for the additional intake of surface water while swimming. Because this volume is small and occurs during an active event, it is unlikely to impact drinking water intake so double counting of water intake is not likely to be an issue.

The results of the RecUse Survey (SRC, 2019a) for time spent recreating at public beaches (5.9 days/season) fails to meet the minima for exposure frequency and duration required by the IEUBK model (1 day/week for 13 consecutive weeks; EPA, 2003a). Rather than not assess this pathway, the HHRA assumed an exposure frequency of 13 days recreating over a 91-day exposure season. This adjustment from 5.9 exposure days/season based on the RecUse Survey (SRC, 2019a) to 13 exposure days/season to meet the IEUBK model minimal exposure requirements (EPA, 2003a) is more than a two-fold increase in the Site-specific exposure frequency and is closer to the RME than the CTE; however, this adjustment does not significantly impact predicted blood lead concentration (see Section 6 and Appendix 15 for more information).

Because fish consumption resulted in lead intake <1 µg/day for recreational visitors during camping trips (as shown in Table 5-1), risk for this receptor was assessed both with and without consumption of fish from the UCR. Risk was calculated using lead intake from consuming fish as an alternate intake in the IEUBK model (added to the incidental ingestion of surface water while swimming intake). Because both of these exposures (lead in surface water and lead in fish) have the same absorption fraction (50%), this was easily accommodated. Lead exposure from consuming fish from the UCR was assessed by species, since anglers may target specific fish species. The intake was the EPC for fish multiplied by the DCR for fish from the RecUse Survey (SRC, 2019a). Because the DCR for fish is small (3.2 g fish/day) relative to all meat (which is 64.4 g/day for 1-2-year-old children, 79.9 g/day for 2-3-year-old children, and 95 g/day for 3-6-year-old children), it was not considered as a significant source of uncertainty from double counting meat intake.

Results without Fish Consumption

Table 5-8 and Figure 5-8 show the results for this exposure pathway for P3, P5, and P8. As shown, incidental ingestion of sediment and surface water while swimming during a camping trip at public beaches exceeded the P3 risk benchmark at all public beaches. Incidental ingestion of sediment and surface water while swimming at public beaches during a camping trip exceeded the P5 risk benchmark only at Bossburg Flat (based on 2011 sampling and 2013 reanalysis). Incidental ingestion of sediment and surface water while swimming at public beaches during a camping trip did not exceed the P8 risk benchmark at any public beach.

Results including Fish Consumption

Results for this receptor that include consumption of fish from the UCR (by species) are presented in Appendix 5 and Table 5-1. These results are similar to the results for this pathway without consumption of fish from the UCR, suggesting that fish consumption is not a major contributor to overall lead risk to recreational visitors to public beaches who swim during camping trips to public beaches. As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

5.1.1.7 Child Recreational Visitor Exposed to Lead during a Bossburg Flat Beach Day Trip from Beach Soil and Surface Water while Swimming with and without Fish Consumption

Methodology

The exposure pathway was analyzed using the IEUBK model to assess risk from Site air, Site surface water, and a time-weighted combination of an assumed residential house soil lead concentration of 129 ppm (see Table 3-16) and Bossburg Flat UDU soil for solid media exposure. The time weighting of solid intake used an assumed beach exposure of 1 day/week (adjusted from the RecUse Survey data for swimming during beach day trips; SRC, 2019a). As discussed in Section 6, this adjustment is unlikely to significantly impact predicted PbB from the results of the AALM and is a health protective assumption. Because Bossburg Flat UDUs are not necessarily located close to the residence, track-in of soil from the beach was not assumed and the MSD-dust lead concentration was derived from the assumed residential soil lead concentration (129 ppm). Incidental ingestion of surface water while swimming by beachgoers at UDUs was added as an additional intake of water (where the average Site-wide concentration of lead in disturbed surface water was multiplied by the number of hours spent swimming per day during a beach day trip [0.98 hours/day] from the RecUse Survey [SRC, 2019a] and volume of water consumed while swimming per hour [40 mL/hour] from EPA, 2019b). The EPC for surface water was the Site-wide disturbed surface water EPC (0.003151 µg Pb/mL). An absorption fraction of 50% was used for the additional intake of surface water while swimming. Because this volume is small and occurs during an active event, it is unlikely to impact drinking water intake so double counting of water intake is not likely to be an issue.

The results of the RecUse Survey (SRC, 2019a) for time spent recreating at public beaches (5.9 days/season) fails to meet the minima for exposure frequency and duration required by the IEUBK model (1 day/week for 13 consecutive weeks; EPA, 2003a). Rather than not assess this pathway, the HHRA assumed an exposure frequency of 13 days recreating over a 91-day exposure season. This adjustment from 5.9 exposure days/season based on the RecUse Survey (SRC, 2019a) to 13 exposure days/season to meet the IEUBK model minimal exposure requirements (EPA, 2003a) is more than a two-fold increase in the Site-specific exposure frequency and is closer to the RME than the CTE; however, this adjustment does not significantly impact predicted blood lead concentration (see Section 6 and Appendix 15 for more information).

Because fish consumption resulted in lead intake <1 µg/day for recreational visitors during beach day trips (as shown in Table 5-1), risk for this receptor was assessed both with and without consumption of fish from the UCR. Risk was calculated using lead intake from consuming fish as an alternate intake in the IEUBK model (added to the incidental ingestion of surface water while swimming intake). Because both exposures (lead in surface water and lead in fish) have the same absorption fraction (50%), this was easily accommodated. Lead exposure from consuming fish from the UCR was assessed by individual species, since anglers may target specific fish species. The intake was the EPC for fish multiplied by the DCR for fish from the RecUse Survey (SRC, 2019a). Because the DCR for fish is small (3.2 g fish/day) relative to all meat (which is 64.4 g/day for 1-2-year-old children, 79.9 g/day for 2-3-year-old children, and 95 g/day for 3-6-year-old children), it was not considered as a significant source of uncertainty from double counting meat intake.

Results without Fish Consumption

Table 5-9 shows the results for this exposure pathway for P3, P5, and P8. As shown, incidental ingestion of soil and surface water while swimming during a beach day trip to upland portions of

Bossburg Flat Beach exceeded the P3 risk benchmark at all UDUs. Incidental ingestion of soil and surface water while swimming at Bossburg Flat UDUs during a beach day trip exceeded the P5 risk benchmark and the P8 risk benchmark only at UDU-04 (Figure 2-6).

Results including Fish Consumption

Results for this receptor that include consumption of fish from the UCR (by species) are presented in Appendix 5 and Table 5-1. These results are similar to the results for this pathway without consumption of fish from the UCR, suggesting that fish consumption is not a major contributor to overall lead risk to recreational visitors to public beaches who swim during beach day trips to public beaches, suggesting that fish consumption is not a major contributor to overall lead risk to recreational visitors who swim during beach day trips to Bossburg Flat UDUs. As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

5.1.1.8 Child Recreational Visitor Exposed to Lead during a Boating Trip at Bossburg Flat Beach from Beach Soil and UCR Surface Water while Swimming with and without Fish Consumption

Methodology

The exposure pathway was analyzed using the IEUBK model to assess risk from Site air, Site surface water, and a time-weighted combination of an assumed residential house soil lead concentration of 129 ppm (see Table 3-16) and Bossburg Flat UDU soil concentrations for solid media exposure. The time weighting of solid intake used an assumed beach exposure of 1 day/week (adjusted from the RecUse Survey data for swimming during boat trips; SRC, 2019a). As discussed in Section 6, this adjustment is unlikely to significantly impact predicted PbB from the results of the AALM and is a health protective assumption. Because Bossburg Flat UDUs are not necessarily located close to the residence, track-in of soil from the beach was not assumed and the MSD-dust lead concentration was derived from the assumed residential soil lead concentration (129 ppm). Incidental ingestion of surface water while swimming by boaters at Bossburg Flat UDUs was added as an additional intake of water (where the average Site-wide concentration of lead in disturbed surface water was multiplied by the number of hours spent swimming per day for boaters [0.79 hours/day] from the RecUse Survey [SRC, 2019a] and volume of water consumed while swimming per hour [40 mL/hour] from EPA, 2019b). The EPC for surface water was the Site-wide disturbed surface water EPC (0.003151 µg Pb/mL). An absorption fraction of 50% was used for the additional intake of surface water while swimming. Because this volume is small and occurs during an active event, it is unlikely to impact drinking water intake so double counting of water intake is not likely to be an issue.

The results of the RecUse Survey (SRC, 2019a) for time spent recreating at public beaches (5.9 days/season) fails to meet the minima for exposure frequency and duration required by the IEUBK model (1 day/week for 13 consecutive weeks; EPA, 2003a). Rather than not assess this pathway, the HHRA assumed an exposure frequency of 13 days recreating over a 91-day exposure season. This adjustment from 5.9 exposure days/season based on the RecUse Survey (SRC, 2019a) to 13 exposure days/season to meet the IEUBK model minimal exposure requirements (EPA, 2003a) is more than a two-fold increase in the Site-specific exposure frequency and is closer to the RME than the CTE; however, this adjustment does not

significantly impact predicted blood lead concentration (see Section 6 and Appendix 15 for more information).

Because fish consumption resulted in lead intake <1 µg/day for recreational visitors during boat trips (as shown in Table 5-1), risk for this receptor was assessed both with and without consumption of fish from the UCR. Risk was calculated using lead intake from consuming fish as an alternate intake in the IEUBK model (added to the incidental ingestion of surface water while swimming intake). Because both of these exposures (lead in surface water and lead in fish) have the same absorption fraction (50%), this was easily accommodated. Lead exposure from consuming fish from the UCR was assessed by individual species, since anglers may target specific fish species. The intake was the EPC for fish multiplied by the DCR for fish from the RecUse Survey (SRC, 2019a). Because the DCR for fish is small (3.2 g fish/per day) relative to all meat (which is 64.4 g/day for 1-2-year-old children, 79.9 g/day for 2-3-year-old children, and 95 g/day for 3-6-year-old children), it was not considered as a significant source of uncertainty from double counting meat intake.

Results without Fish Consumption

Table 5-9 shows the results for this exposure pathway for P3, P5, and P8. As shown, incidental ingestion of soil and surface water while swimming during a boat trip to Bossburg Flat UDUs exceeded the P3 risk benchmark at all UDUs. Incidental ingestion of soil and surface water while swimming at UDUs during a boat trip exceeded the P5 risk benchmark and the P8 risk benchmark only at UDU-04.

Results including Fish Consumption

Results for this receptor that include consumption of fish from the UCR (by species) are presented in Appendix 5 and Table 5-1. These results are similar to the results for this pathway without consumption of fish from the UCR, suggesting that fish consumption is not a major contributor to overall lead risk to recreational visitors who swim during boat trips to Bossburg Flat UDUs. As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

5.1.1.9 Child Recreational Visitor Exposed to Lead during a Camping Trip at Bossburg Flat Beach from Beach Soil and Surface Water While Swimming with and without Fish Consumption

Methodology

The exposure pathway was analyzed using the IEUBK model to assess risk from Site air, Site surface water, and a time-weighted combination of an assumed residential house soil lead concentration of 129 ppm (see Table 3-16) and Bossburg Flat UDU soil concentrations for solid media exposure. The time weighting of solid intake used an assumed beach exposure of 1 day/week (adjusted from the RecUse Survey data for swimming during camping trips; SRC, 2019a). As discussed in Section 6, this adjustment is unlikely to significantly impact predicted PbB from the results of the AALM and is a health protective assumption. Because Bossburg Flat UDUs are not necessarily located close to the residence, track-in of soil from the beach was not assumed and the MSD-dust lead concentration was derived from the assumed residential soil lead concentration (129 ppm). Incidental ingestion of surface water while swimming by campers at Bossburg Flat UDUs was added as an additional intake of water (where the average Site-wide

concentration of lead in disturbed surface water was multiplied by the number of hours spent swimming per day during camping trips [1.8 hours/day] from the RecUse Survey [SRC, 2019a] and volume of water consumed while swimming per hour [40 mL/hour] from EPA, 2019b). The EPC for surface water was the Site-wide disturbed surface water EPC (0.003151 µg Pb/mL). An absorption fraction of 50% was used for the additional intake of surface water while swimming. Because this volume is small and occurs during an active event, it is unlikely to impact drinking water intake so double counting of water intake is not likely to be an issue.

The results of the RecUse Survey (SRC, 2019a) for time spent recreating at public beaches (5.9 days/season) fails to meet the minima for exposure frequency and duration required by the IEUBK model (1 day/week for 13 consecutive weeks; EPA, 2003a). Rather than not assess this pathway, the HHRA assumed an exposure frequency of 13 days recreating over a 91-day exposure season. This adjustment from 5.9 exposure days/season based on the RecUse Survey (SRC, 2019a) to 13 exposure days/season to meet the IEUBK model minimal exposure requirements (EPA, 2003a) is more than a two-fold increase in the Site-specific exposure frequency and is closer to the RME than the CTE; however, this adjustment does not significantly impact predicted blood lead concentration (see Section 6 and Appendix 15 for more information).

Because fish consumption resulted in lead intake <1 µg/day for recreational visitors during camping trips (as shown in Table 5-1), risk for this receptor was assessed both with and without consumption of fish from the UCR. Risk was calculated using lead intake from consuming fish as an alternate intake in the IEUBK model (added to the incidental ingestion of surface water while swimming intake). Because both of these exposures (lead in surface water and lead in fish) have the same absorption fraction (50%), this was easily accommodated. Lead exposure from consuming fish from the UCR was assessed by individual species, since anglers may target specific fish species. The intake was the EPC for fish multiplied by the DCR for fish from the RecUse Survey (SRC, 2019a). Because the DCR for fish is small (3.2 g fish/day) relative to all meat (which is 64.4 g/day for 1-2-year-old children, 79.9 g/day for 2-3-year-old children, and 95 g/day for 3-6-year-old children), it was not considered as a significant source of uncertainty from double counting meat intake.

Results without Fish Consumption

Table 5-9 shows the results for this exposure pathway for P3, P5, and P8. As shown, incidental ingestion of soil and surface water while swimming during a camping trip to Bossburg Flat UDUs exceeded the P3 risk benchmark at all UDUs. Incidental ingestion of soil and surface water while swimming at Bossburg Flat UDUs during a camping trip exceeded the P5 risk benchmark and the P8 risk benchmark only at UDU-04.

Results including Fish Consumption

Results for this receptor that include consumption of fish from the UCR (by species) are presented in Appendix 5 and Table 5-1. These results are similar to the results for this pathway without consumption of fish from the UCR, suggesting that fish consumption is not a major contributor to overall lead risk to recreational visitors who swim during camping trips to Bossburg Flat UDUs. As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

5.1.1.10 Child Recreational Visitor Exposed to Lead in Soil and Surface Water by Beachgoers who Swim at Relict Floodplains along the UCR with and without Fish Consumption

Methodology

The exposure pathway was analyzed using the IEUBK model to assess risk from Site air, Site surface water, and a time-weighted combination of an assumed residential house soil lead concentration of 129 ppm (see Table 3-16) and relict floodplain soil for solid media exposure. The time weighting of solid intake used an assumed beach exposure of 1 day/week (adjusted from the RecUse Survey data for swimming during beach day trips; SRC, 2019a). As discussed in Section 6, this adjustment is unlikely to significantly impact predicted PbB from the results of the AALM and is a health protective assumption. Because relict floodplains are not necessarily located close to the residence, track-in of soil from the relict floodplain was not assumed and the MSD-dust lead concentration was derived from the assumed residential soil lead concentration (129 ppm). Incidental ingestion of surface water while swimming by beachgoers at relict floodplains was added as an additional intake of water (where the average Site-wide concentration of lead in disturbed surface water was multiplied by the number of hours spent swimming per day during beach day trips [0.98 hours/day] from the RecUse Survey [SRC, 2019a] and volume of water consumed while swimming per hour [40 mL/hour] from EPA, 2019b). The EPC for surface water was the Site-wide disturbed surface water EPC (0.003151 µg Pb/mL). An absorption fraction of 50% was used for the additional intake of surface water while swimming. Because this volume is small and occurs during an active event, it is unlikely to impact drinking water intake so double counting of water intake is not likely to be an issue.

The results of the RecUse Survey (SRC, 2019a) for time spent recreating at public beaches (5.9 days/season) fails to meet the minima for exposure frequency and duration required by the IEUBK model (1 day/week for 13 consecutive weeks; EPA, 2003a). Rather than not assess this pathway, the HHRA assumed an exposure frequency of 13 days recreating over a 91-day exposure season. This adjustment from 5.9 exposure days/season based on the RecUse Survey (SRC, 2019a) to 13 exposure days/season to meet the IEUBK model minimal exposure requirements (EPA, 2003a) is more than a two-fold increase in the Site-specific exposure frequency and is closer to the RME than the CTE; however, this adjustment does not significantly impact predicted blood lead concentration (see Section 6 and Appendix 15 for more information).

Because fish consumption resulted in lead intake <1 µg/day recreational visitors during beach day trips to relict floodplains (as shown in Table 5-1), risk for this receptor was assessed both with and without consumption of fish from the UCR. Risk was calculated using lead intake from consuming fish as an alternate intake in the IEUBK model (added to the incidental ingestion of surface water while swimming intake). Because both of these exposures (lead in surface water and lead in fish) have the same absorption fraction (50%), this was easily accommodated. Lead exposure from consuming fish from the UCR was assessed by individual species, since anglers may target specific fish species. The intake was the EPC for fish multiplied by the DCR for fish from the RecUse Survey (SRC, 2019a). Because the DCR for fish is small (3.2 g fish/day) relative to all meat (which is 64.4 g/day for 1-2-year-old children, 79.9 g/day for 2-3-year-old children, and 95 g/day for 3-6-year-old children), it was not considered as a significant source of uncertainty from double counting meat intake.

Results without Fish Consumption

Table 5-10 shows the results for this exposure pathway for P3, P5, and P8. As shown, incidental ingestion of soil and surface water while swimming during a beach day trip to relict floodplains exceeds the P3 risk benchmark at all relict floodplains. Incidental ingestion of soil and surface water while swimming at relict floodplains during a beach day trip did not exceed the P5 risk benchmark or the P8 risk benchmark at any of the relict floodplains.

Results including Fish Consumption

Results for this receptor that include consumption of fish from the UCR (by species) are presented in Appendix 5 and Table 5-1. These results are similar to the results for this pathway without consumption of fish from the UCR, suggesting that fish consumption is not a major contributor to overall lead risk to recreational visitors who swim during beach day trips to relict floodplains. As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

5.1.1.11 Child Recreational Visitor Exposed to Lead in Soil and Surface Water while Boating and Swimming at Relict Floodplains along the UCR with and without Fish Consumption

Methodology

The exposure pathway was analyzed using the IEUBK model to assess risk from Site air, Site surface water, and a time-weighted combination of an assumed residential house soil lead concentration of 129 ppm (see Table 3-16) and relict floodplain soil for solid media exposure. The time weighting of solid intake used an assumed beach exposure of 1 day/week (adjusted from the RecUse Survey data for swimming during boat trips; SRC, 2019a). As discussed in Section 6, this adjustment is unlikely to significantly impact predicted PbB from the results of the AALM and is a health-protective assumption. Because relict floodplains are not necessarily located close to the residence, track-in of soil from relict floodplains was not assumed and the MSD-dust lead concentration was derived from the assumed residential soil lead concentration (129 ppm). Incidental ingestion of surface water while swimming by boaters at relict floodplains was added as an additional intake of water (where the average Site-wide concentration of lead in disturbed surface water was multiplied by the number of hours spent swimming per day while boating [0.79 hours/day for boaters] from the RecUse Survey [SRC, 2019a] and volume of water consumed while swimming per hour [40 mL/hour] from EPA, 2019b). The EPC for surface water was the Site-wide disturbed surface water EPC (0.003151 µg Pb/mL). An absorption fraction of 50% was used for the additional intake of surface water while swimming. Because this volume is small and occurs during an active event, it is unlikely to impact drinking water intake so double counting of water intake is not likely to be an issue.

The results of the RecUse Survey (SRC, 2019a) for time spent recreating at public beaches (5.9 days/season) fails to meet the minima for exposure frequency and duration required by the IEUBK model (1 day/week for 13 consecutive weeks; EPA, 2003a). Rather than not assess this pathway, the HHRA assumed an exposure frequency of 13 days recreating over a 91-day exposure season. This adjustment from 5.9 exposure days/season based on the RecUse Survey (SRC, 2019a) to 13 exposure days/season to meet the IEUBK model minimal exposure requirements (EPA, 2003a) is more than a two-fold increase in the Site-specific exposure frequency and is closer to the RME than the CTE; however, this adjustment does not

significantly impact predicted blood lead concentration (see Section 6 and Appendix 15 for more information).

Because fish consumption resulted in lead intake <1 µg/day for recreational visitors during boat trips to relict floodplains (as shown in Table 5-1), risk for this receptor was assessed both with and without consumption of fish from the UCR. Risk was calculated using lead intake from consuming fish as an alternate intake in the IEUBK model (added to the incidental ingestion of surface water while swimming intake). Because both exposures (lead in surface water and lead in fish) have the same absorption fraction (50%), this was easily accommodated. Lead exposure from consuming fish from the UCR was assessed by individual species, since anglers may target specific fish species. The intake was the EPC for fish multiplied by the DCR for fish from the RecUse Survey (SRC, 2019a). Because the DCR for fish is small (3.2 g fish/day) relative to all meat (which is 64.4 g/day for 1-2-year-old children, 79.9 g/day for 2-3-year-old children, and 95 g/day for 3-6-year-old children), it was not considered as a significant source of uncertainty from double counting meat intake.

Results without Fish Consumption

Table 5-10 shows the results for this exposure pathway for P3, P5, and P8. As shown, incidental ingestion of soil and surface water while swimming during a boat trip to relict floodplains exceeded the P3 risk benchmark at all relict floodplains. Incidental ingestion of sediment and surface water while swimming at relict floodplains during a boat trip did not exceed the P5 risk benchmark or the P8 risk benchmark at any of the relict floodplains.

Results including Fish Consumption

Results for this receptor that include consumption of fish from the UCR (by species) are presented in Appendix 5 and Table 5-1. These results are similar to the results for this pathway without consumption of fish from the UCR, suggesting that fish consumption is not a major contributor to overall lead risk to recreational visitors who swim during boat trips to relict floodplains. As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

5.1.1.12 Exposure to Lead in Soil and Surface Water by Campers who Swim at Relict Floodplains along the UCR with and without Fish Consumption

Methodology

The exposure pathway was analyzed using the IEUBK model to assess risk from Site air, Site surface water, and a time-weighted combination of an assumed residential house soil lead concentration of 129 ppm (see Table 3-16) and relict floodplain soil for solid media exposure. The time weighting of solid intake used an assumed beach exposure of 1 day/week (adjusted from the RecUse Survey data for swimming during camping trips; SRC, 2019a). As discussed in Section 6, this adjustment is unlikely to significantly impact predicted PbB from the results of the AALM and is a health protective assumption. Because relict floodplains are not necessarily located close to the residence, track-in of soil from relict floodplains was not assumed and the MSD-dust lead concentration was derived from the assumed residential soil lead concentration (129 ppm). Incidental ingestion of surface water while swimming by campers at relict floodplains was added as an additional intake of water (where the average Site-wide concentration of lead in disturbed surface water was multiplied by the number of hours spent

swimming per day during camping trips [1.8 hours/day for campers] from the RecUse Survey [SRC, 2019a] and volume of water consumed while swimming per hour [40 mL/hour] from EPA, 2019b). The EPC for surface water was the Site-wide disturbed surface water EPC (0.003151 µg Pb/mL). An absorption fraction of 50% was used for the additional intake of surface water while swimming. Because this volume is small and occurs during an active event, it is unlikely to impact drinking water intake so double counting of water intake is not likely to be an issue.

The results of the RecUse Survey (SRC, 2019a) for time spent recreating at public beaches (5.9 days/season) fails to meet the minima for exposure frequency and duration required by the IEUBK model (1 day/week for 13 consecutive weeks; EPA, 2003a). Rather than not assess this pathway, the HHRA assumed an exposure frequency of 13 days recreating over a 91-day exposure season. This adjustment from 5.9 exposure days/season based on the RecUse Survey (SRC, 2019a) to 13 exposure days/season to meet the IEUBK model minimal exposure requirements (EPA, 2003a) is more than a two-fold increase in the Site-specific exposure frequency and is closer to the RME than the CTE; however, this adjustment does not significantly impact predicted blood lead concentration (see Section 6 and Appendix 15 for more information).

Because fish consumption resulted in lead intake <1 µg/day for recreational visitors during camping trips at relict floodplains (as shown in Table 5-1), risk for this receptor was assessed both with and without consumption of fish from the UCR. Risk was calculated using lead intake from consuming fish as an alternate intake in the IEUBK model (added to the incidental ingestion of surface water while swimming intake). Because both of these exposures (lead in surface water and lead in fish) have the same absorption fraction (50%), this was easily accommodated. Lead exposure from consuming fish from the UCR was assessed by individual species, since anglers may target specific fish species. The intake was the EPC multiplied by the DCR for fish from the RecUse Survey (SRC, 2019a). Because the DCR for fish is small (3.2 g fish/day) relative to all meat (which is 64.4 g/day for 1-2-year-old children, 79.9 g/day for 2-3-year-old children, and 95 g/day for 3-6-year-old children), it was not considered as a significant source of uncertainty from double counting meat intake.

Results without Fish Consumption

Table 5-10 shows the results for this exposure pathway for P3, P5, and P8. As shown, incidental ingestion of soil and surface water while swimming during a camping trip to relict floodplains exceeded the P3 risk benchmark at all relict floodplains. Incidental ingestion of soil and surface water while swimming at relict floodplains during a camping trip did not exceed the P5 risk benchmark or the P8 risk benchmark at any of the relict floodplains.

Results including Fish Consumption

Results for this receptor that include consumption of fish from the UCR (by species) are presented in Appendix 5 and Table 5-1. These results are similar to the results for this pathway without consumption of fish from the UCR, suggesting that fish consumption is not a major contributor to overall lead risk to recreational visitors who swim during camping trips to relict floodplains. As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

5.1.1.13 Exposure to Lead from Inhalation of Outdoor Air

As discussed in the footnote to Table 3-30, the concentration of lead in UCR air (CH2MHill, 2015) is an order of magnitude lower than the default air lead concentration in the IEUBK model and is not likely to influence the results. Additionally, EPA evaluated air lead concentration data measured at the Sheep Creek monitoring station near Northport. EPA has analyzed numerous studies that compared air lead concentration to PbBs in children.[86] The analysis indicates that the air-associated exposure from inhalation or ingestion of lead in house dust and surface soil can lead to a PbB that is 5 to 10 times the value in air. Based on air results at the Sheep Creek monitoring station during the 1999-2009 monitoring period, the lead concentration is 0.0242 µg/m^3 in air. This could lead to a 0.12 to 0.24 µg/dL level of lead in blood, which is low risk (EPA, 2018b). The EPC value used in this HHRA, for lead in air measured at Sheep Creek from 2002-2009, was 0.023 µg/m^3 (Table 3-30).

For lead, the air concentration from the Sheep Creek monitoring is well below the levels expected to pose significant human health risks based on current understanding of relationships between air lead concentrations and PbBs resulting from direct and indirect exposures to air lead (Appendix 8). As such, risk from lead in air was included in the risk calculations as an input to the IEUBK model for all evaluations; however, this pathway was not evaluated as a separate exposure for lead.

5.1.2 ALM Analyses for Adult Exposure Scenarios

Methodology: Adult Outdoor Worker Exposure to Lead in Subsurface Sediment and Soil at Public Beaches and Bossburg Flat Beach UDUs

The ALM was used for all analyses involving lead exposure where adults (and not children) contact contaminated media (EPA, 1996d). At the UCR Site, the only exposure pathway that is exclusive to adults is outdoor worker contact with exposure to deep sediment or beach soil (0-45 cm) during beach maintenance activities (i.e., digging holes, trenches, footings, etc.). All other outdoor workers are protected by assuming child exposure (children are the more sensitive receptor, so site decisions based on child exposure are protective for adults). The exposure frequency was assumed to be 219 days/year, which is from the ALM guidance (EPA, 1996d) and is equivalent to 54 days out of 90 days if the work were to occur during the summer months. The default variable values in the ALM (EPA, 1996d; updated to include NHANES information [EPA, 2017d] as shown on the TRW Lead Committee website[87]) were used to assess this exposure.

Results: Adult Outdoor Worker Exposure to Lead in Subsurface Sediment and Soil at Public Beaches and Bossburg Flat Beach UDUs

Table 5-11 shows the average RBA-adjusted lead concentration in sediment samples from 0 to 45 cm and the P3, P5, and P8 results for adults. As shown, incidental ingestion of deep sediment or soil by adult outdoor workers exceeded the P3 risk benchmark at only three exposure areas: F-01, UDU-01, and UDU-04, all located at Bossburg Flat Beach. Incidental ingestion of deep sediment by adult outdoor workers exceeded the P5 risk benchmark and the P8 risk benchmark

[86] Integrated Science Assessment (ISA) for Lead (https://www.epa.gov/isa/integrated-science-assessment-isa-lead); April 2017.
[87] See https://www.epa.gov/superfund/lead-superfund-sites-software-and-users-manuals#update.

at none of the public beaches or UDUs. Additional details of the ALM results are shown in Appendix 16.

5.2 Non-Lead COPCs

5.2.1 Non-Cancer Approach

Risk of non-cancer effects is evaluated by comparing the estimated exposure concentration over a specified time period to a toxicity reference value (threshold) that represents the exposure below which it is unlikely for sensitive populations to experience adverse health effects (EPA, 1989). This ratio of exposure to toxicity is called an HQ. To assess the overall potential for non-cancer effects posed by more than one chemical, or if exposure to a COPC occurs by more than one route, HQs are summed to yield a Hazard Index (HI). This approach assumes that simultaneous subthreshold exposures to several chemicals could result in an adverse health effect. It also assumes that the magnitude of the adverse effect is proportional to the sum of the ratios of the subthreshold exposures to acceptable exposures. Within an exposure pathway (e.g., incidental ingestion of soil), HQs were summed across COPCs to derive an HI. HQs were also summed across pathways (e.g., incidental ingestion of and dermal contact with soil) to derive an HI.

Non-cancer hazards were assessed against risk benchmarks. Risk benchmarks are risk levels that EPA uses to distinguish risks that are a potential concern from risks that are below the level of concern. The benchmark for non-cancer hazards used in this assessment was an HQ >1. If the HQ for a chemical is ≤1, there is no appreciable risk (e.g., the probability is close to zero that non-cancer health effects will occur). If an HQ is >1, then there is some possibility that an adverse non-cancer effect may occur (e.g., the probability is >0, but is likely not close to 1). This is because of the margin of safety inherent in the derivation of all toxicity values (see Section 4.3). However, the larger the HQ value, the more likely it is that an adverse effect may occur. When an HI is calculated, the risk benchmark is also HI >1. The HI can exceed one even if no single chemical exposure exceeds its RfD/RfC (EPA, 1989). This is likely to overestimate actual exposures.

The assumption of dose additivity in the HI approach is most relevant to chemicals that induce the same effect by the same mode of action. If an HI is >1 as a consequence of summing several HQs, then the chemicals can be segregated by effect and mode of action, and an HI can be calculated for each target organ group which is often more informative than simply summing across all non-cancer endpoints (EPA, 1989). Target organs were assigned to each COPC as shown in Tables 4-2 and 4-3, based on data in EPA (2019a). Target organ group HI calculation results are discussed below for receptors with HI >1.

In this assessment, the following terminology was used to categorize non-cancer hazard based on the HQ or HI. Risks were considered to be below non-cancer hazard benchmarks used by CERCLA if the HQ or HI ≤1; if the HQ or HI >1, there was a possibility that non-cancer health effects may occur (EPA, 1989, 1997; EPA and Clay, 1991). If an HI summed across COPCs within a pathway or summed across pathways was >1, target organ HIs were calculated. In this HHRA, calculated HQs and HIs were presented with one significant digit. The following are

examples of how calculated results were reported as one significant digit and then compared to the non-cancer benchmark of 1:

- Calculated result of 0.92 was reported as 0.9 – it does not exceed benchmark
- Calculated result of 0.96 was reported as 1 – it does not exceed benchmark
- Calculated result of 1.3 was reported as 1 – it does not exceed benchmark
- Calculated result of 1.5 was reported as 2 – it exceeds benchmark

Non-cancer HQs for each chemical were calculated as described below.

Ingestion Exposures

For most chemicals, the potential for non-cancer effects following ingestion exposure is evaluated by comparing the estimated daily intake of the chemical over a specific period with the RfD for that chemical derived for a similar exposure period, as follows (EPA, 1989):

$$HQ = DI / RfD$$

where:

DI = Daily intake (mg/kg-day)
RfD = Reference dose (mg/kg-day)

Dermal Exposures

For most chemicals, the potential for non-cancer effects following dermal exposure is evaluated by comparing the estimated absorbed dose of the chemical over a specific time period with the RfD for that chemical derived for a similar exposure period, as follows (EPA, 1989):

$$HQ = DAD / RfD_{abs}$$

where:

DAD = Dermally-absorbed dose (mg/kg-day)
RfD_{abs} = Absorbed reference dose (mg/kg-day)
$RfD_{abs} = RfD \cdot ABS_{GI}$ (gastrointestinal absorption fraction)

The ABS_{GI} term is unitless, chemical-specific, and is applied to the available oral toxicity values to account for the absorption efficiency of an administered dose across the GI tract and into the bloodstream.

Inhalation Exposures

For inhalation exposures, the potential for non-cancer effects is evaluated by comparing the time-weighted EC over a specific time period to the RfC for that chemical, as follows (EPA, 1994d):

$$HQ = EC / RfC$$

where:

EC = Exposure concentration ($\mu g/m^3$)
RfC = Reference concentration ($\mu g/m^3$)

5.2.2 Cancer Approach

The excess risk of cancer from exposure to a chemical is described in terms of the increased probability of developing cancer. Cancer risks are summed across all carcinogenic chemicals and all exposure pathways that contribute to exposure for a given population. If there is exposure to the same chemical in more than one medium, the total risk from that chemical is the sum of the risks across each medium. If exposure is to more than one chemical, the total cancer risk is estimated by summing the chemical-specific risks across all carcinogenic chemicals.

In general, EPA considers excess cancer risks below 10^{-6} to be negligible, and risks above 10^{-4} to warrant action.[88] Excess cancer risks that range between 10^{-6} and 10^{-4} are generally considered to be acceptable (EPA, 1989; EPA and Clay, 1991), although this is evaluated on a case-by-case basis. In this assessment, the following terminology was used to categorize cancer risks. Excess cancer risks that are $\leq 10^{-4}$ are within an acceptable range and risks above 10^{-4} are unacceptable (National Contingency Plan [NCP], 1990). As recommended in Exhibits 8-2 and 8-3 of EPA's *Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual, Part A* (EPA, 1989), calculated cancer risks retained more than one significant figure and are presented in summary tables shown to one significant figure.

Cancer risks for each chemical were calculated as described below.

<u>Ingestion Exposures</u>
The excess risk of cancer from ingestion exposure to a chemical is calculated as follows (EPA, 1989):

$$\text{Excess Cancer Risk} = 1 - \exp(-DI_L \cdot SF)$$

where:

DI_L = Daily Intake, averaged over a lifetime (mg/kg-day)
SF = Slope Factor $(mg/kg\text{-}day)^{-1}$

In most cases (except when the product of $DI_L \cdot SF$ is larger than about 0.01), this equation may be approximated by the following:

$$\text{Excess Cancer Risk} = DI_L \cdot SF$$

<u>Dermal Exposures</u>

[88] Note that excess cancer risk can be expressed in several formats. A cancer risk of 10^{-6} is equivalent to 1 in 1,000,000 and can be expressed in scientific notation format as 1E-06. Similarly, a cancer risk of 10^{-4} is equivalent to 1 in 10,000 or 1E-04. For the purposes of this document, all cancer risks are presented as 10^{-4}, 10^{-6}, etc.

The excess risk of cancer from dermal exposure to a chemical is calculated as follows (EPA, 2004b):

$$\text{Excess Cancer Risk} = DAD_L \cdot SF_{ABS}$$

where:

DAD_L = Dermally-Absorbed Dose, averaged over a lifetime (mg/kg-day)
SF_{ABS} = Absorbed Slope Factor $(mg/kg\text{-}day)^{-1}$
$SF_{ABS} = SF/ABS_{GI}$

Inhalation Exposures

The excess risk of cancer from inhalation exposure for COPCs is calculated based on IUR values as follows (EPA, 2009a):

$$\text{Excess Cancer Risk} = EC \cdot IUR$$

where:

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

5.2.2.1 Evaluation of PCB Mixtures

Because PCBs can cause cancer through both dioxin-like and non-dioxin-like modes of action, it is important to consider the contribution from both when estimating the total risk. In accordance with EPA guidance (EPA, 1996a, 2000), cancer risk from ingestion of dioxin-like PCB congeners was evaluated based on a TEQ approach (see Section 4.5.2 above) using the dioxin SF of 1.3E+05 $(mg/kg\text{-}day)^{-1}$. Cancer risk from non-dioxin-like PCB congeners was evaluated based on the high risk and persistence PCB upper-bound SF of 2.0 $(mg/kg\text{-}day)^{-1}$ (see Table 5-12). Dose estimates for non-dioxin-like PCBs were calculated two ways: 1) as the sum of detected non-dioxin-like PCB congener concentrations, and 2) as the sum of detected Aroclor concentrations.[89] The larger of the two values was used when summing to derive HIs across pathways.

5.2.3 Non-Cancer Hazard Summary

The results of the non-cancer evaluation of COPCs other than lead are summarized below for each exposure pathway described in Section 3.1.3.

5.2.3.1 Surface Soil/Indoor Dust: Current Residential Exposure to Non-Lead COPCs

The current adult and child resident population was assumed to be exposed to outdoor soil (via incidental ingestion and dermal contact) and indoor dust (via incidental ingestion) at all DUs sampled in the 2014 and 2016 residential soil studies (except residential beaches, CCT tribal

[89] Ideally, Aroclor concentrations would be adjusted to exclude the dioxin-like PCB congeners (these congeners are evaluated based on a TEQ approach). However, if PCB congener data are not available, this adjustment cannot be performed.

allotments [which are evaluated in Appendix 1], driplines, and a DU from 2016 that was identified as a former ore mill or stockpiling area[90]). An additional twenty-one residential DUs are beach DUs that were sampled on residential properties. Non-cancer hazards from exposure to non-lead COPCs in surface soil/indoor dust were evaluated separately for "non-beach" and "beach" DUs. Each is described separately below.

Incidental Ingestion of Residential Soil and Indoor Dust at Non-Beach DUs

For the current adult resident population evaluated using default exposure parameters at 588 residential non-beach DUs, all total soil/dust ingestion non-cancer hazards (summed across COPCs for each DU) were below one under both the RME and CTE scenarios (Tables 5-13 and 5-14). Total soil/dust ingestion non-cancer hazards for child residents (summed across COPCs) were >1 at 287 DUs under the RME scenario, 91 of which are currently house DUs and 10 of which are currently play area DUs. Under the CTE scenario, soil/dust ingestion non-cancer hazards for children (summed across COPCs) did not exceed 1 at any residential DU. Under RME exposure assumptions for children, the individual HQ for thallium was >1 for children at one house DU. No COPCs had individual HQs >1 using CTE exposure assumptions.

Dermal Contact with Residential Soil at Non-Beach DUs

Current residents were assumed to be exposed to non-lead COPCs in soil via dermal exposure at all DUs sampled in the 2014 and 2016 residential soil studies (except residential beaches, CCT tribal allotments, driplines, and DU 172-O1). For all receptors (adults and children), the total non-cancer hazard from dermal contact with soil under both RME and CTE scenarios did not exceed 1 (Tables 5-13 and 5-14).

Total Exposure to Residential Soil at Non-Beach DUs

When exposure to non-lead COPCs in residential soil for current residential use was summed across pathways (incidental ingestion of soil/dust and dermal contact with soil), total non-cancer HIs >1 at 315 DUs and ranged from 0.8 to 5 for current child residents at the RME, not considering target organ effects. Total non-cancer HIs did not exceed 1 for current child residents at the CTE and did not exceed 1 for adult residents at the RME and CTE (Tables 5-13 and 5-14). Chemicals that contributed the most to risk included arsenic, cobalt, iron, manganese, and thallium. Because HIs were >1 for total exposure to residential soil/dust (via ingestion and dermal contact) for current child residents, HIs calculated for each target organ group are presented in Tables 5-15 through 5-23 for the RME scenario and Tables 5-24 through 5-32 for the CTE scenario. One house DU under RME exposure assumptions exceeded an HI of 1 when aggregated by target organ group (skin/hair/nails target organ system; Figure 5-9) for soil and dust ingestion. For current child residents evaluated under CTE exposure assumptions, no target organ HIs >1. Based on an HI being >1 when evaluated by target organ system at the RME, there is potential for non-cancer health effects to children from non-lead COPCs through the total exposure to surface soil and indoor dust pathway.

[90] The field notes taken during the 2016 reconnaissance for the DU identified as a former small ore mill or ore stockpiling site (DU 172-O1) note that it is a flat, open area with a fire pit used for camping and for target practice. The property owner was not present during reconnaissance and no interview with the owner was conducted, though ECY conducted follow-up visual field inspection (Roland, 2019). As a result, there may be other sources of contamination besides those listed here. Because non-Site related COPCs associated with debris from target practice (broken clay pigeons, shotgun shells) and remains of the ore stockpiles may affect the risk estimates for this DU, the analytical results from this DU were not included in the risk assessment.

Incidental Ingestion of Residential Soil and Indoor Dust Associated with Beach DUs

For the current adult resident population evaluated using default exposure parameters for residential soil/indoor dust paired with 21 residential beach DUs, all total soil/dust ingestion non-cancer hazards (summed across COPCs for each DU) did not exceed 1 under both the RME and CTE scenarios (Tables 5-33 and 5-34). Total soil/dust ingestion non-cancer hazards for child residents (summed across COPCs) were >1 at 11 residential beach DUs under the RME scenario, not considering target organ effects. Under the CTE scenario, soil/dust ingestion non-cancer hazards for children (summed across COPCs) did not exceed 1 at all paired residential beach DUs. No COPCs had individual HQs >1 using either RME or CTE exposure assumptions.

Dermal Contact with Residential Soil Associated with Beach DUs

Current residents were assumed to be exposed to non-lead COPCs in soil via dermal exposure for residential soil paired with 21 residential beach DUs sampled in the 2014 and 2016 residential soil studies. For all receptors (adults and children), the total non-cancer hazard from dermal contact with soil under both RME and CTE scenarios did not exceed 1 (Tables 5-33 and 5-34).

Total Exposure to Residential Soil Associated with Beach DUs

When exposure to non-lead COPCs in residential soil paired with residential beach DUs was summed across pathways (incidental ingestion of soil/dust and dermal contact with soil), total non-cancer HIs >1 at 13 residential beach DUs for current child residents (ranged from 1 to 2 at the RME and 0.3 to 0.7 at the CTE), not considering target organ effects, and did not exceed 1 for adult residents at the RME and CTE (Tables 5-33 and 5-34). When HIs were aggregated by target organ system, no residential soil paired with beach DUs had HIs >1 for either adults or children under RME and CTE exposure scenarios (Tables 5-33 and 5-34). Based on the target organ HI not exceeding 1, risks of non-cancer health effects from exposure to non-lead COPCs through this pathway were below non-cancer risk benchmarks used by CERCLA (EPA, 1989; EPA and Clay, 1991; EPA, 1997).

5.2.3.2 Surface Soil/Indoor Dust: Potential Future Residential Exposure to Non-Lead COPCs

Incidental Ingestion of Upland Soil

The potential future resident population was assumed to be exposed to non-lead COPCs in outdoor soil and indoor dust via incidental ingestion at all 142 ADAs sampled in the 2014 Upland Soil Study. Houses were not observed on ADAs during sampling, though ADAs could potentially be developed for residential use in the future. For potential future adult residents evaluated using default exposure parameters, all total soil ingestion non-cancer hazards (summed across COPCs for each ADA) were below one under both the RME and CTE scenarios for incidental soil ingestion (Tables 5-35 and 5-36). Total soil ingestion non-cancer hazards for potential future child residents (summed across COPCs) were >1 for 133 of 142 ADAs under the RME scenario and did not exceed 1 at all ADAs under the CTE scenario, not considering target organ effects. Individual HQs did not exceed 1 for any COPC under the RME or CTE exposure scenario.

Dermal Contact with Upland Soil

Potential future residents were assumed to be exposed to non-lead COPCs in soil via dermal exposure to soil at all ADAs sampled in the 2014 Upland Soil Study. For all receptors (adults and children), the total non-cancer hazard from dermal contact with soil under both RME and CTE scenarios did not exceed 1 (Tables 5-35 and 5-36).

Total Exposure to Upland Soil

When exposure to non-lead COPCs in upland soil for potential future residential use was summed across pathways (incidental ingestion of soil/dust and dermal contact with soil), total non-cancer HIs for future child residents were >1 at 136 ADAs (HIs ranged from 1 to 4 at the RME, and 0.4 to 1 at the CTE) (Tables 5-35 and 5-36), not considering target organ effects. Because HIs were >1 for total exposure to upland soil/dust (via ingestion and dermal contact) for potential future child residents at the RME, HIs calculated for each target organ group are presented in Tables 5-37 through 5-45 for the RME scenario and Tables 5-46 through 5-54 for the CTE scenario. When HIs were aggregated by target organ system, 2 ADAs had HIs >1 for the skin/hair/nails system for the potential future child population under RME assumptions (Figure 5-10). Under the CTE scenario for the potential future child population, no target organ systems had HIs >1. No HIs were >1 by target organ system for the potential future adult population under either the RME or CTE exposure scenarios (Tables 5-37 through 5-54). Chemicals that contributed the most to risk included thallium, manganese, cobalt, arsenic, and iron. Based on HIs being >1 when evaluated by target organ system at the RME, there is potential for non-cancer health effects to children from non-lead COPCs through the total exposure to surface soil and indoor dust pathway.

5.2.3.3 Residential Beach Sediment: Current Residential Exposure to Non-Lead COPCs

During the 2014 and 2016 residential soil studies, 21 DUs were sampled on beaches on residential properties that are not CCT tribal allotments. The current adult and child residential population owning property with beach DUs was assumed to be exposed to non-lead COPCs in sediment via incidental ingestion and dermal contact. Total non-cancer HIs did not exceed 1 for adults and children for both incidental ingestion and dermal contact of beach sediment (summed across COPCs) under both RME and CTE scenarios (Tables 5-55 and 5-56). Summed across pathways (incidental ingestion plus dermal contact with sediment), total non-cancer HIs did not exceed 1 for adults and children (summed across COPCs) under both RME and CTE scenarios (Tables 5-55 and 5-56). No target organ HIs were >1 for either adult or child population under RME or CTE scenarios. Based on the HI not exceeding 1, risks of non-cancer health effects from exposure to non-lead COPCs through this pathway were below non-cancer risk benchmarks used by CERCLA (EPA, 1989; EPA and Clay, 1991; EPA, 1997).

5.2.3.4 Surface Sediment at Public Beaches along the UCR: Occupational and Recreational Exposure to Non-Lead COPCs

The recreational visitor and outdoor worker populations were assumed to be exposed to non-lead COPCs in surface sediment via incidental ingestion and dermal contact at each of the 33 public beaches sampled along the UCR. Bossburg Flat and Evans Campground beaches were sampled in 2011 and 2015. The SDUs sampled at these beaches in 2015 were evaluated as separate exposure areas, so a total of 43 public beach exposure areas were evaluated in this section.

Outdoor Workers: Total Exposure to Public Beach Surface Sediment

For the adult outdoor worker population, the HIs (summed across COPCs) for both incidental ingestion of and dermal contact with surface sediment did not exceed 1 at any public beach for both the RME and CTE exposure scenarios (Tables 5-57 and 5-58). When exposure to surface sediment on public beaches was summed across pathways (incidental ingestion and dermal contact), total non-cancer HIs did not exceed 1 for both RME and CTE scenarios for the adult outdoor worker. This was also true when HIs were aggregated by target organ system (Tables 5-57 and 5-58). Based on the HI not exceeding 1, risks of non-cancer health effects from exposure to non-lead COPCs through these pathways were below non-cancer risk benchmarks used by CERCLA (EPA, 1989; EPA and Clay, 1991; EPA, 1997).

Recreational Visitors: Total Exposure to Public Beach Surface Sediment

For the adult and child recreational visitor population (on beach day trips to public beaches, boating trips near public beaches, or camping trips near public beaches), the HIs (summed across COPCs) for incidental ingestion of and dermal contact with surface sediment did not exceed 1 at any public beach for adults and children for both the RME and CTE exposure scenarios (Tables 5-59 and 5-60). When exposure to surface sediment on public beaches was summed across pathways (incidental ingestion and dermal contact), total non-cancer HIs did not exceed 1 for either RME or CTE scenarios for the adult and child recreational beach visitor, boater, or camper. This was also true when HIs were aggregated by target organ system (Tables 5-59 and 5-60). Based on the HI not exceeding 1, risks of non-cancer health effects from exposure to non-lead COPCs through these pathways were below non-cancer risk benchmarks used by CERCLA (EPA, 1989; EPA and Clay, 1991; EPA, 1997).

5.2.3.5 Surface Soil at Relict Floodplains along the UCR: Recreational Visitor Exposure to Non-Lead COPCs

As part of the 2014 Upland Soil study, 16 RFDAs were sampled on 4 relict floodplains. The recreational visitor population was assumed to be exposed to non-lead COPCs in surface soil at 16 RFDAs along the UCR while on day trips to the beach, on boating trips, or on camping trips. For both adults and children, the HIs (summed across COPCs) for incidental ingestion of and dermal contact with surface soil did not exceed 1 at any RFDA for the RME or CTE exposure scenarios (Tables 5-61 and 5-62). When exposure to surface soil on RFDAs was summed across pathways (incidental ingestion and dermal contact), total non-cancer HIs did not exceed 1 for either the RME or CTE scenario for the adult and child recreational visitor. This was also true when HIs are aggregated by target organ system (Tables 5-30 and 5-31). Based on the HI not exceeding 1, risks of non-cancer health effects from exposure to non-lead COPCs through these pathways were below non-cancer risk benchmarks used by CERCLA (EPA, 1989; EPA and Clay, 1991; EPA, 1997).

5.2.3.6 Surface Soil near Bossburg Flat Beach: Occupational and Recreational Exposure to Non-Lead COPCs

The outdoor worker and recreational visitor populations were assumed to be exposed to non-lead COPCs in surface soil near Bossburg Flat Beach via incidental ingestion and dermal contact at each UDU sampled in 2015.

Outdoor Workers: Total Exposure to Bossburg Flat Beach Surface Soil

For the adult outdoor worker population, the HIs (summed across COPCs) for incidental ingestion of and dermal contact with surface soil did not exceed 1 at any Bossburg Flat UDU for both the RME and CTE exposure scenarios (Tables 5-63 and 5-64). When exposure to surface soil on UDUs was summed across pathways (incidental ingestion and dermal contact), total non-cancer HIs did not exceed 1 for both RME and CTE scenarios for the adult outdoor worker. This was also true when HIs are aggregated by target organ system (Tables 5-63 and 5-64). Based on the HI not exceeding 1, risks of non-cancer health effects from exposure to non-lead COPCs through these pathways were below non-cancer risk benchmarks used by CERCLA (EPA, 1989; EPA and Clay, 1991; EPA, 1997).

Recreational Visitors: Total Exposure to Bossburg Flat Beach Surface Soil

For the adult and child recreational visitor population on beach day, boating, or camping trips to Bossburg Flat Beach UDUs, the HIs (summed across COPCs) for incidental ingestion of and dermal contact with surface soil did not exceed 1 at any UDU for adults and children for both the RME and CTE exposure scenarios (Tables 5-65 and 5-66). When exposure to surface soil on UDUs was summed across pathways (incidental ingestion and dermal contact), total non-cancer HIs did not exceed 1 for both RME and CTE scenarios for the adult and child recreational visitor. This was also true when HIs were aggregated by target organ system (Tables 5-65 and 5-66). Based on the HI not exceeding 1, risks of non-cancer health effects from exposure to non-lead COPCs through these pathways were below non-cancer risk benchmarks used by CERCLA (EPA, 1989; EPA and Clay, 1991; EPA, 1997).

5.2.3.7 Subsurface Sediment and Soil at Public Beaches along the UCR: Occupational Exposure to Non-Lead COPCs

Subsurface sediment was collected at Bossburg Flat and Evans Campground beaches (SDUs and former cable ferry landing sample F-01), as described in Section 2.6.2.4. Subsurface soil was also collected at Bossburg Flat Beach UDUs, as described in Section 2.6.2.4. The outdoor worker population was assumed to be exposed to non-lead COPCs in subsurface sediment and soil via incidental ingestion and dermal contact during excavation activities at public beaches along the UCR. All HIs (summed across COPCs) did not exceed 1 for receptors exposed to subsurface sediment and soil via incidental ingestion under both RME and CTE scenarios (Tables 5-67 through 5-70). All HIs for the dermal contact scenario (summed across COPCs) did not exceed 1 for receptors exposed to subsurface sediment and soil under both RME and CTE scenarios (Tables 5-67 through 5-70). When exposure to subsurface sediment and soil at public beaches was summed across pathways (incidental ingestion and dermal contact), total non-cancer HIs did not exceed 1 for adult outdoor workers under RME and CTE scenarios. This was also true when HIs are aggregated by target organ system (Tables 5-67 through 5-70). Risks of non-cancer health effects from exposure to non-lead COPCs through this pathway were below non-cancer risk benchmarks used by CERCLA (EPA, 1989; EPA and Clay, 1991; EPA, 1997).

5.2.3.8 Public Beach Near-shore UCR Surface Water: Residential, Occupational, and Recreational Exposure to Non-Lead COPCs

The resident owning beach property, outdoor worker, and recreational visitor populations were assumed to be exposed to non-lead COPCs in UCR surface water via incidental ingestion and dermal contact while swimming, participating in water-related activities, or working. Non-cancer hazards for each receptor due to surface water exposure are detailed below.

Resident with a Beach: Total Exposure to UCR Surface Water

For adult and child residents with beaches, all HIs (summed across COPCs) did not exceed 1 due to both incidental ingestion of and dermal contact with UCR surface water under RME and CTE exposure scenarios (Tables 5-71 and 5-72). When exposure to UCR surface water was summed across pathways (incidental ingestion and dermal contact), total non-cancer HIs did not exceed 1 for child and adult residents recreating on their own beaches under both CTE and RME scenarios. This was also true when HIs were aggregated by target organ system (Tables 5-71 and 5-72). Risks of non-cancer health effects from exposure to non-lead COPCs through this pathway were below non-cancer risk benchmarks used by CERCLA (EPA, 1989; EPA and Clay, 1991; EPA, 1997).

Outdoor Worker: Total Exposure to UCR Surface Water

For the adult outdoor worker population, all HIs (summed across COPCs) did not exceed 1 due to both incidental ingestion of and dermal contact with UCR surface water under RME and CTE exposure scenarios (Tables 5-73 and 5-74). When exposure to UCR surface water was summed across pathways (incidental ingestion and dermal contact), total non-cancer HIs did not exceed 1 for the adult outdoor worker population under both CTE and RME scenarios. This was also true when HIs were aggregated by target organ system (Tables 5-73 and 5-74). Risks of non-cancer health effects from exposure to non-lead COPCs through this pathway were below non-cancer risk benchmarks used by CERCLA (EPA, 1989; EPA and Clay, 1991; EPA, 1997).

Recreational Visitors: Total Exposure to UCR Surface Water

For adult and child recreational visitors taking day trips to UCR beaches, boat trips, or camping trips, all HIs (summed across COPCs) did not exceed 1 due to both incidental ingestion of and dermal contact with UCR surface water under RME and CTE exposure scenarios (Tables 5-75 and 5-76). When exposure to UCR surface water was summed across pathways (incidental ingestion and dermal contact), total non-cancer HIs did not exceed 1 for adult and child recreational visitors under both CTE and RME scenarios. This was also true when HIs are aggregated by target organ system (Tables 5-75 and 5-76). Risks of non-cancer health effects from exposure to non-lead COPCs through this pathway were below non-cancer risk benchmarks used by CERCLA (EPA, 1989; EPA and Clay, 1991; EPA, 1997).

5.2.3.9 Outdoor Air: Exposure to Non-Lead COPCs

Current and potential future residents, outdoor workers, and recreational visitors were assumed to be exposed to non-lead COPCs (arsenic and cadmium) in outdoor air in the vicinity of the UCR. For all receptors (adults and children), total non-cancer HIs did not exceed 1 (summed across COPCs) under both RME and CTE scenarios (Tables 5-77 through 5-79). This was also true when HIs were aggregated by target organ system. Based on the HI not exceeding 1, risks of non-cancer health effects from exposure to non-lead COPCs through this pathway were below non-cancer hazard benchmarks used by CERCLA (EPA, 1989, 1997; EPA and Clay, 1991).

Additionally, EPA evaluated the air results from the 1999-2009 Sheep Creek air monitoring. The evaluation for exposure to cadmium and arsenic is discussed in Section 5.2.4.1. For zinc, which was not identified as a COPC for this HHRA, the air concentration from the Sheep Creek monitoring is well below all RBSLs (EPA, 2018b; Appendix 8).

5.2.3.10 Indoor Air: Exposure to Non-Lead COPCs

Current and potential future residents live in the vicinity of the Site and are therefore exposed to non-lead COPCs in air inside their homes. For both adult and child residents (with and without beaches), total non-cancer HIs did not exceed 1 (summed across COPCs) under both RME and CTE scenarios (Table 5-80). This was also true when HIs were aggregated by target organ system. Based on the HI not exceeding 1, risks of non-cancer health effects from exposure to non-lead COPCs through this pathway were below non-cancer hazard benchmarks used by CERCLA (EPA, 1989, 1997; EPA and Clay, 1991).

5.2.3.11 UCR Fish Consumption: Exposure to Non-Lead COPCs

The residential (with and without a beach) and recreational visitor populations were assumed to be exposed to non-lead COPCs in fish caught from the UCR and consumed. Residents with and without beaches and recreational visitors were evaluated for consumption of individual fish species, as anglers may target specific fish species. Non-cancer hazards are presented separately below for these two populations.

Residential (With and Without Beach) Exposure to Non-Lead COPCs from Consumption of Fish Harvested from the UCR

The resident population[91] (with and without residential beaches) was evaluated for exposure to each individual fish species sampled as described in Section 2.6.4.1. Non-cancer and target organ hazards for the adult and child resident population are shown in Tables 5-81 through 5-89 for the RME exposure scenario and Tables 5-90 through 5-98 for the CTE exposure scenario. When HQs were summed across COPCs, the HI for the adult resident population did not exceed 1 for consumption of Northern Pike and White Sturgeon under RME and CTE exposure assumptions. The HI was >1 under RME exposure assumptions for consumption of Burbot, kokanee, Rainbow Trout, Smallmouth Bass, sucker, Walleye, and whitefish (Tables 5-81 through 5-89). Using the CTE fish consumption rate, adult HIs did not exceed 1 (Tables 5-90 through 5-98). No individual COPCs had HQs >1 for the adult resident population under RME or CTE exposure assumptions. When HIs were aggregated by target organ under RME or CTE assumptions, no target organ HIs >1 for the current adult resident population.

For the child resident population, when HQs were summed across COPCs, HIs using RME exposure assumptions were >1 for all fish species evaluated (Tables 5-81 through 5-89). Individual COPC child RME HQs that exceeded 1 were:

- Methylmercury: Burbot, Smallmouth Bass, sucker, and Walleye
- Thallium: kokanee, Rainbow Trout, Smallmouth Bass, Walleye, and whitefish
- TEQ (dioxins/furans plus dioxin-like PCBs): sucker and whitefish

Using the RME fish consumption rate for child residents, no target organ HIs were >1 for Northern Pike or White Sturgeon. In the other species evaluated, at least one of the following

[91] While Appendix 1 evaluates the high-intensity resource users within the CCT population, the non-subsistence CCT population is represented by the residential population evaluated in this HHRA.

target organ systems had HIs>1: developmental, nervous, reproductive, and/or skin/hair/nails systems (Tables 5-81 through 5-89).

Using CTE exposure assumptions (Tables 5-90 through 5-98), HIs for the child resident population (summed across COPCs) were not >1 for Burbot, kokanee, Northern Pike, Rainbow Trout, White Sturgeon, and whitefish. HIs for consumption of Smallmouth Bass, sucker, and Walleye were >1 under CTE assumptions for the child resident population. No individual COPCs had HQs >1 for the child resident population under CTE exposure assumptions. When HIs were aggregated by target organ system for the child resident population, using the CTE fish consumption rate, there were no target organ HIs >1 for any fish species.

COPCs that contributed the most to risk based on consumption of UCR fish included methylmercury, thallium, and TEQ. Based on target organ HIs being >1, there was potential for non-cancer health effects from exposure to non-lead COPCs for the residential child population consuming fish from the UCR (with and without residential beaches).

Recreational Exposure to Non-Lead COPCs from Consumption of Fish from the UCR
The recreational visitor population was evaluated for exposure to each individual fish species sampled as described in Section 2.6.4.1. When HQs were summed across COPCs, the adult HI did not exceed 1 for Northern Pike and White Sturgeon using the RME fish consumption rate. The adult HI was >1 using the RME fish consumption rate for Burbot, kokanee, Rainbow Trout, Smallmouth Bass, sucker, Walleye, and whitefish (Tables 5-99 through 5-107). No individual COPCs had HQs >1 under the RME or CTE scenarios for the adult recreational visitor. Using the CTE fish consumption rate, HIs for the adult recreational visitor population did not exceed 1 (Tables 5-108 through 5-116).

For the child recreational visitor population, when HQs were summed across COPCs, HIs using RME exposure assumptions were >1 for all fish species evaluated (Tables 5-99 through 5-107). Individual COPC child RME HQs that exceeded 1 were:

- Methylmercury: Burbot, Smallmouth Bass, sucker, and Walleye
- Thallium: kokanee, Rainbow Trout, Smallmouth Bass, Walleye, and whitefish
- TEQ (dioxins/furans plus dioxin-like PCBs): sucker and whitefish

Using CTE exposure assumptions (Tables 5-106 through 5-116), the HI for the child recreational visitor population (summed across COPCs) was >1 for Walleye. No individual COPCs had HQs >1 for the child recreational visitor under the CTE scenario.

Using the RME fish consumption rate, no target organ HIs were >1 for the adult recreational visitor population for any fish species (Tables 5-99 through 5-107). For children, using the RME fish consumption rate, no target organ HI was >1 for Northern Pike or White Sturgeon. For other fish species, at least one of the following target organ systems had HI >1: developmental, nervous, reproductive, and/or skin/hair/nails systems (Tables 5-99 through 5-107). When HIs were aggregated by target organ system, no target organ HIs >1 for the child and adult recreational visitor using the CTE fish consumption rate (Tables 5-106 through 5-116).

Chemicals that contributed the most to risk based on consumption of UCR fish by recreational visitors were methylmercury, thallium, and TEQ. Based on target organ HIs being >1, there is potential for non-cancer health effects from exposure of child recreational visitors to non-lead COPCs through this pathway.

5.2.4 Cancer Summary

In the assessment described below, cancer risks that were $\leq 10^{-4}$ were considered to be within an acceptable range and risks above 10^{-4} were categorized as unacceptable (NCP, 1990). Total excess cancer risks for each COPC, receptor, and exposure pathway are described separately by exposure pathway below.

5.2.4.1 Surface Soil/Indoor Dust: Current Residential Exposure to Non-Lead COPCs

Because arsenic is the only non-lead COPC in residential soil that has both a slope factor and a dermal absorption fraction, cancer risk from exposure to soil is solely attributable to arsenic. Cancer risk from exposure to non-lead COPCs in surface soil/indoor dust was evaluated separately for "non-beach" and "beach" residential DUs. Each is described separately below.

Exposure to Residential Soil and Indoor Dust at Non-Beach DUs

Current residents were evaluated for exposure to arsenic in residential soil and dust via incidental ingestion under both RME and CTE exposure scenarios. Total excess cancer risks from soil/dust ingestion were below 10^{-4} (Tables 5-13 and 5-14) for both children and adults, as well as summed across a lifetime. Total excess cancer risks from dermal contact with soil were also below 10^{-4} (Tables 5-13 and 5-14) for both children and adults and summed across a lifetime.[92] When exposure to non-lead COPCs in residential soil for current residential use was summed across pathways (incidental ingestion of soil/dust and dermal contact with soil), total excess cancer risks were below 10^{-4} (Tables 5-13 and 5-14) for both children and adults at the RME and CTE. These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

Exposure to Residential Soil and Indoor Dust Paired with Beach DUs

For the current resident population evaluated for exposure to arsenic in residential soil and dust paired with 21 residential beach DUs via incidental ingestion under both RME and CTE exposure scenarios, total excess cancer risks from soil/dust ingestion were below 10^{-4} (Tables 5-33 and 5-34) for both children and adults, as well as summed across a lifetime. Total excess cancer risks from dermal contact with soil were also below 10^{-4} (Tables 5-33 and 5-34) for both children and adults and summed across a lifetime. When exposure to non-lead COPCs in residential soil for current residential use was summed across pathways (incidental ingestion of soil/dust and dermal contact with soil), total excess cancer risks were below 10^{-4} (Tables 5-33 and 5-34) for both children and adults at the RME and CTE. These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

[92] Note that the EPC of 52.8 mg/kg for arsenic in Tables 5-13 and 5-14 is not RBA-adjusted because the exposure route is dermal. Additionally, the 95UCL was higher than the maximum concentration.

5.2.4.2 Surface Soil/Indoor Dust: Potential Future Residential Exposure to Non-Lead COPCs

Potential future residents where houses may be built were evaluated for exposure to arsenic in upland soil via incidental ingestion for receptors exposed under both RME and CTE scenarios. Total excess cancer risks from soil and dust ingestion were below 10^{-4} (Tables 5-35 and 5-36) for both children and adults and summed across a lifetime. Potential future residents were also evaluated for exposure to arsenic in upland soil via dermal contact with soil for receptors exposed under both RME and CTE scenarios. Total excess cancer risks from dermal contact with soil were also below 10^{-4} (Tables 5-35 and 5-36) for both children and adults and summed across a lifetime. When exposure to non-lead COPCs in upland soil that may be utilized for future residential use was summed across pathways (incidental ingestion of soil/dust and dermal contact with soil), total excess cancer risks were below 10^{-4} (Tables 5-35 and 5-36) for both children and adults and summed across a lifetime. These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

5.2.4.3 Residential Beach Sediment: Current Residential Exposure to Non-Lead COPCs

During the 2014 and 2016 residential soil studies, 21 DUs were sampled on beaches on residential properties that are not CCT tribal allotments. The resident population assumed to be recreating on residential beach DUs on their property was evaluated for exposure to non-lead COPCs in sediment via incidental ingestion for adult and child receptors exposed under both CTE and RME scenarios. Total excess cancer risks (summed across COPCs for each residential beach DU) due to incidental ingestion of sediment were below 10^{-4} for adults, children, and summed across a lifetime (Tables 5-55 and 5-56). Current adult and child residents were also evaluated for exposure to non-lead COPCs in sediment via dermal exposure for adults and children exposed under both CTE and RME scenarios. Total excess cancer risks (summed across COPCs for each residential beach DU) for dermal contact with sediment were below 10^{-4} (Tables 5-55 and 5-56) for both children and adults and summed across a lifetime. When exposure to sediment on residential beaches was summed across pathways (incidental ingestion and dermal contact), total cancer risk was below 10^{-4} for adult and child residents and summed across a lifetime at both the RME and the CTE.

5.2.4.4 Surface Sediment at Public Beaches along the UCR: Occupational and Recreational Exposure to Non-Lead COPCs

The outdoor worker and recreational visitor populations were assumed to be exposed to non-lead COPCs in surface sediment via incidental ingestion and dermal contact at each of the 33 public beaches sampled along the UCR. Bossburg Flat and Evans Campground beaches were sampled in 2011 and 2015. The SDUs sampled at these beaches in 2015 were evaluated as separate exposure areas, so a total of 43 public beach exposure areas were evaluated in this section.

Outdoor Workers: Total Exposure to Public Beach Surface Sediment

For the adult outdoor worker population exposed to non-lead COPCs in public beach surface sediment, total excess cancer risks for both incidental ingestion and dermal contact were below 10^{-4} under both RME and CTE exposure scenarios. When exposure to surface sediment on public beaches was summed across pathways (incidental ingestion and dermal contact; Tables 5-57 and 5-58), total cancer risk was below 10^{-4} for the adult outdoor worker population under the RME and CTE scenarios. These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

Recreational Visitors: Total Exposure to Public Beach Surface Sediment

For the adult and child recreational visitor population (and summed across a lifetime) on beach day trips, boating trips, or camping trips to public beaches, the total excess cancer risks for both incidental ingestion and dermal contact were below 10^{-4} under both RME and CTE exposure scenarios (Tables 5-59 and 5-60). When exposure to surface sediment on public beaches was summed across pathways (incidental ingestion and dermal contact), total cancer risk was below 10^{-4} for the adult and child recreational visitor (as well as summed across a lifetime) under the RME and CTE scenarios (Tables 5-59 and 5-60). These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

5.2.4.5 Surface Soil at Relict Floodplains along the UCR: Recreational Exposure to Non-Lead COPCs

As part of the 2014 Upland Soil study, 16 RFDAs were sampled on four relict floodplains. The recreational visitor population was assumed to be exposed to non-lead COPCs in surface soil at 16 RFDAs along the UCR while on day trips to the beach, boating trips, or camping trips. For both adults and children (and summed across a lifetime), the total excess cancer risks for both incidental ingestion and dermal contact were below 10^{-4} under both RME and CTE exposure scenarios (Tables 5-61 and 5-62). When exposure to surface soil on relict floodplains is summed across pathways (incidental ingestion and dermal contact), total cancer risk was below 10^{-4} for the adult and child recreational visitor (as well as summed across a lifetime) under the RME and CTE scenarios (Tables 5-61 and 5-62). These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

5.2.4.6 Surface Soil at Bossburg Flat Beach: Occupational and Recreational Exposure to Non-Lead COPCs

The outdoor worker and recreational visitor populations were assumed to be exposed to non-lead COPCs in surface soil near Bossburg Flat Beach via incidental ingestion and dermal contact at each UDU sampled in 2015.

Outdoor Workers: Total Exposure to Bossburg Flat Beach Surface Soil

For the adult outdoor worker population exposed to non-lead COPCs in public beach surface soil at UDUs, total excess cancer risks for both incidental ingestion and dermal contact were below 10^{-4} under both RME and CTE exposure scenarios at each UDU (Tables 5-63 and 5-64). When exposure to surface soil at Bossburg Flat Beach UDUs was summed across pathways (incidental ingestion and dermal contact), total cancer risk was below 10^{-4} for the adult outdoor worker population under the RME and CTE scenarios (Tables 5-63 and 5-64). These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

Recreational Visitors: Total Exposure to Bossburg Flat Beach Surface Soil

For the adult and child recreational visitor population (and summed across a lifetime) on beach day, boating, or camping trips to Bossburg Flat Beach UDUs, the total excess cancer risks for both incidental ingestion and dermal contact were below 10^{-4} under both RME and CTE exposure scenarios (Tables 5-65 and 5-66). When exposure to surface soil on Bossburg Flat Beach UDUs was summed across pathways (incidental ingestion and dermal contact), total cancer risk was below 10^{-4} for the adult and child recreational visitor (as well as summed across

a lifetime) under the RME and CTE scenarios (Tables 5-65 and 5-66). These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

5.2.4.7 Subsurface Sediment and Soil at Public Beaches along the UCR: Occupational Exposure to Non-Lead COPCs

Subsurface sediment was collected at Bossburg Flat and Evans Campground beach SDUs and former cable ferry landing location F-01, as described in Section 2.6.2.4. Subsurface soil was also collected at Bossburg Flat Beach UDUs, as described in Section 2.6.2.4. The outdoor worker population was assumed to be exposed to non-lead COPCs in subsurface sediment and soil via incidental ingestion and dermal contact during excavation activities at public beaches along the UCR. Total excess cancer risks at each of the SDUs and UDUs for this receptor population exposed at both the RME and CTE, for both incidental ingestion and dermal contact with subsurface sediment or soil, were below 10^{-4} (Tables 5-67 through 5-70). When exposure to subsurface sediment or soil was summed across pathways (incidental ingestion and dermal contact), total cancer risk was below 10^{-4} for adult outdoor workers under both RME and CTE conditions (Tables 5-67 through 5-70). These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

5.2.4.8 Public Beach Near-shore UCR Surface Water: Residential, Occupational, and Recreational Exposure to Non-Lead COPCs

The resident owning beach property, outdoor worker, and recreational visitor populations were assumed to be exposed to non-lead COPCs in UCR surface water via incidental ingestion and dermal contact while swimming, participating in water-related activities, or working. Cancer risks for each receptor population due to UCR surface water exposure are detailed below.

Resident with a Beach: Total Exposure to UCR Surface Water

For adult and child residents with beaches on their property, total excess cancer risks summed across a lifetime, evaluated under RME and CTE scenarios, were below 10^{-4} both for incidental ingestion and dermal contact with surface water (Tables 5-71 and 5-72). When exposure to UCR surface water was summed across pathways (incidental ingestion and dermal contact), total cancer risk was below 10^{-4} for adult and child residents owning beach property summed across a lifetime at both the RME and CTE (Tables 5-71 and 5-72). These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

Outdoor Worker: Total Exposure to UCR Surface Water

For the adult outdoor worker, total excess cancer risks, evaluated under RME and CTE scenarios, were below 10^{-4} both for incidental ingestion and dermal contact with surface water (Tables 5-73 and 5-74). When exposure to UCR surface water was summed across pathways (incidental ingestion and dermal contact), total cancer risk was below 10^{-4} for the adult outdoor worker population at both the RME and CTE. These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

Recreational Visitors: Total Exposure to UCR Surface Water

For adult and child recreational visitors taking beach day trips, boating trips, or camping trips to UCR beaches, total excess cancer risks summed across a lifetime, evaluated under RME and CTE scenarios, were below 10^{-4} both for incidental ingestion and dermal contact with surface

water (Tables 5-75 and 5-76). When exposure to UCR surface water was summed across pathways (incidental ingestion and dermal contact), total cancer risk was below 10^{-4} for adult and child recreational visitors summed across a lifetime at both the RME and CTE (Tables 5-75 and 5-76). These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

5.2.4.9 Outdoor Air Exposure to Non-Lead COPCs

Current and potential future resident, outdoor worker, and recreational visitor populations were assumed to be exposed to non-lead COPCs in outdoor air via inhalation. Total excess cancer risks for each receptor population, evaluated for receptors under RME and CTE scenarios, were below 10^{-4} (Tables 5-77 through 5-79). These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

Additionally, EPA evaluated the air results from the 1999-2009 Sheep Creek air monitoring. For arsenic, the measured air concentration corresponds to a cancer risk of 2×10^{-5} (an increased risk of cancer of two in one hundred thousand for a lifetime of exposure). For comparison, this is the same level of risk from naturally occurring arsenic in soil. For cadmium, the measured air concentration corresponds to a cancer risk of 2×10^{-6} (an increased risk of cancer of two in one million for a lifetime of exposure; EPA, 2018b; Appendix 8).

5.2.4.10 Indoor Air Exposure to Non-Lead COPCs

As described previously, current and potential future residents live in the vicinity of the Site and are exposed to non-lead COPCs in air inside their homes. These populations were assumed to be exposed to non-lead COPCs in indoor air via inhalation. Indoor air COPC concentrations were assumed to be equal to outdoor air COPC concentrations. Total excess cancer risks for these receptor populations, evaluated under RME and CTE scenarios, were below 10^{-4} (Table 5-80). These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

5.2.4.11 Exposure to Non-Lead COPCs from Consumption of Fish from the UCR

The residential (with and without a beach) and recreational visitor populations were assumed to be exposed to non-lead COPCs in fish caught from the UCR and consumed. Residents with and without beaches and recreational visitors were evaluated for consumption of individual fish species since anglers may target specific fish species. Cancer risks are presented separately below for these two populations.

Residential (Beach and Non-beach) Exposure to Non-Lead COPCs from Consumption of Fish from the UCR

For residents with and without beaches on their property, total excess cancer risks for adults, children, and summed across a lifetime were below 10^{-4} under RME and CTE scenarios for the consumption of fish (Tables 5-81 through 5-98). These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

Recreational Exposure to Non-Lead COPCs from Consumption of Fish from the UCR

The recreational visitor population was evaluated for exposure to individual fish species sampled as described in Section 2.6.4.1. Total excess cancer risks for adults, children, and summed across

a lifetime were below 10^{-4} under both RME and CTE scenarios for the consumption of fish (Tables 5-99 through 5-116). These risks were within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

5.2.5 Non-Lead COPC Risks by Receptor

The discussion above described calculated risk to receptors from exposure to a particular environmental medium, across exposure pathways relevant for that medium. An individual residing at, working at, or visiting the UCR Site may be exposed to COPCs in multiple environmental media on-Site through several exposure pathways, as described in the CSM developed for this Site (Figure 3-1). The total exposure to various chemicals will equal the sum of the exposures from all exposure pathways.

To assess the overall potential for non-carcinogenic effects posed by several exposure pathways, the HIs for each exposure pathway evaluated for that receptor population are summed. For multiple exposure pathways, the total HI can be >1 even if no single exposure pathway HI exceeds one. If an HI is >1 as a consequence of summing several HQs, the COPCs can be segregated by effect and mode of action, and an HI can be calculated for each target organ group (EPA, 1989). HIs summed across exposure pathways and aggregated by target organ group are discussed below for each receptor population.

Cancer risks from various exposure pathways are also assumed to be additive, as long as less-than lifetime exposures have all been converted to equivalent lifetime exposures. Total exposure cancer risk is equal to the sum of cancer risk for each exposure pathway evaluated for a receptor population.

5.2.5.1 Risks to Current Residents from DUs that are Not Beaches

The exposure pathways evaluated for current residents from DUs that are not beaches were:

- inhalation of outdoor air,
- inhalation of indoor air,
- dermal contact with outdoor soil and incidental ingestion of outdoor soil and indoor dust, and
- consumption of fish caught from the UCR evaluated by species.

Risks were evaluated on a DU-by-DU basis for each of the 588 residential DUs sampled in 2014 and 2016 except beaches, CCT tribal allotments, driplines, or the DU that was potentially a previous ore stockpile. As noted previously, while Appendix 1 evaluates the high-intensity resource users within the CCT population, the non-subsistence CCT population is represented by the residential population evaluated in this HHRA.

Non-cancer HIs aggregated by target organ system for the current adult and child resident population, both summed across COPCs by exposure pathway, and summed across pathways, are shown in Tables 5-15 through 5-32 for the RME and CTE scenarios. For the RME scenario, inhalation of outdoor and indoor air did not result in exceedances of target organ HIs for the

child or adult (Tables 5-15 through 5-23). Target organ HIs for outdoor air were 0.03 and 0.05 for the nervous system for the resident child and adult, respectively, and 0.01 and 0.02 for the urinary system for the resident child and adult, respectively. Target organ HIs for indoor air were 0.3 for the nervous system and 0.2 for the urinary system for both children and adults. Incidental ingestion of outdoor soil and indoor dust, plus dermal contact with outdoor soil, did not result in any target organ HIs above the benchmark of 1 for adults for the RME scenario. For children, the soil/dust pathway resulted in target organ HI >1 for the skin/hair/nails system at one house DU at the RME (HI = 2; Tables 5-15 through 5-23).

For consumption of fish at the RME, no target organ HIs exceeded 1 for adults for any fish species evaluated. No target organ HIs exceeded 1 for children due to consumption of Northern Pike or White Sturgeon at the RME. Consumption of fish under RME exposure assumptions resulted in target organ HIs >1 for children for the following species and target organ systems:

- Consumption of Burbot: developmental and nervous systems (Table 5-15)
- Consumption of kokanee (Table 5-16) and Rainbow Trout (Table 5-18): skin/hair/nails target organ system
- Consumption of Smallmouth Bass and Walleye: developmental, nervous, and skin/hair/nails systems (Tables 5-19 and 5-22, respectively)
- Consumption of sucker: developmental, nervous, and reproductive target organ system (Table 5-21)
- Consumption of whitefish: reproductive and skin/hair/nails systems (Table 5-23)

When these four exposure pathways were summed together under the RME scenario for the current adult resident population, no DUs had non-cancer target organ HIs >1 when the fish consumed was Burbot, kokanee, Northern Pike, Rainbow Trout, Smallmouth Bass, White Sturgeon, or whitefish (Tables 5-15 through 5-20 and Table 5-23). All 588 DUs had non-cancer target organ HIs >1 for the nervous system for adults when the fish consumed was sucker (Table 5-21), and one house DU had a target organ HI >1 for the skin/hair/nails system for adults who were assumed to consume Walleye (Table 5-22; Figure 5-11). For children, when the exposure pathways were summed together for the RME scenario, at least some DUs had non-cancer target organ HIs >1 for each fish species consumed (Tables 5-15 through 5-23; Figures 5-12 through 5-17). Target organ systems exceeding non-cancer benchmarks for children due to fish consumption included the cardiovascular, developmental, endocrine, nervous, reproductive, and skin/hair/nails systems.

For the CTE scenario, no exposure pathways by themselves had target organ HIs >1 for adults or children (Tables 5-24 through 5-32). When the four exposure pathways were summed, there were no target organ HIs >1 for adults. For children, there were no target organ HIs >1 when the fish species consumed was Burbot, Northern Pike, Rainbow Trout, Smallmouth Bass, White Sturgeon, sucker, or whitefish. There were target organ HIs >1 at one house DU for the skin/hair/nails system when the fish consumed was kokanee (Table 5-25) or Walleye (Table 5-31) (Figure 5-18).

Based on some target organ HIs being >1, risks of non-cancer health effects from exposure to non-lead COPCs for the adult and child current resident receptor population exceeded the acceptable risk range (NCP, 1990).

Cancer risk for the current resident population, summed across COPCs by exposure pathway and summed across pathways, is shown in Tables 5-117 through 5-125 for the RME and CTE scenarios. For the RME scenario, inhalation of outdoor and indoor air, consumption of individual fish species, and/or incidental ingestion of outdoor soil and indoor dust plus dermal contact with outdoor soil, did not result in exceedances of cancer risk benchmarks by themselves. This was also true for the CTE scenario. When the four exposure pathways were summed, no TWA cancer risk benchmarks were exceeded at the RME or CTE.

5.2.5.2 Risks to Current Residents with Beach DUs

The exposure pathways evaluated for current residents with beach DUs were:

- inhalation of outdoor air,
- inhalation of indoor air,
- dermal contact with outdoor soil and incidental ingestion of outdoor soil and indoor dust,
- dermal contact with and incidental ingestion of surface sediment,
- dermal contact with and incidental ingestion of UCR surface water, and
- consumption of fish caught from the UCR evaluated by species.

Risks were evaluated on a DU-by-DU basis for each of the 21 residential beach DUs sampled in 2014 and 2016 excluding one CCT tribal allotment beach.

Non-cancer HIs aggregated by target organ system for the current adult and child resident-with-beach population, both summed across COPCs by exposure pathway and summed across pathways, are shown in Tables 5-126 through 5-167 for each residential beach, for the RME and CTE scenarios. For the RME scenario, no exposure pathways resulted in target organ HIs >1 for adults. The only exposure pathways with target organ HIs >1 for children under the RME scenario were consumption of individual fish species other than Northern Pike and White Sturgeon. For the child RME scenario, consumption of Burbot, kokanee, Rainbow Trout, Smallmouth Bass, sucker, Walleye, and whitefish had target organ HIs >1 for the developmental, nervous, reproductive, and/or skin/hair/nails systems (HIs ranged from 2 to 4 depending on the species).

When all exposure pathways were summed using RME exposure assumptions, target organ HIs were >1 for the adult at each residential beach for the nervous system when sucker was the fish species consumed (Tables 5-126 through 5-146). For children, when all exposure pathways were summed using RME exposure assumptions, target organ HIs were >1 at each residential beach for the developmental, nervous, reproductive, and/or skin/hair/nails systems, depending on the fish species consumed. For the CTE scenario, none of the exposure pathways evaluated had target organ HIs >1 by themselves for adults or children regardless of the fish species consumed

(Tables 5-147 through 5-167). When exposure pathways were summed, no target organ HIs were >1 for adults or children at any residential beach.

Based on some target organ HIs being >1, risks of non-cancer health effects from exposure to non-lead COPCs for this receptor population exceeded the acceptable risk range (NCP, 1990).

Cancer risks for the current resident-with-beach population, summed across COPCs by exposure pathway and summed across pathways, are shown in Tables 5-126 through 5-167 for the RME and CTE scenarios. For the RME and CTE scenarios, no exposure pathways evaluated for the resident-with-beach population exceeded a TWA cancer risk of 10^{-4} either separately or when they are summed across pathways.

5.2.5.3 Risks to Potential Future Residents

Risks were evaluated for the adult and child potential future resident population on an ADA-by-ADA basis for each of the 142 upland ADAs sampled in 2014. The exposure pathways evaluated for future residents were:

- Inhalation of outdoor air,
- Inhalation of indoor air,
- Dermal contact with upland soil and incidental ingestion of upland soil and indoor dust, and
- Consumption of fish caught from the UCR evaluated by species.

Non-cancer HIs aggregated by target organ system for the potential future adult and child resident population, both summed across COPCs by exposure pathway and summed across pathways, are shown in Tables 5-37 and 5-54 for the RME and CTE scenarios. For the RME scenario, inhalation of outdoor air and inhalation of indoor air do not result in exceedances of target organ HIs for the adult or child. Incidental ingestion of outdoor soil and indoor dust plus dermal contact with outdoor soil did not result in any target organ HI exceedances for adults. For children, the soil/dust exposure pathway resulted in target organ HIs >1 for the skin/hair/nails systems at 2 ADAs (HIs = 2).

No target organ HIs exceeded 1 for adults due to consumption of any fish species evaluated for the RME scenario. No target organ HIs exceeded 1 for children due to consumption of Northern Pike or White Sturgeon at the RME. Consumption of Burbot under the RME scenario resulted in target organ HIs >1 for the developmental and nervous systems in children (Table 5-37). Consumption of kokanee (Table 5-38) and Rainbow Trout (Table 5-40) by children resulted in the skin/hair/nails target organ system HI >1. Consumption of Smallmouth Bass and Walleye resulted in target organ HIs >1 for the developmental, nervous, and skin/hair/nails systems in children (Tables 5-41 and 5-44, respectively). Sucker consumption by children (Table 5-43) resulted in developmental, nervous, and reproductive target organ system HIs >1. Consumption of whitefish (Table 5-45) resulted in target organ HIs >1 for the reproductive and skin/hair/nails systems for children under the RME scenario.

When these four exposure pathways were summed together for the RME scenario, the target organ HI for adults was >1 for the nervous system at all 142 ADAs when the fish species consumed was sucker (Table 5-43). For children, at least some ADAs had non-cancer target organ HIs >1 for each fish species consumed (Tables 5-37 through 5-45; Figures 5-19 through 5-22). Target organ systems exceeding non-cancer benchmarks for children due to fish consumption included the developmental, nervous, reproductive, and skin/hair/nails systems.

For the CTE scenario, no exposure pathways by themselves had target organ HIs >1 for adults or children (Tables 5-46 through 5-54). When the four exposure pathways were summed, no target organ HIs were >1 for adults. For children, two ADAs had target organ HIs >1 for the nervous system when sucker was the fish species consumed (Table 5-52), and one ADA had a target organ HI >1 for the skin/hair/nails system when Walleye was the fish species consumed (Table 5-53) (Figure 5-23).

Based on some target organ HIs being >1, risks of non-cancer health effects from exposure to non-lead COPCs for this receptor population exceeded the acceptable risk range (NCP, 1990).

Cancer risks for the potential future resident population, summed across COPCs by exposure pathway and summed across pathways, are shown in Tables 5-168 through 5-176 for the RME and CTE scenarios. For the RME scenario, none of the four exposure pathways evaluated exceeded cancer risk benchmarks by themselves, or summed across a lifetime, either for adults or children (Table 5-133). This was also true for the CTE scenario. When the four exposure pathways were summed, no TWA cancer risk benchmarks were exceeded at the RME or CTE.

5.2.5.4 Risks to Outdoor Workers

The exposure pathways evaluated for the outdoor worker population were:

- Inhalation of outdoor air,
- Incidental ingestion of and dermal contact with surface water,
- Incidental ingestion of and dermal contact with surface sediment or soil at public beaches, and
- Incidental ingestion of and dermal contact with subsurface sediment or soil at public beaches.

Non-cancer HIs aggregated by target organ system for the adult outdoor worker population, both summed across COPCs by exposure pathway and summed across pathways, are shown in Tables 5-177 and 5-178 for the RME and CTE scenarios, respectively. Using both RME and CTE exposure assumptions, none of the exposure pathways, either alone or summed together, had target organ HIs that exceed the benchmark of one. The same is true of cancer risks: whether evaluated by exposure pathway or summed across exposure pathways, estimated adult cancer risks were all $<10^{-4}$. These risks were below non-cancer hazard benchmarks and within the acceptable cancer risk range used by CERCLA (EPA and Clay, 1991; EPA, 1997).

5.2.5.5 Risks to Recreational Visitors

The adult and child recreational visitor population was evaluated for exposure to non-lead COPCs via the following exposure pathways:

- Consumption of fish caught from the UCR evaluated by species,
- Inhalation of outdoor air on beach trips, boating trips, and camping trips,
- Incidental ingestion of and dermal contact with surface water on beach trips, boating trips, and camping trips,
- Incidental ingestion of and dermal contact with public beach surface sediment or soil, or relict floodplain surface soil, on beach day trips, boating trips, and camping trips.

Risks were evaluated on a beach-by-beach, SDU-by-SDU, RFDA-by-RFDA, or UDU-by-UDU basis.

Non-cancer HIs aggregated by target organ system for the adult and child recreational visitor, both summed across COPCs by exposure pathway and summed across pathways, are shown in Tables 5-179 through 5-308 for each individual public beach, SDU, RFDA, and UDU for the RME and CTE scenarios. For the RME scenario, no individual exposure pathways for adults had target organ HIs >1. For children under the RME scenario, the only exposure pathway with target organ HIs >1 was consumption of fish (for most of the individual fish species evaluated). For the child RME scenario, consumption of each fish species evaluated except Northern Pike and White Sturgeon has at least one of the following target organ HIs >1: developmental (HIs ranged from 2 to 3), nervous (HIs ranged from 2 to 3), reproductive (HIs = 2), and/or skin/hair/nails systems (HIs ranged from 2 to 4).

When all exposure pathways were summed using RME exposure assumptions, no target organ HIs were >1 for the adult recreational visitor at any public beach, SDU, relict floodplain, or UDU (regardless of trip type) (Tables 5-179 through 5-243). For children, when all exposure pathways were summed using RME exposure assumptions, no target organ HIs >1 at any public beach, SDU, relict floodplain, or UDU (regardless of trip type) when the fish species consumed was Northern Pike or White Sturgeon. Depending on the remaining fish species consumed, target organ HIs were >1 at each public beach, SDU, relict floodplain, or UDU (regardless of trip type) for the developmental, nervous, reproductive, and/or skin/hair/nails systems for child recreational visitors under RME exposure assumptions (HIs ranged from 2 to 4).

For the CTE scenario, none of the exposure pathways evaluated had target organ HIs >1 by themselves for adults or children at any public beach, SDU, RFDA, or UDU, regardless of trip type (Tables 5-244 through 5-308). When exposure pathways were summed, no target organ HIs were >1 for adults or children.

Based on some target organ HIs being >1, risks of non-cancer health effects from exposure to non-lead COPCs for the recreational visitor receptor population exceeded the acceptable risk range (NCP, 1990).

Cancer risk for the recreational visitor population, summed across COPCs by exposure pathway and summed across pathways, are shown in Tables 5-179 through 5-308 for the RME and CTE scenarios. Under both RME and CTE exposure assumptions, no exposure pathways evaluated for any of the trip types exceeded a TWA cancer risk of 10^{-4} either separately or when they are summed across pathways for any public beach, SDU, relict floodplain, or UDU.

6 UNCERTAINTY ANALYSIS

This section discusses and, where possible, estimates the direction and magnitude of uncertainty associated with potentially influential parameters used to estimate risk.

Risk estimates reported in this HHRA are derived from combining exposure and toxicity estimates for non-cancer and cancer effects. Uncertainty is usually accounted for in risk estimates by making health-protective assumptions in estimating the parameters used in risk calculations. Health-protective assumptions result in risk estimates that err on the side of overestimating actual exposures and risk. These assumptions can take various forms, depending on the specific parameter. Examples include UFs in dose-response relationships that are part of the process for using chemical-specific toxicity studies to derive toxicity values (RfCs, RfDs, and cancer slope factors),[93] UCLs on estimates of mean exposure concentrations measured in environmental media, and upper percentile estimates of exposure factors that are used to estimate RMEs for receptor populations. The toxicity estimates for arsenic and lead are notable exceptions and are discussed below.

As described by EPA (2014c; see Section 4.3), areas of uncertainty that may make an appreciable difference in the risk assessment results or conclusions are appropriate topics for an uncertainty discussion. Influential parameters that contribute to exposure estimates that exceed risk benchmarks are a particularly important focus for uncertainty discussion because consideration of uncertainty in these parameters can inform risk management decisions. Influential parameters are those that have the largest effect on the risk estimate when varied across a plausible range, as determined by Site-specific information that informs that range of possible values.

Uncertainties can be categorized as follows:

1. Uncertainties that can be quantified
2. Uncertainties that cannot be quantified, but the direction of the effect of the uncertainty can be determined: as an underestimation or an overestimation of risk
3. Uncertainties that cannot be quantified and the direction of the effect of the uncertainty is also unknown

An example of the first category is the uncertainty associated with chemical exposure concentrations at the Site. The uncertainties in the concentrations can be quantified as confidence limits on the mean exposure concentration. For fish, numerous species and individuals were collected and analyzed. Each sample consisted of between five and eight individual fish. Sample sizes ranged from 19 to 51 depending on the species and analyte (Table 3-29) and resulted in a robust estimate of the EPC for COPCs in fish tissue. For soil, uncertainties in the exposure concentrations are very low because many DUs and ADAs were sampled in triplicate, with 30 soil increments collected per sample, and with very similar results between the replicates.

[93] For more information see https://www.epa.gov/iris/basic-information-about-integrated-risk-information-system.

An example of the second category of uncertainty is estimation of risk-based concentrations or toxicity values (e.g., oral SF, RfD, RfC, IUR) used to calculate HQs or cancer risks. There are varying degrees of uncertainty associated with toxicity values. Uncertainties in toxicity values can arise from the following sources:

- Extrapolation from animal studies to humans
- Extrapolation from high dose to low dose
- Extrapolation from continuous exposure to intermittent exposure
- Limited or inconsistent toxicity studies

These sources of uncertainty are accounted for in the derivation of toxicity values by applying UFs that decrease the toxicity value, even though it is likely that the uncertainty range includes higher values. This approach results in downward bias of the toxicity value and, as a result, uncertainties in toxicity values will contribute to overestimation rather than an underestimation of risk.

Two notable exceptions in this HHRA include arsenic and lead. Toxicity values for arsenic are based on a substantial body of epidemiology studies of human populations. Arsenic has been the subject of multiple EPA toxicity evaluations but has not been revised since 1989. Although lead lacks a toxicity value, it has no safe level of exposure (NTP, 2012). Rather than assessing lead risk from a toxicity value (e.g., a not-to-exceed lead intake rate), lead risks in young children are estimated from application of the IEUBK Model for Lead in Children, which predicts a distribution of PbBs for a population of children for user-defined Site-specific exposures and bioavailability. This approach eliminates the need for some of the health protective assumptions built into toxicity values for other chemicals.

An example of the third category of uncertainty is exposure to chemicals for which there are no toxicity values. For these chemicals, we cannot determine whether they contribute to risk at the measured exposure levels. The uncertainty associated with chemicals that lack toxicity values cannot be quantified and the direction of the effect of the uncertainty is unknown. These chemicals, which were detected in media at the Site but not analyzed as COPCs because of the lack of toxicity values, included acenaphthylene, alpha-benzenehexachloride, benzo(e)pyrene, benzo(g,h,i)perylene, bismuth, 4-bromophenol-phenylether, carbazole, cerium, cesium, 4-chlorophenyl-phenyl ether, delta-BHC, 1,3-dichlorobenzene, 1,4-dichlorobenzene, dimethyl phthalate, dysprosium, endosulfan I, endosulfan II, endosulfan sulfate, endrin aldehyde, endrin ketone, europium, fluoride, gadolinium, gallium, gamma-chlordane, germanium, gold, hexachlorobutadiene, indium, lanthanum, lutetium, neodymium, 3-nitroaniline, 2-nitrophenol, 4-nitrophenol, cis-nonachlor, trans-nonachlor, oxychlordane, phenanthrene, praseodymium, radium-226, rubidium, samarium, scandium, silicon, sulfide, sulfur, total NTV PBDEs, thorium, titanium, uranium-238, and yttrium.

6.1 Site-Specific Uncertainty Considerations

At this Site, numerous studies were conducted to collect Site-specific information to reduce the uncertainty in the risk estimates, primarily uncertainty associated with the following parameters:

concentrations of chemicals in environmental media (abiotic and biotic), and human exposure factors. The use of Site-specific estimates of EPCs and exposure factors in risk calculations can help reduce the uncertainty in the risk estimates (EPA, 1989). Site-specific EPCs can be obtained by sampling Site media, and exposure estimates can be informed by studying the local population (e.g., through surveys).

In some media, the concentration of COPCs represent a snapshot in time and future conditions could vary that would alter the concentration. For example, measured COPC concentrations in the <250 µm particle size fraction of sediment for beaches and floodplains does not necessarily account for the potential for weathering or redistribution of metal-enriched slag along the river. As discussed in the Introduction (Section 1) of this HHRA, the risks described herein are based on the data currently available.

For this HHRA, quantitative risk estimates were derived only for COPCs: COIs that exceeded RBSLs (see COI Screen Section 3.2 for more information). Chemicals that were measured in Site samples but were not retained as COPCs may contribute a small amount of added risk, but their contribution is expected to be so small that this is not a significant source of uncertainty.

Additionally, quantitative risk estimation is only possible for COPCs with toxicity values. Chemicals without toxicity values may contribute to total Site risk, but the magnitude of their potential contribution is unknown.

This uncertainty assessment considers all COPCs. Some COPCs identified in this uncertainty section are inorganic chemicals that are naturally occurring in the environment. Additionally, some inorganics are essential nutrients for humans, animals, and plants. In some cases, essential nutrients that were retained as COPCs contributed to HIs which exceeded risk benchmarks. Information is presented below concerning the ADI, the toxicity value, and recommended dietary allowance (RDA) for these nutrients for which calculated exposure as part of an HI exceeded risk benchmarks.

The uncertainties associated with lead and non-lead COPCs are discussed in the following sections (6.2 and 6.3, respectively). Although these are described in separate sections, some of the concepts apply to both lead and non-lead COPCs (for example, Sections 6.3.1.1 and 6.3.1.2 also apply to lead).

6.2 Uncertainty in Lead Risks

As discussed in Section 3, lead risks were calculated based on predictions of blood lead concentrations expected to result from estimated Site exposures. In order to predict the geometric mean PbB (which is the basis for risk predictions), the rate of absorption of lead from all exposure sources must be calculated and the distribution and elimination of lead from the body must be calculated. This is achieved by the IEUBK model using inputs of measured lead EPCs in air and soil, measured soil lead bioavailability; and assumptions about lead concentrations in drinking water and lead intakes from diet. Similarly, the ALM evaluation of outdoor workers incorporated Site-specific exposure, concentration, and bioavailability information in the assessment of sediment and soil at the surface and at depth. Uncertainties apply to evaluation of

lead exposures/risks in general; uncertainties associated with the IEUBK model are generally applicable to the ALM.

Quantification of risks from exposures to lead is subject to data limitations and uncertainties. The most important factors at the Site are summarized below. Because of these uncertainties, the probabilities (P values) reported above should be understood to be estimates; however, this analysis is based on adequate Site characterization (for example, the EPCs for lead are based on comprehensive sampling of representative areas where exposure is likely to occur) and realistic, Site-specific inputs for other exposure variables for the assessment. Furthermore, by using a risk range (P3, P5, P8), risk managers are presented with information that can support a decision. This section presents the uncertainties in lead risk assessment for this Site and discusses those that are likely to influence risk-based decisions.

Uncertainties in blood lead predictions (and corresponding lead risk calculations and PRGs) come from numerous sources:

1. Uncertainty in estimates of parameters measured directly at the Site: soil lead concentrations and bioavailability; recreational exposure frequencies and durations. Some of these uncertainties can be described with confidence limits based on sampling results. These are discussed in greater detail below.

2. Uncertainties related to the assumption that exposure parameter values estimated in other populations apply to the Site (e.g., the default soil ingestion rates and dietary lead intakes from national estimates). These uncertainties cannot be quantified, since we have no direct measurements of these parameters at the Site; however, the potential impacts of uncertainty on risk estimates can be evaluated with sensitivity analysis. The sensitivity analysis for the IEUBK model (EPA, 1999) showed that IEUBK model results are most sensitive to the amount of soil ingested per day (soil-dust ingestion rate). The IEUBK model is moderately sensitive to several other variables, including absorption fractions for dust, diet and soil; soil lead concentration; indoor dust lead concentration; lead in the diet; and contribution of outdoor soil lead to indoor dust lead (MSD). These are discussed in greater detail below.

3. Uncertainties in the IEUBK model representation of lead biokinetics in young children. Biokinetics in the IEUBK model are intended to represent the relationship between lead absorption rate and blood lead in a “typical individual”. Expected variability in this relationship is represented with a probability distribution (as determined by the GSD variable), which is also based on variability in blood lead observed in other populations. Uncertainties in the prediction of blood lead at the Site cannot be quantitatively addressed since we lack direct measurements of the absorption - blood lead relationships for the Site population. These uncertainties are not discussed further in this assessment, because the uncertainty in blood lead predictions made with the IEUBK model has been determined to be acceptable for Site-specific risk assessment and is discussed elsewhere (Science Advisory Board [SAB], 1990, 1992, 1998; EPA, 1994a, 1998b; Hogan et al., 1998; von Lindern et al., 2016; NAS, 2005).

The methodology for lead risk calculations is based on exposure to a "typical child" and with a few exceptions (primarily Site-specific media lead concentrations such as soil lead concentrations in DUs or ADAs, but also Site-specific media intake rates such as fish consumption) use national default values for risk calculations. To the extent that these represent average or typical exposures for the Site, the IEUBK model results may be considered applicable.

6.2.1 Uncertainty in Measured Inputs

6.2.1.1 Uncertainty in Average Lead Concentrations

EPCs for lead in residential soil were calculated using data from the IC surface soil samples collected within a DU. IC sampling entails the collection of multiple individual volumes of soil ("increments") from a target area (DU) that are composited and subsampled. The UCR surface soil samples were collected using 30 increments per IC sample. IC was selected as the sampling strategy because it provides a cost-effective alternative to discrete sampling when the objective is to estimate the mean concentration for a DU (Hathaway et al., 2008).

For these exposure pathways, full-time residential exposure was assumed (which may overestimate exposure for those DUs that are not used daily). There is some uncertainty in the assumed mass transfer of outdoor soil to indoor dust (the MSD term in the IEUBK model; see Section 6.2.1.7); however, using the <149 µm particle size fraction would be expected to increase the correlation in the relationship between outdoor soil concentrations of lead and indoor dust concentrations of lead due to the focus on fine particles which are more mobile in the environment. To inform risk management decisions, separate counts based on the DU type are provided (i.e., for house and play area DUs as well as other DU types) for selected target PbBs (Section 5.1.1).

For lead, risk estimates associated with current residential exposure to soil were conducted with Site-specific sampling data that were obtained in accordance with the DQOs (SRC, 2014a; TAI, 2016b). Also, soil lead EPCs are based on ICs for each DU and the DUs were established based on use information from interviews with the current residents. For future residential exposure, soil lead EPCs are based on ICs for each ADA; however, there is some uncertainty in the exposure area (the location of the future residence within the ADA is not known and is likely limited to approximately 1-5 acres within the larger ADA area [approximately 25 acres]).

The mean lead concentration in each environmental medium is used in the exposure and risk calculations to represent the average exposure concentration for a DU or ADA. IC sampling was used for soil and some sediment data sets to increase confidence in the estimate of the mean. The precision achieved using the ICS method is summarized in Table 6-1 using the margin of error (ME) and relative error (RE) of the means for DUs that had 3 ICS results. The ME equals one-half the width of the confidence interval (CI) for the mean (i.e., [95UCL-mean]/2) and the RE expresses the ME as a percentage of the estimate of the mean (i.e., ME/estimate of the mean). The true average concentration within a DU could be above (or below) the estimated mean, but it is unlikely (<5% chance) that it is above (or below) the mean by more than the ME. The median ME (RE) for all DU types combined was 22 ppm (14%). The median ME ranged from 6.4 ppm (gardens) to 47 ppm (CCT tribal allotment soil) and the median RE for each DU type was less than 20% (excluding the soil DU at Bossburg Flat Beach). Across all DU types, 95% had an RE

less than 50%. Because soil and sediment exposure are the principal contributors to lead exposure, careful characterization of these media EPCs is particularly important in reducing uncertainty in lead risk calculations.

6.2.1.2 Uncertainty in Site-Specific Exposure Estimates for Recreational Visitors

As noted in the Data Analysis Report (DAR) for the RecUse Survey (SRC, 2019a), there is uncertainty in exposure frequency for beach visits because of an extreme drawdown during the survey that resulted in the loss of a beach interview location and a corresponding loss of data. While there is uncertainty in the actual exposure frequency and duration for on-Site recreational visitors, the best available information was used in the risk assessment calculations (based on a comprehensive Site-specific Recreational Use Survey (IEC, 2012, 2013a). Risks would be higher for more frequent users. Another source of uncertainty in the results is the assumption of Bossburg Flat beach exposure by beach, boating, or camping visitors. Bossburg Flat beach is currently closed; however, because it is unknown how long the closure will continue, this assessment assumed that Bossburg Flat beach was open for recreation. For recreational visitors, the principal contributing media are soil and sediment (including sediment contaminants that are taken up by fish). The principal media contributing to lead exposure for adult outdoor workers are residential soil (which is accounted for in the ALM in baseline blood lead) and Site sediment, with smaller contributions from surface water and fish.

6.2.1.3 Uncertainty in IEUBK Model Inputs

Potential sources of uncertainty in model predictions include the use of surrogate variables, missing variables that should have been included, abnormal conditions, and incorrect model forms. This is of special concern in lead risk assessment, as pathways of lead exposure have both direct effects (from contact with contaminated media) and secondary impacts such as the soil and paint contributions to house dust lead. Failure to correctly specify these variables can lead to uncertainties in interpreting quantitative results. For this reason, the DQOs for data collection at this Site (for both media COPC concentrations and media contact or ingestion rates where applicable) were rigorous in terms of data quality to ensure that representative data were obtained that allow for adequate characterization of exposure for risk assessment.

The IEUBK model was developed to predict blood lead levels (BLLs) for a child based on exposure to lead in environmental media, including soil or sediment, dust, air, water, and food. As such, lead risks from surface water alone, for example, cannot be presented separately. To illustrate how consumption of UCR surface water contributes to BLLs, the IEUBK model was run for children recreating on a residential beach and consuming fish from the UCR (also including exposures to residential soil, dust, diet, residential water, and air), but not incidentally ingesting UCR surface water while swimming. Results for this analysis using the P5 benchmark are shown in Table 6-2. The difference in the probability of exceeding P5 varies by residential beach DU but is generally very small (less than 0.5% for each DU except for DU 202 for all fish species but Walleye). For Walleye, the difference in the probability of exceeding P5 ranges from 0.2 to 2.4%. Incidental ingestion of UCR surface water while swimming has the greatest impact on the probability of exceeding P5 at residential beach DU 202 when Burbot is consumed (P5 is 10.2% with surface water ingestion and 8.8% without surface water ingestion) and when Walleye is consumed (P5 is 11.3% with surface water ingestion and 8.9% without surface water

ingestion) (Table 6-2). This analysis illustrates that lead in UCR surface water does not contribute substantially to overall lead risks to children.

6.2.1.4 Uncertainty in Site-Specific Bioavailability of Lead in Soil and Sediment

While IVBA analysis was used to assess bioavailability of lead in soil and sediment at this Site on an exposure unit basis (by DU, ADA, and beach), there is some uncertainty in applying the information obtained from those analyses to exposure units or areas where IVBA data were not obtained. As described in Appendix 11, the IVBA results do not vary greatly across media and soil studies, so this source of uncertainty is small.

6.2.1.5 Uncertainty in Soil and Dust Ingestion Rates

Ingestion of soil is the principal route of intake of soil lead. EPA has extensively reviewed the various studies and the associated variability and uncertainty in observed soil ingestion rates (EPA, 2017b). Overall, the current recommended selections for age-variable soil intakes for the most vulnerable subsets of children in the 12-72 month age range in the IEUBK model are based on consensus and are in the range of multiple studies using different methods of estimation (EPA, 2017b).

Actual incidental soil-dust ingestion rates are uncertain and may be higher or lower for any given individual than the default assumed in this assessment. Soil ingestion estimates in the IEUBK model are based on fifteen years of data amassed from a large residential study population based in northern Idaho, located within 200 miles of the UCR Study Area (von Lindern et al., 2016). Alternative soil-dust ingestion rates have been used with the IEUBK model for other non-HHRA site risk assessment applications (Zartarian et al., 2017); however, the authors of that publication acknowledge the limitations of the work: "*Although we simulated correlations in Pb exposure among dust, soil, and water (using NHEXAS [National Human Exposure Assessment Survey] and HUD [U.S. Department of Housing and Urban Development] data), stratified data by housing age, and assessed BLL [blood lead concentration] at upper percentiles of the BLL distribution, our current analyses are not focused on specific at-risk populations, such as Flint, Michigan, and East Chicago, Indiana, or other environmental justice communities or homes with high Pb in soil, dust, or water*." The soil-dust ingestion rates used herein are the values currently recommended for site-specific HHRA lead risk assessment by the TRW Lead Committee.

To account for wet soil and sediment having a greater tendency to adhere to hands, there are several approaches for assessing sediment exposure. For this Site, instead of adjusting soil-dust ingestion rates (as described in EPA, 2003a), a larger particle size fraction was used for sediment (<250 µm) than was used for soil (<149 µm).

6.2.1.6 Uncertainty in Partitioning Ingestion Rate to Assess Soil and Dust Exposure

While contact and subsequent incidental ingestion of outdoor soil contributes to overall soil and dust exposure, it is generally accepted that house dust is the proximate environmental pathway medium for lead exposures of infants and toddlers (Mushak, 1998; Succop et al., 1998). EPA has evaluated the soil ingestion rate studies conducted to date and recommended an appropriate partition for outdoor soil and indoor dust ingestion rates for lead with a ratio of 45% of the intake coming from outdoor soil and 55% from indoor dust (EPA, 1994b).

6.2.1.7 Transport of Outdoor Soil or Sediment into the Residence

In the absence of indoor sources of lead (such as deteriorating lead-based paint), outdoor soil lead is generally the primary source of lead in household dusts. The EPA default assumption in the IEUBK model is that the concentration of lead in indoor dust is 70% that of the outdoor yard soil lead concentration (along with a small contribution from outdoor air; this is the MSD). The TRW Lead Committee has recommended that site-specific information used to support changing the MSD at specific sites should not be considered representative of the conditions at other sites unless site conditions (e.g., proximity, climate, housing type, socioeconomic status, grass cover) are demonstrated to be sufficiently similar. Lacking Site-specific indoor dust data (that would be obtained through vacuum sampling of residences at the Site), this default assumption was used. Some studies conducted at other sites have suggested the default assumption to be too high (Brattin and Griffin, 2011); however, these studies have not been determined by EPA to be generalizable to sites other than those included in the analysis.

In the absence of measured indoor dust lead concentration data, the default MSD was used. This value likely over predicts indoor dust lead concentration as shown at other sites (Schoof et al., 2019); however, Site-specific information is not available from the UCR Site to derive an alternative Site-specific estimate applicable to the UCR.

6.2.1.8 Dietary Lead Intake

Dietary intake of lead for this assessment was based on the TRW Lead Committee's most recent recommendation (EPA, 2019c). As described in that report, the recommended default values are based on national food supply monitoring data and population-based data for dietary intake. Alternative estimates of dietary intake for lead exist (e.g., Zartarian et al., 2017) suggesting the TRW Lead Committee's recommended values may slightly overestimate dietary intake. Appendix 17 shows a comparison between the two alternative dietary assumptions. The recommended TRW Lead Committee values for dietary lead intake assume most calories are obtained from grocery stores, rather than home gardens. It is not known whether these national estimates over or underestimate estimates for the Site population. In general, lead in food is a minor contributor of lead intake as compared to soil and dust sources, so dietary lead intake is not considered a principal source of uncertainty. The gardening exposure scenario was not employed in this HHRA because those residences with acceptable concentration of lead in outdoor soil are generally considered acceptable for unlimited use and unlimited exposure (including gardening) (EPA, 2014b). Evaluating garden DUs for full-time residential exposure, as was done in this HHRA, is a health-protective exposure assumption.

6.2.1.9 Incremental Exposures and Time Weighting

The methodology used to assess recreational exposure pathways described in Chapter 5 of this HHRA is based on TRW Lead Committee recommendations (EPA, 2003a). There is some uncertainty in this approach as described in the guidance, primarily arising from the adjustment of the frequency of exposure, which must be increased from approximately 6 days (mean value from the RecUse Survey; SRC, 2019a) over a 90-day period to 13 days over a 90-day period (or once per week) to meet the minimum requirements of the IEUBK model. To assess the magnitude of uncertainty introduced by this adjustment, the AALM was used to predict PbBs resulting from 6 days over a 90-day period and these results were compared to the IEUBK model results. The details of that analysis are described in Appendix 15. As discussed in that appendix,

the adjustment to the exposure frequency necessary to use the IEUBK model is small and unlikely to influence risk-based decisions.

6.2.1.10 Uncertainty in Residential Use and Future Use Scenario Assumptions

Exposure to soil at this Site assumed full-time residential use of residential DUs that are not house DUs and for upland ADAs without residences. This simplifying assumption overestimates exposure for DUs and ADAs that are not used regularly by young children. This overestimate must be balanced with the possibility that future development may occur and the need to inform risk decisions for the near and long term. Because the Site is near a National Recreation Area, it is possible this area could experience pressure for land use change in the future, including residential expansion (both year-round and seasonal). Nevertheless, many of the upland areas are presumably unavailable to residential development because they are protected by government ownership/management. The exposure assumptions used herein are provided to enable risk managers considering the risks associated with future residential development to inform risk reduction strategies for these areas. The difference in soil lead PRG for residential land use versus for non-residential land use is quite large (ranging from 5- to 10-fold) as shown in Table 6-3. If these non-residential soil lead PRGs were used for land that is later developed, it is likely that the risk to children would be unacceptable. The respective child risk estimates for each of these non-residential land use PRGs are shown in Table 6-4. PRG calculations are detailed in Appendix 16.

6.2.2 Summary of Uncertainty in Lead Risk Assessment

The principal areas of uncertainty in lead risk assessment modeling for this Site are: (1) mass of ingested soil and dust; (2) partitioning ingestion rate to assess soil and dust exposure; and (3) transport of outdoor soil into the residence. These contributors to uncertainty are important to identify for the following reasons: (1) these parameter values are based on the national defaults from other populations; (2) their applicability to the UCR population is an assumption, and (3) these parameters have been demonstrated to strongly influence risk calculation results (EPA, 1999). The use of default assumptions for these parameters would be unlikely to underestimate risk in this HHRA.

6.3 Uncertainty in Risks from Exposure to Non-Lead COPCs

6.3.1 Uncertainty in EPC Estimates

All studies conducted to support the HHRA followed the EPA DQO and Uniform Federal Policy (UFP)-QAPP processes to ensure that field and laboratory methods produced environmental data of sufficient quality and quantity to support the risk assessment. Some uncertainty is always present in estimates of EPCs because of sampling variability. In accordance with the DQOs and QAPP, this source of uncertainty is managed by using the 95UCL of the mean as the EPC (or a surrogate value if a 95UCL cannot be calculated).[94] This tends to minimize the likelihood of underestimating the exposure concentration but may result in an overestimation of actual exposures. Analytical error is another potential source of uncertainty in the estimation of Site-specific EPCs. However, because Site-specific data were collected and analyzed in accordance

[94] See Section 6.3.1.3 and Appendix 12 for a description of how surrogate values were derived for soil samples.

with the UFP-QAPP, analytical error is expected to be one of the smaller sources of uncertainty in this risk assessment.

6.3.1.1 Surface Water EPCs

6.3.1.1.1 Disturbed versus Non-Disturbed Samples

Human exposures to surface water may occur under a variety of exposure scenarios. In most instances, on-Site exposures are expected to occur primarily in shallower water near beach areas (*e.g.*, recreational visitors during wading/playing activities, outdoor workers during occupational activities) which may be directly influenced by sediment disturbances resulting from these activities. Data which represent disturbed surface water from shallow areas near beaches was identified as a data gap in the HHRA Work Plan (SRC, 2009). The unfiltered, disturbed surface water samples collected in shallow water near beach areas were used to evaluate exposure to surface water during all recreational and occupational activities in this HHRA. The EPCs from the disturbed shallow water samples provide a conservative estimate of exposure from swimming in deeper water where disturbance of the underlying sediment would not be expected. The EPCs calculated for unfiltered, undisturbed surface water samples were always lower than for unfiltered, disturbed samples. Use of the disturbed, unfiltered surface water data to evaluate risk from swimming in deeper water may overestimate risk from this exposure pathway.

6.3.1.1.2 All Regions Combined versus Reach by Reach or Region by Region

The surface water EPCs were calculated using data from all disturbed, unfiltered samples collected from UCR Reaches 1 through 6. The rationale for combining the surface water data to estimate one EPC for the Site was based on the number of samples available for each reach of the UCR (n = 4 to 6). Surface water samples were collected from one transect per river reach. Each reach of the UCR includes 12 to 59 miles of surface water. The differences in the numbers of beach trips per year, and the time spent swimming and wading in water more than waist deep during beach trips, are not substantially different among the three regions of Lake Roosevelt ("upper," "middle," and "lower"; SRC, 2019a; Figure 3-4). Therefore, a Site-overall EPC for surface water was calculated (i.e., exposure to COPCs via incidental ingestion of surface water while swimming).

6.3.1.2 Sediment EPCs

6.3.1.2.1 RBA and Non-Lead COPC EPC Estimates Calculated for Beaches Sampled in 2009 and 2010

All sediment samples collected from beaches sampled in the 2009 – 2011 beach sediment study were sieved to <2 mm. A single surface sediment composite from each beach was randomly selected and further sieved into four size fractions: <2 mm to 250 µm, <250-125 µm, <125-63 µm, and <63 µm. Analyses for metals and IVBA of arsenic and lead were conducted on each of these finer fractions. In 2013, EPA requested that the archived samples from beaches sampled in 2011 be reanalyzed for arsenic and lead and IVBA of lead to verify that results based on estimated means from the three size fractions (<250-125, <125-63, and <63 µm) were comparable to measured results on sediment sieved to <250 µm. Five beaches were included in the reanalysis: Bossburg Flat, Evans Campground, Flat Creek, Lyons Island, and Swimming Hole. Archived samples were sieved to <250 µm and submitted for analysis of lead, arsenic, and

IVBA of lead (EPA, 2013a). The RPDs ranged from 7 to -82% for arsenic and from -4 to -74% for lead (negative values indicate the 2013 measured RBA-adjusted mean was less than the estimated RBA-adjusted mean). The RPDs for lead and arsenic each exceeded the 35% decision criterion (EPA, 2013a) for two of the five beaches. However, the estimated RBA-adjusted mean lead concentration exceeded the mean of the measured RBA-adjusted concentration for each of the five beaches while the mean of the measured RBA-adjusted arsenic concentration exceeded the estimated mean of the RBA-adjusted arsenic concentration at each of the beaches except Bossburg Flat.

There is uncertainty associated with the calculated sediment EPCs for COPCs other than lead and arsenic in beach sediment samples collected in the 2009-2011 beach sediment study and the direction and magnitude of this uncertainty is unknown. However, the results of the 2013 reanalysis indicate the estimated means may be biased high.

6.3.1.2.2 Subsurface Sediment and Soil EPCs

Outdoor workers may be in contact with subsurface sediment and beach soil at public beaches during occasional occupational activities such as digging holes, trenches, and footings, which can lead to the incidental ingestion of small amounts of sediment or soil. Subsurface sediment data for the <250 µm fraction are only available for the 2015 Bossburg Flat and Evans Campground SDUs and the former cable ferry landing location (F-01), and subsurface soil data for the <149 µm fraction are only available for the 2015 Bossburg Flat UDUs. Quantitative risk calculations could only be done using subsurface data from that sampling event (TAI, 2016a).

Subsurface sediment data for the <2mm particle size fraction are available for public beaches where IC surface samples were also collected. The subsurface samples were collected from 0-15, 15-30, and 30-45 cm depth intervals with soil cores.[95] Table 3-2 compares the mean concentrations of COPCs in the 0-15 cm depth interval (surface sediment) to the concentrations in the entire 0-45 cm depth interval on a Site-wide comparison (comparisons for each beach are provided in Appendix 9). Table 3-2 shows that the Site-wide concentrations of COPCs in the subsurface sediment are similar to surface sediment concentrations. The magnitude and direction of uncertainty associated with exposure to the <250 µm subsurface sediment fraction at other public beaches is unknown, but it is likely that risk calculations conducted using EPCs from surface sediment samples at those beaches are sufficient to estimate risk from exposure to subsurface sediment. Subsurface beach soil data are not available for any public beach except Bossburg Flat at the Site.

6.3.1.3 Soil EPCs

EPCs for soil were estimated based on IC samples collected within each DU or ADA. The 95UCL t-statistic COPC concentration was identified as the EPC for each residential soil DU and upland ADA where triplicate ICS samples were collected. For residential soil DUs and upland ADAs where a single ICS sample was collected, the EPC was calculated by multiplying the sample result by a CF derived based on the 95UCL/mean ratio estimated for DUs with three composite samples (Appendix 12). Residential soil sampling DUs were not randomly located; they were selected based on high potential for contact with soil based on land use as determined

[95] ICS samples were also collected from the subsurface at DUs located at Bossburg Flat Beach and Evans Campground Beach.

from interviews of property owners and site inspections. ADAs were randomly located within three designated sampling areas; a primary area adjacent to the UCR and within the river valley (an area most likely to have received aerial deposition of contaminants emitted by smelters); a high density sampling area immediately downstream of the U.S. – Canada border (selected because of the perceived likelihood of higher historic deposition rates based on historic SO_2 vegetation damage attributed to the Teck Smelter); and a reserve sampling area located adjacent and to the east of the primary sampling area. Use of the 95% UCL or a surrogate COPC concentration within a DU or ADA as the EPC and collection of 30 increments for each composite sample will likely minimize the potential that the soil EPC is underestimated.

Soil EPC calculations for COPCs other than lead and arsenic assumed that 100% of the measured concentration of COPC was bioavailable. As part of the 2014 Upland Soil Study (TAI, 2015a), IVBA was measured in soil samples for TAL metals and molybdenum, as shown in Table 6-5. However, though current EPA guidance recognizes validated methods for assessing RBA of arsenic and lead in soils and soil-like materials for applications to HHRA at sites assessed by EPA (EPA, 2017f, 2020), IVBA data available for COPCs other than lead and arsenic have not been validated. Consistent with current EPA guidance, RBA adjustments made in this HHRA were limited to arsenic and lead. Risk estimates for other metals are overestimated to an unknown extent based on the assumption of 100% bioavailability. EPA Region 10 continues to work with the TRW Bioavailability Committee to review bioavailability research conducted for metals other than lead and arsenic.

6.3.1.4 Outdoor Air EPCs

The last Site-specific air data were collected in 2009, and air data were only collected at Sheep Creek monitoring station located near Northport. There is temporal uncertainty associated with the EPCs for COPCs in air because of the lack of current Site-specific data and there is spatial uncertainty with the air EPC, because data were only collected at the Sheep Creek monitoring station. Currently, air quality at the Trail smelter meets all applicable regulatory standards for air pollutants including lead and arsenic (Trail Area Health and Environment Program, http://www.thep.ca/pages/airquality). Figure 2-13 shows Trail, B.C., Canada arsenic, lead, and cadmium aerial emissions from 2002 to 2017 reported to the Canadian National Pollutant Release Inventory (https://open.canada.ca/data/en/dataset/1fb7d8d4-7713-4ec6-b957-4a882a84fed3). Emissions of arsenic and lead are lower than in years prior to 2009; however, individual metals may still be emitted at varying and sometimes higher rates. The magnitude of uncertainty associated with lack of air samples between the border and the Sheep Creek station is unknown. However, risk from inhalation of air is likely to be overestimated.

Based on emissions reported to the Canadian National Pollutant Release Inventory, COPC concentrations in air emissions have generally been decreasing over time. ECY (2017a, 2017b) estimates air concentrations south of the border based on air concentrations measured in Butler Park and Columbia Gardens, B.C., Canada. To further evaluate potential uncertainty in air concentrations between the Trail facility and Northport, EPA used methodology from the ECY (2017a) report to derive estimated air concentrations from data collected between 2012 and 2014 at the Columbia Gardens air monitoring station, which is located approximately 5 km north of the border (McAlpine, 2020). Based on evaluation of air monitoring data and applying concentration gradients determined in the ECY (2017a) report, EPA concluded that air

concentrations at the U.S. – Canada border can generally be assumed to be 80% of the concentrations measured at the Columbia Gardens site when accounting for the additional travel distance (Table 6-6) (McAlpine, 2020).

As shown in Table 6-6, the estimated lead concentration in air at the border (0.046 µg/m^3) is approximately twice as high as the lead concentration in air used in this HHRA (0.023 µg/m^3), but is still lower than the EPA National Ambient Air Quality Standards (NAAQS) of 0.15 µg/m^3. Estimated concentrations of arsenic at the border using more recent concentrations measured at Columbia Gardens have decreased from the air EPC used in this HHRA, while the cadmium concentration is the same (Table 6-6). To measure the effect of the estimated lead concentration at the border (0.046 µg/m^3) on risks to child residents, the IEUBK model was run for the current and potential future child resident populations. All input parameters were kept the same as those described in Section 3.4 except for the lead EPC, which was changed from 0.023 µg/m^3 to 0.046 µg/m^3 (McAlpine, 2020). As shown in Table 6-7, for the current residential child population, the number of DUs exceeding P3 increases slightly from 389 to 394 DUs with the increase in the lead air concentration, though the number of DUs exceeding P5 and P8 remains the same. For the potential future residential child population, the number of ADAs exceeding P3 and P5 increases slightly from 139 to 140 (at P3) and from 68 to 70 (at P5); the number of ADAs exceeding P8 remains the same (Table 6-7). This analysis and the analyses conducted by ECY (2017a, 2017b) support the conclusion that metal concentrations in air remain safe for people to breathe.

EPA estimated the 3-year average annual concentration of PM10 at the U.S.-Canada border to be 12.2 µg/m^3. The annual PM2.5 NAAQS is 12.0 µg/m^3. If all measured PM10 mass is assumed to be PM2.5, then the annual PM2.5 NAAQS is just slightly exceeded at the border; however, this assumption represents a most-conservative scenario, and is highly unlikely as the total PM10 mass also includes particle sizes between PM2.5 and PM10. Thus, it is unlikely that the PM2.5 annual NAAQS is exceeded at the border. Given that Trail smelter emissions have not increased since the 2014-2017 period, air concentrations of PM2.5 due to the smelter at the border should not exceed the annual NAAQS.

EPA also estimated a 24-hour PM2.5 design concentration at the border, assuming all PM10 was PM2.5 during the 2014-2016 period. The design concentration (3-year average of 98th percentile of 24-hour average values) was 26 µg/m^3, which is well below the PM2.5 24-hour NAAQS of 35 µg/m^3 (EPA, 2016c).[96] EPA's current conservative estimates of PM10 impacts at the border give high confidence that concentrations of PM2.5 are at or below annual the NAAQS. These estimates, using monitoring from the 2014-2016 period, are likely valid given emissions from the smelter have not increased. The evidence does not support the need for continued air monitoring at the U.S.-Canada border for the purpose of the HHRA.

To evaluate whether the positioning of air monitors could affect air EPCs used in this HHRA, EPA requested and reviewed aerial photographs and supplemental location information provided by Teck for the Sheep Creek and Columbia Gardens Monitoring Stations during the periods in which data have been used in the HHRA (2002-2009 for Sheep Creek, used to calculate air EPCs, and 2012-2014 for Columbia Gardens, used in the above uncertainty analysis). For the

[96] https://www.epa.gov/lead-air-pollution/national-ambient-air-quality-standards-naaqs-lead-pb.

Columbia Gardens Monitoring Station, this included the monitor location that was relocated in 2011 due to vegetative growth. Based on this review, EPA concluded that it is strongly unlikely that vegetation inhibited monitor performance to the extent needed to impact results in the HHRA. Furthermore, the relative exposure contribution from air inhalation in the HHRA is well below screening levels. The ratio of screening levels to the estimated EPCs for the highest airborne contaminants of potential concern are shown in Table 6-8.

As shown in Table 6-8, the estimated air EPC for arsenic would need to be off by a factor of 2.6 in order to equate to an HQ of 1. Currently, it is a level where its risk and exposure are equivalent to background levels of arsenic in soil. For cadmium and lead the ratios are twice as high (5.2 and 6.4, respectively). The estimated EPCs for these COPCs could increase by a factor of 5 or 6, respectively, before reaching or exceeding the screening value (Table 6-8).

6.3.1.5 Indoor Air EPCs

In the absence of measured indoor air concentration data for COPCs, an assumption was made that the indoor air concentration was equal to the outdoor air concentration. This is a health protective assumption that is uncertain. There are studies showing that the home provides little, if any, filtration of airborne particles in ambient air (Thatcher and Layton, 1995) and other studies that suggest indoor air concentrations may be lower than outdoor air concentrations for small (PM2.5) particles (Allen et al., 2012).

6.3.1.6 Fish EPCs

For the fish tissue EPCs, sample size ranged from 9 to 51, depending on the analyte and species (Table 3-29). Fillet tissue data were collected for nine species consumed by human receptors (see Voigt et al. [2015] for information on distribution of metals in fish tissue). Site-specific data for the EPCs in fish tissue are considered robust and adequate to characterize risk from this pathway.

Species representative of various feeding guilds (piscivore, omnivore, insectivore, benthivore/ detritivore) were selected for analysis and were assumed to be representative of all fish species with similar feeding habits. Where a sufficient number of the target species could not be collected (e.g., Largescale Sucker, representative of benthivore/detritivores), a similar species was collected (e.g., Longnose Sucker). While feeding habits of these two species may differ and introduce uncertainty in the EPCs calculated for benthivore/detritivores, the increased sample size will reduce uncertainty in the EPC estimates. Based on the RecUse and CCT Tribal surveys (SRC, 2019a, 2019b), Rainbow Trout (an omnivore) and Walleye (a piscivore) were the most frequently consumed species of fish sourced from the UCR. Both surveys indicated that Burbot (piscivore), perch (omnivore), carp and catfish (benthivore/detritivore) were infrequently consumed. Creel surveys indicated similar trends, with Walleye and Rainbow Trout reported as the most frequently harvested species, and Northern Pike, whitefish, Burbot, perch, and carp harvested infrequently (STF, 2020). Although there is uncertainty associated with evaluation of individual species that are consumed infrequently, the direction and magnitude of that uncertainty is unknown. Species representative of the piscivore and omnivore feeding guild were reported as both frequently (Walleye, Rainbow Trout) and infrequently consumed (Burbot, perch).

6.3.1.6.1 Inorganic Arsenic in Fish

Inorganic arsenic was not detected in any fish fillet samples collected on-Site (n = 98, 2009 study [TAI, 2013a]; n = 9, 2016 White Sturgeon study [TAI, 2017d]; n = 12, 2018 Northern Pike study [TAI, 2018]). However, both inorganic and total arsenic were detected in whole body samples of some fish collected in 2005. Inorganic arsenic was detected in 3 of the 25 whole body fish submitted for analysis of both total and inorganic arsenic, and total arsenic was detected in all 25 whole body fish. When a mean inorganic arsenic concentration was calculated using the reporting limit as the concentration value for non-detects, the mean inorganic arsenic was 13.45 µg/kg ww, and the mean total arsenic in whole body fish was 246.4 µg/kg ww. This results in a Site-specific fraction of inorganic arsenic (compared to total arsenic) of 5.5% in whole body fish.[97] For this assessment, if a fish tissue sample was analyzed for inorganic arsenic, the result for that sample was used as the inorganic arsenic concentration in the EPC calculations. If a fish tissue sample was not analyzed for inorganic arsenic, then the Site-specific fraction of 5.5% was applied to the total arsenic concentration and that result was used as the inorganic arsenic concentration in the EPC calculations. Use of a Site-specific fraction based on measured concentrations reduces the uncertainty associated with making assumptions about the fraction of total arsenic present in fish tissue as toxic inorganic arsenic. Risks associated with inorganic arsenic in fish fillets are not likely overestimated.

6.3.1.6.2 Fish by Species

Risk calculations were done using EPCs calculated by fish species[98] with data collected from Reaches 1 through 6 combined. For the COPCs that contributed the most to risk from fish consumption (methylmercury, TEQ, Aroclors, lead and thallium), fillet concentrations were highest in sucker (mercury, Aroclors and lead), whitefish (TEQ), and Walleye (thallium) (Table 6-9). Exposure to these COPCs can be reduced by targeting consumption of less contaminated species.

Although risk calculations were not conducted using data by Reach, lead concentrations in fish tissue were evaluated further. Arithmetic mean lead concentrations ranged from 0.00355 mg/kg ww in Northern Pike to 0.163 mg/kg ww in sucker. Lead in sucker was highest in fish collected in Reach 1 (0.393 mg/kg ww) and Reach 2 (0.183 mg/kg ww) and ranged from 0.0642 to 0.0993 in sucker collected from Reaches 3 to 6. As noted above, exposure to lead can be reduced by limiting consumption of this species to fish caught in the lower river reaches.

6.3.1.7 Estimating Total (Summation) Concentrations of Certain Chemical Classes

There are 209 PCB congeners. Of the 209 congeners, twelve congeners are classified as “dioxin-like”. There are potential risks associated with possible enhancement of these dioxin-like PCB congeners (toxicologically related to TCDD), whereby congener-based analysis can be utilized to ensure that overall PCB risks are not underestimated (EPA, 1996a). Because dioxin and furan congeners and dioxin-like PCBs all act by the same mechanism as 2,3,7,8-TCDD, data for the dioxin and furan congeners and dioxin-like PCBs were converted to a TCDD TEQ by computing the sum across congeners of the product of congener-specific concentration and relative TEF.

[97] The average % inorganic arsenic was also calculated using measured total arsenic in fillets and the method detection limit as the value for inorganic arsenic, as per the Arsenic Data Usability Report (SRC, 2012); the calculated % arsenic was 5.6%, similar to the measured value for whole body fish.

[98] Data for Lake Whitefish and Mountain Whitefish were combined to calculate an EPC for whitefish, and data for Largescale Sucker and Longnose Sucker were combined to calculate an EPC for sucker.

TEFs were based on EPA (2010a). Separate TEQ values were calculated for dioxins and furans and dioxin-like PCBs assuming one-half the detection limit for concentrations qualified as non-detects. This assumption reduces the likelihood that TEQ EPCs were underestimated.

A total PCB concentration was calculated two ways, by summing across all non-dioxin-like PCB congeners and by summing across all Aroclors for each sample. Not all samples had Aroclor results. Non-detects were set equal to zero. The larger detected concentration of these two values was retained and used as the PCB EPC. This assumption reduces the likelihood that PCB EPCs were underestimated.

6.3.2 Uncertainty in Exposure Parameter Estimates

At this Site, numerous studies were conducted to collect Site-specific information to reduce the uncertainty in the risk estimates, primarily uncertainty associated with the following parameters: concentrations of chemicals in environmental media (abiotic and biotic), and human exposure factors. The use of Site-specific estimates of EPCs and exposure factors in risk calculations can help reduce the uncertainty in the risk estimates (EPA, 1989). Site-specific EPCs were obtained through numerous sampling events to quantify COPC concentrations in Site media (uncertainty described in the previous section), and exposure estimates were informed by conducting two high-effort, local surveys to develop Site-specific exposure parameters (SRC, 2019a and b).

6.3.2.1 Current and Future Resident Population Exposure Parameters

EPA has collected a wide variety of data and performed a number of studies to establish national default values for most residential exposure parameters. For the resident receptor population in this HHRA, the primary sources of exposure parameters were EPA (1989), EPA (1994b), EPA (1998a), EPA (2004a), EPA (2011a), EPA (2014a), and EPA (2019a). Uncertainty associated with non-default exposure parameters utilized in this risk assessment for residents is discussed below.

6.3.2.1.1 Uncertainty in Exposure Time for Current and Future Residents

The current and future resident populations were evaluated for potential risk from incidental ingestion of surface soil and indoor dust; for some COPCs, these risks exceeded non-cancer benchmarks. Important sources of uncertainty associated with estimating risk from incidental soil and indoor dust ingestion include the exposure assumptions utilized in the risk calculations and the COPCs that contribute to the calculated risk. For this risk assessment, exposure to surface soil assumed full time exposure of a resident on any DU or ADA for a lifetime, which may overestimate exposure for those DUs and ADAs that are not used daily. It was also assumed that exposure to indoor dust occurred on all residential DUs and ADAs that were sampled, even if no house was present on that DU.

Exposure to soil at this Site assumed full-time residential use of upland ADAs without residences. This simplifying assumption overestimates exposure for ADAs that are not used regularly by 12-17-month-old children who are most susceptible to lead exposure. This overestimate must be balanced with the possibility that future development may occur and the need to inform risk decisions for the near and long term. Because the Site is near a National Recreation Area, it is possible this area could experience pressure for land use change in the future, including residential expansion (both year-round and seasonal). Nevertheless, many of the

upland areas are presumably unavailable to residential growth because they are protected by government ownership/management. The exposure assumptions used here are provided to inform risk managers who may want to consider the possibility of expanded use of this area for residential development in developing risk reduction strategies for these areas.

6.3.2.1.2 Uncertainty in Exposure Time for Residents with a Beach

Residents with a beach on their property were assumed to spend 350 days/year on the property under RME exposure assumptions. They were also assumed to spend 32 days/year on the beach under RME exposure assumptions (2 days/week for 16 weeks/year, assumed to represent months with seasonable weather; professional judgement) and 20 days/year on the beach under CTE exposure assumptions based on beach utilization estimates from the RecUse Survey (SRC, 2019a). Although this totals more than 365 days/year, Site-specific exposure time of 2.9 hours/day for adults and 2.8 hours/day for children (SRC, 2019a) was incorporated into the surface water exposure calculations for the resident with beach population.

6.3.2.1.3 Uncertainty in Fish Consumption Estimates for Residents

The resident with a beach population and the current and potential future resident populations were evaluated for potential risk from exposure to COPCs through consumption of fish caught from the UCR; for some COPCs, these risks exceeded non-cancer benchmarks. Fish consumption rates were estimated based on the RecUse Survey (adult RME) and the CCT Tribal Survey (adult CTE) and are described in Section 6.3.2.3.3.

6.3.2.2 Outdoor Worker Population

The EPA has collected a wide variety of data and performed a number of studies to establish national default values for most outdoor worker exposure parameters. For this HHRA, the primary sources of exposure parameters for the outdoor worker population were EPA (1989, 1994b, 1998a, 2004a, 2011a, 2014a, 2019a). The only Site-specific exposure parameter used for this receptor population was an exposure frequency estimate based on interviews with park employees and worker activity during the drawdown season (DOI, 2019).

6.3.2.3 Recreational Visitor Population

Site-specific parameters derived from the RecUse Survey (SRC, 2019a) used in this HHRA were EF for trips to the beach, boat trips, and camping trips; ET for swimming during beach day trips, boat trips, and camping trips; and fish consumption rates for fish harvested from the UCR. Use of Site-specific exposure estimates instead of default exposure parameters will reduce the uncertainty associated with exposure estimates. It is assumed that evaluation of the recreational visitor receptor population is representative of local area residents and outdoor workers that utilize the UCR for recreation.

Some parameters derived from the RecUse Survey were also used to evaluate the resident with beach receptor population: EF and ET for swimming during beach trips, and the adult RME fish consumption rate for fish harvested from the UCR.

6.3.2.3.1 General Trips to the Beach

There is uncertainty associated with the recreational use exposure parameters derived from the RecUse Survey and used in the HHRA. Uncertainty in estimates of EF is quantified by CIs for

the means and tolerance intervals for the P95s that are presented in Tables 12–14 of the RecUse DAR (SRC, 2019a). Estimates of the mean EF for adults for all regions of the UCR combined are very precise, with MEs (1/2 the CI width) of ±1 day/year or less for all three trip types: beach, boating, and camping. MEs for the 95th percentile (P95) values for adults range from ±5.1 boating trips/year, to ±2.5 days spent camping per year and ±4.7 beach trips per year. While the sample sizes for children are much smaller than adult sample sizes, the MEs for the estimated means for children are ±2.2 days/year or less for all three trip types. The MEs for the estimates of the P95s for children are similar to adults except for boating; the ME for the estimate of the P95 for annual boating trips by children is 14.1, which corresponds to an RE of 88%. The estimates of the mean and P95 for number of trips per year, as well as their CIs, are considered very reliable given the large sample sizes available, particularly for adults.

The estimates for the means and P95s for EF for individual lake regions are less precise as would be expected given the smaller sample sizes. The decrease in precision varies depending on the type of trip and the lake region. The MEs for the estimated mean and P95 for beach trips by adults to the lower region are substantially larger than the MEs for the middle and upper lake region. For children, the estimates to the upper region are more precise than the lower and middle regions. For each of the trip types, estimates of the mean and P95 are less precise in the lower lake region compared to the middle and upper regions, except for the number of boating trips per year to the lower lake region by children: the MEs for the mean and P95 are 2.5 and 48 days/year, respectively.

6.3.2.3.2 Swimming, Exposure Frequency and Time

Estimated means and P95s for time spent swimming are presented in Tables 17a and 17b of the RecUse DAR (SRC, 2019a). Estimates of the mean time spent swimming during beach trips are very precise for adults and children for all regions of the UCR combined. The MEs for the means are less than ±0.2 hours for adults and children. The ME for the P95 for adults is also small (±0.25 hours). A tolerance interval for the P95 is not available for children (Woodruff's method was not able to produce an estimate; SAS, 2017). However, the estimates of the mean and P95s for time spent swimming during beach trips are very similar between adults and children. Therefore, the estimates of the mean and P95 for adults and children are also considered reliable.

The estimates for the time spent swimming during boating trips for children are not considered reliable given the small sample size (n = 16).

Estimates of the mean time spent swimming during camping trips are very precise for adults for all regions of the UCR combined. The ME for the mean is approximately ±0.25 hours. The estimate for the P95 is 6.0 hours with 95% tolerance limits of [4.7, 5.7]. The sample size for children (n = 24) produces less precise estimate of the mean; the ME is 0.70 hours.

6.3.2.3.3 Uncertainty in Fish Consumption Estimates for Recreational Visitors

The recreational visitor population was evaluated for potential risk from exposure to COPCs through consumption of fish caught from the UCR; for some COPCs, these risks exceeded non-cancer benchmarks. Important sources of uncertainty associated with estimating risk from fish consumption include the fish consumption rate utilized in the risk calculations and the COPCs that contribute to the calculated risk. The fish consumption data for the RecUse Survey came

from two sources: 12-month recall questionnaires and detailed 3-month food diaries. The fish consumption data for the RecUse Survey were combined to increase limited data provided by the fish consumption diaries for children (n=7) and for women of child-bearing age (n = 4). Combining data from the questionnaires and diaries entailed merging data that covered different time periods (3-month diaries versus 12-month recall questionnaires). A comparison of the number of meals reported on the questionnaires and diaries did not indicate a clear relationship. Therefore, there is a degree of uncertainty (of unknown magnitude and direction) in the fish consumption rates for the recreational visitor population.

As described in Section 3.5.2.1, the lack of sufficient data to estimate a fish consumption rate for children was addressed by using a ratio of 0.5 for the child:adult DCR for fish that was based on caloric intake (IOM, 2005; Appendix 14). There is some uncertainty in applying this ratio to estimate the child DCR for fish as the ratio is not specific to fish or any other specific food item. Other sources of DCRs for fish were analyzed to help interpret the ratio of 0.5 that was estimated with the IOM data. Based on EPA's analysis of survey data collected for tribal and recreational anglers in four states, the ratio of the mean child:adult DCRs for fish and shellfish combined was 0.4 for both the tribal and angler populations (EPA, 2013b). The ratios ranged from 0.4 to 0.7 for the tribal populations and from 0.3 to 0.6 for the angler populations.

The fish consumption rates derived from the RecUse Survey reflect consumption at the time the survey was conducted. The effect of fish advisories issued by WDOH on fish consumption rates is unknown (WDOH, 2018a, 2018b, 2019). These data are based on a high effort, local survey conducted to inform a Site-specific fish consumption rate for recreational visitors; the use of Site-specific consumption rates reduces the uncertainty in assessing exposure to contaminants from eating locally-sourced fish.

6.3.3 Uncertainty Associated with Non-Lead COPCs that Contributed the Most to Risk

6.3.3.1 Uncertainty Associated with Key COPCs for Incidental Soil and Dust Ingestion

For the incidental soil and indoor dust ingestion pathway by current residents, the non-lead COPCs that contributed the most to risk in soil are arsenic, cobalt, iron, manganese, and thallium. For the current resident adult population, no COPCs had individual HQs >1. Thallium had an HQ of 2 for one house DU for the current resident child population under the RME scenario. For potential future residents, the COPCs that contributed the most to risk in soil are also arsenic, cobalt, iron, manganese, and thallium. However, no individual COPCs had an HQ >1. Measured concentrations of cobalt, iron, manganese, and thallium are below estimated background concentrations in the majority of residential DUs and ADAs (see Section 6.4, Figures 6-4 to 6-7, and Figures 6-16 to 6-19).

6.3.3.2 Essential Elements Identified as Key COPCs for Soil/Dust Ingestion

Some essential elements were found to contribute to the risk at the UCR Site. Iron and manganese are essential nutrients, and cobalt is an essential trace element which is a constituent of vitamin B12. Table 6-10 provides the estimated ADI, the RfD, the adequate intake (AI) concentration, and the Tolerable Upper Intake Level (TUIL) for cobalt, iron, and manganese (Goyer et al., 2004; EPA, 2007d; National Academies Press [NAP], 2001; IOM, 2018; University of Rochester Medical Center, 2018; National Institutes of Health [NIH], 2018).

Nationwide, the average adult intake of cobalt is 5-8 µg/day, primarily from the diet and water ingestion[99]. No RDA for cobalt has been established (IOM, 2018; University of Rochester Medical Center, 2018). The RDA for B12 is 2.4 µg/day, and no TUIL has been determined. Site-specific ADI for current residents is 1 µg/day, and 2 µg/day for future residents. Although there is no RDA or TUIL for this essential trace element, intake of cobalt via incidental soil ingestion at this Site is below the range reported as the ADI for adults.

The RDA for iron is 18 mg/day for premenopausal females, and 8 mg/day for all other adults NAP, 2001; IOM, 2018). The RfD for iron is 56 mg/day, slightly higher than the TUIL (45 mg/day). Site-specific ADI of iron for current residents is 3.3 mg/day (range 1.6 to 9.1 mg/day), and 3.6 mg/day (range 2.0 to 5.4 mg/day) for future residents, more than 10-fold below both the RfD and the TUIL.

An RDA is not available for manganese. Table 6-10 provides the estimated AI, the RfD (1.92 mg/day), and the TUIL for manganese, and the Site-specific estimated daily intake average and range for children and adults. Average Site-specific daily manganese intake by current residents is more than 20-fold below the RfD (0.09 mg/day, range 0.04 to 1.3 mg/day). ADI by future residents was more than 10-fold below the RfD (0.16 and ranged from 0.05 to 0.33 mg/day).

6.3.3.3 Uncertainty Associated with Key COPCs for Fish Consumption

For the fish consumption pathway by recreational visitors, residents with a beach, and current and potential future residents, the COPCs that contribute the most to risk in fish tissue are methylmercury, dioxin/furans and dioxin-like PCBs, and thallium. State and nationwide monitoring programs indicate that bioaccumulative COPCs in fish tissue are an ongoing problem in Washington and many parts of the U.S. There is a WA State-wide fish consumption advisory for mercury in Smallmouth Bass, Largemouth Bass, and Northern Pikeminnow (Herger et al., 2017). UCR fish advisories are similar to (Largemouth Bass, two meals per month; Northern Pikeminnow, do not eat; WDOH, 2020) or less restrictive than Statewide advisories (Smallmouth Bass, one meal per month, WDOH, 2012).[100] Nationally, the number of fish advisories for mercury and PCBs continues to increase (EPA, 2011b).

For individual fish species, only consumption of Northern Pike and White Sturgeon result in no individual COPC HQs >1. Some fish consumption target organ HIs exceed 1 for the RME child recreational visitor and the RME child resident (with and without beach) populations (Tables 5-9 to 5-111 and 5-136 through 5-200). Consumption of Northern Pike and White Sturgeon results in no target organ HIs >1 for both the child recreational visitor and resident (with and without beach). Sucker, Smallmouth Bass, and Walleye consumption result in target organ HIs >1 for three target organ systems (developmental, nervous, and skin/hair/nails for Smallmouth Bass and Walleye and developmental, nervous, and reproductive for sucker). Consumption of Burbot results in target organ HIs >1 for the developmental and nervous system, and consumption of whitefish results in target organ HIs >1 for the reproductive and skin/hair/nails target organ

[99] https://www.cobaltinstitute.org/cobalt-bioessentiality.html; https://www.urmc.rochester.edu/encyclopedia/content.aspx?contenttypeid=19&contentid=cobalt.
[100] https://www.doh.wa.gov/CommunityandEnvironment/Food/Fish/Advisories/Publications.

systems. Consumption of kokanee or Rainbow Trout only results in a target organ HI >1 for the skin/hair/nails system. Kokanee, bass, Rainbow Trout, and Walleye were fish species reported being consumed most frequently (SRC, 2019a). Anglers typically prefer to harvest and consume trout and Walleye rather than sucker (SRC, 2019a).

6.3.3.3.1 Organic COPCs

Ongoing State contaminant monitoring programs collect data on persistent, bioaccumulative, and toxic chemicals (PBTs) in fish tissue. ECY has a Freshwater Fish Contaminant Monitoring Program (FFCMP). The FFCMP has measured PBTs in over 550 samples from 170 sites within the State of Washington (Seiders et al., 2016). COPCs measured in some or all fish collected are chlorinated pesticides, mercury, PCBs, PCDD, and PCDFs, and PBDEs. In 2010-2011, ECY collected sediment and fish from 17 northeast Washington lakes and rivers and one lake and river in northern Idaho thought to be minimally impacted by local human activities. The goal of the study was to provide regional-scale sediment and fish tissue data representative of reference or background conditions (ECY, 2011).

Measured concentrations of dioxin/furans, and PCBs in fish collected from the UCR are similar to concentrations measured in other locations in the northeastern portion of Washington and in Idaho (Seiders et al., 2014, 2015, 2016; Herger et al., 2017; ECY, 2011). Detailed comparisons are difficult because more fish concentration data are available from the UCR than from other locations in the region, and similar species were not collected for all studies.

6.3.3.3.2 Inorganic COPCs

Mercury is a Site-related COPC that was historically discharged in wastewater effluent from fertilizer plant operations at the Cominco facility (Cominco, 1997). Mercury is also recognized as a ubiquitous global contaminant in fish. Measured concentrations of mercury in fish collected from the UCR are similar to or lower that concentrations measured in other locations in northeast Washington and Idaho (Seiders et al., 2014, 2015, 2016; Herger et al., 2017; ECY, 2011). The mean mercury in fish fillets (all species; ranging from 63.4 [kokanee] to 256 µg/kg ww [sucker]) collected from the UCR is 158 µg/kg ww, similar to the regional-scale fish tissue data representative of reference or background conditions reported in ECY (2011; mean of 69 µg/kg ww for salmonids and 169 µg/kg ww for spiny ray fish). Fish advisories for mercury in the UCR are similar to other advisories in Eastern Washington prepared by the WDOH.[101]

Information on thallium concentrations in fish is limited. The only study located that reported thallium concentrations in fish tissue was Tiller et al. (2004). Mean thallium concentrations in whole-body juvenile salmon captured from the Columbia River upstream and downstream of the Hanford site were 28.9 and 29.5 µg/kg ww, respectively. Mean thallium concentrations measured in fish fillets from the UCR ranged from 3.32 µg/kg ww in White Sturgeon to 35.5 µg/kg ww in Walleye (Table 3-29).

Although the COPCs that contributed the most to non-cancer risks calculated for fish consumption by the recreator and resident with and without beach populations at the UCR Site

[101] https://www.doh.wa.gov/DataandStatisticalReports/HealthDataVisualization/fishadvisory.

are methylmercury and thallium, the concentrations of these COPCs in UCR fish are within the range of concentrations measured at other non-contaminated and contaminated sites.

As thallium is a COPC that contributes a large portion of the risk for consumption of fish collected from the UCR, thallium concentrations in sediment, surface water, and tissue were evaluated on a reach-by-reach basis (Table 6-11). For sediment, detection frequency is highest in Reach 1, and generally decreases on an up- to downstream basis. For surface water, the opposite pattern is observed; the highest detection frequency was in Reach 6 and the lowest in Reach 1. The highest measured thallium concentrations in each medium were observed in the following Reaches: sediment, Reaches 2 to 4; surface water, Reaches 4 and 5; and fish, Reach 6.

6.3.4 Uncertainty Associated with Toxicity Values

The toxicity value for thallium of 1×10^{-5} mg/kg-day is described in an appendix to a PPRTV assessment and is considered a screening toxicity value (EPA, 2012c). The available toxicity database for thallium was considered insufficient for development of a provisional RfD (p-RfD). The screening p-RfD designation reflects considerable uncertainty in the available data used to derive the screening p-RfD including critical limitations with the principal study. This is represented in the screening p-RfD as a 10-fold downward adjustment of the dose-response point of departure (in this case, a NOAEL): "a database uncertainty factor (UF_D) of 10 is applied to account for a lack of adequate developmental toxicity studies and a two-generation reproductive study, and additional uncertainty associated with the limited data available on neurotoxicity" (EPA, 2012c). The total UF represented in the screening p-RfD is a 3000-fold downward adjustment of the NOAEL (UF_D * UF_H * UF_A *UF_S). UF_H, UF_A, and UF_S are described as: "a UF_H of 10 applied to account for variation in human susceptibility in the absence of information on the variability of response to thallium in the human population; a UF_A of 10 applied for extrapolation from laboratory animals to humans since no information is available to characterize the toxicokinetic or toxicodynamic differences between experimental animals and humans; and a UF_S of 3 applied to account for extrapolation from subchronic to chronic exposure duration." Thus, risk estimates derived using the available screening-level p-RfD for thallium should be interpreted with caution. The 3000-fold UF in the screening p-RfD makes it likely that risks for exposure to thallium may be overestimated. EPA Region 10 will monitor EPA ORD/IRIS evaluations on thallium for significant changes.[102]

EPA has not derived a chronic inhalation RfC for arsenic (EPA, 2019d). ATSDR reviewed data on inorganic arsenic in 2007 and 2016 and declined to derive inhalation MRLs based on their conclusion that inhalation studies were inadequate for deriving MRLs (ATSDR 2007, 2016). Associations between exposures to air-borne inorganic arsenic and neurological effects have been reported in epidemiological studies of occupational exposures (ATSDR, 2007). However, exposure levels were highly uncertain and exposures to other neurotoxic agents (e.g., lead) confounded conclusions regarding the role of arsenic in the observed outcomes. ATSDR reported a LOAEL of 0.31 mg/m^3 for decrements in nerve conduction velocity in adult workers, who were also exposed to lead.

[102] For more information, see Hubbard et al. (2019); https://www.toxicology.org/pubs/docs/Tox/2019Tox.pdf; https://www.rti.org/publication/dose-range-studies-thallium-i-sulfate-subchronic-toxicity-perinatally-exposed-hsd.

California EPA (Office of Environmental Health and Hazard Assessment [OEHHA], 2008) derived a chronic inhalation reference exposure level (REL) for inorganic arsenic of 0.015 µg/m^2 by extrapolating a dose-response relationship derived from study of exposures of children to arsenic in drinking water. The REL was based on results of an epidemiological study that found evidence for decreased intellectual function in 10-year-old children (Wasserman et al., 2004). A regression model relating decrement in Full Scale IQ predicted a 0.44 IQ point decrement per µg arsenic/liter (L) drinking water exposure, from which OEHHA calculated that a 1 point decrement in IQ would be associated with a 2.27 µg/L increase in drinking water arsenic concentration. OEHHA assigned 2.27 µg/L as a LOAEL and a calculated the corresponding LOAEL arsenic intake of 2.3 µg arsenic/day, assuming a daily intake of 1 L water/day (2.3 µg/day = 2.3 µg/L × 1 L/day). The LOAEL ingestion intake was converted to a corresponding LOAEL air concentration of 0.46 µg arsenic/m^3 assuming a breathing volume rate of 9.9 m^3/day and that 50% of the inhaled arsenic would be absorbed (0.46 µg/m^3 = 2.3 µg/day/9.9 m^3/day). The LOAEL air arsenic concertation was adjusted by a 300-fold UF to derive the REL of 0.015 µg/m^3 (0.015 µg/m^3 = 0.46 µg/m^3/300). EPA continues to work with ORD/IRIS to monitor the arsenic research that may be used to revise the toxicity value in the future.

6.3.5 Summary of Uncertainty in Non-Lead COPC Risk Estimates

For COPCs other than lead, the risks for surface water and sediment exposures, as well as from dermal contact with surface soil, were well below risk benchmarks. For non-lead COPCs in surface water and sediment, uncertainties are not likely to be of a magnitude or direction that would encompass risk benchmarks. For all receptor populations, risk from inhalation of air was also below risk benchmarks. For non-lead COPCs in these media, uncertainties are not likely to be of a magnitude or direction that would encompass risk benchmarks.

The primary source of risk from exposure to COPCs other than lead derives from incidental ingestion of surface soil and indoor dust by both current and future child residents, and from consumption of fish by the recreational visitor and resident with and without beach populations. In most cases, when target organ HIs >1, those HIs are generally in the range of 2 to 3. For these exposure scenarios, while non-lead risk benchmarks are exceeded for non-cancer endpoints, the magnitude of the exceedance is of less concern than higher HIs. HIs are calculated by dividing estimated daily intakes by RfDs. RfDs are derived from no effect levels divided by UFs to account for uncertainty in the measured NOAEL. There is a "margin of safety" built into the RfD values (Section 4.3). Exposures higher that the RfD or RfC may carry some risk, but an exposure above the RfD or RfC does not mean that an effect will necessarily occur. In addition, as discussed in Section 6.3.3.3, the concentrations of COPCs in fish in the UCR are either similar to or lower than concentrations in other freshwater fish in eastern Washington, and UCR fish advisories are similar to (e.g., Largemouth Bass, two meals/month; Northern Pikeminnow, do not eat; WDOH, 2020) or less restrictive than (e.g., Smallmouth Bass, one meal/month; WDOH, 2012) Statewide advisories.[103]

6.4 Consideration of Background

Many chemicals of concern commonly found at Superfund sites are also found in non-impacted areas. Characterizing such "background" information is important to risk managers because the

[103] https://www.doh.wa.gov/CommunityandEnvironment/Food/Fish/Advisories/Publications.

CERCLA program does not clean up to concentrations below natural or anthropogenic background levels because doing so would likely result in recontamination and a return to background levels (EPA, 2002g). The contribution of background concentrations to risks associated with CERCLA releases may be useful to inform Site-specific risk management decisions for COPCs that exceed risk benchmarks. Recognizing that many of the COPCs in this HHRA may be influenced by natural (geogenic) or man-made (anthropogenic) sources, an assessment of Site background concentration of COPCs in soil was undertaken as described below and in Appendix 4.

For this HHRA, stream sediment and soil samples collected within the UCR drainage basin as part of the National Uranium Resource Evaluation (NURE) program (Smith, 2006; Smith et al., 2013) were used as the basis for determining a Site-specific soil background estimate for COPCs (see Table 6-12). This background assessment was conducted using DQOs for human health receptors (i.e., using soil data sieved to the <149 µm particle size; EPA [2016a]; Attachment 1 of SRC [2014a]). Data used in the HHRA evaluation included the NURE data as well as data from Grossman et al. (2008), Church et al. (2008), and the upland soil data collected for the UCR risk assessments (see Section 2.6.2.5 for a description of the upland soil data). The background analysis for the HHRA relies on soil data sieved to <149 µm (EPA, 2016a). All of the data used to estimate the background concentrations were derived from stream sediment and soil samples collected by the NURE program in the Colville River, Kettle River, Lower Spokane, Sanpoil River, and UCR sub-basins of the Columbia River drainage basin (Figure 6-1).[104] The majority of the COPC results in the background dataset were determined by the Savannah River Laboratory (SRL); however, when available, the original SRL data were replaced with data provided by reanalysis of archived NURE sample material by Grossman et al. (2008) and Church et al. (2008). The NURE data included some errors in the arsenic data for sample locations in the Kettle River, Sanpoil River, and UCR sub-basins (Church, 2010); these data were replaced with data provided by Smith (2007).

Four records were removed from the background dataset because the NURE database indicated they were potentially affected by mining activities or may have contained tailings; or were located within the 100-year floodplain of the UCR.[105] Seven sample locations, all located south of the community of Marble, are within the area that had exhibited effects of SO_2 emissions on vegetation (Scheffer and Hedgcock, 1955). These data were retained in the background analysis dataset because they had no effect on estimates of the 95% upper tolerance limit for the 95th percentile of the population (UTL95-95 or 95UTL95). The only results available for cadmium and thallium are provided by the Church et al. (2008) reanalysis of the NURE archived samples from the Lower Spokane sub-basin. The background dataset does not include any results for antimony.

[104] The five sub-basins correspond to hydrologic unit code (HUC) 8-digit hydrologic units, while the Columbia River Drainage Basin is a HUC-4 or subregion level unit. The five sub-basins correspond to the Washington State Water Resource Inventory Areas (WRIAs) hydrologic units.

[105] One record (rec_no = 5184673) was removed from the background dataset because the sample was located within the 100-year floodplain of the UCR. Two records (rec_no = 5185476 and 5185477) were removed because the NURE data indicated they were potentially affected by mining activities (field [contamc] contained the word mining). Another record (rec_no = 5184696) was removed because the NURE database indicated the sampled material may have contained mine tailings [field [samptyp] = 99). Database fields are defined in Smith (2006).

Potential outliers identified using the *MM* method (Yohai, 1987) had no discernable effect on the background estimates, likely due to the large sample sizes that were available for each COPC (Table 6-12). The data were also checked for spatial outliers using the local Moran I statistic (Anselin, 1995). A group of 19 sample locations with high chromium concentrations was identified in the Kettle River sub-basin (Figure 6-1). The mean and SD of the chromium concentration for the 19 locations were 467 and 441 ppm, respectively while the mean and SD for the rest of the chromium data were 47 and 46 ppm, respectively. The 19 sample locations were removed from the dataset prior to estimating the background concentration for chromium. The final background dataset consisted of 552 sample locations (Figure 6-1).

An estimate of the UTL95-95 was used to define the background concentration for each COPC. The UTL95-95 was estimated using ProUCL version 5.1 (EPA, 2015d). Goodness-of-fit tests indicated the normal, gamma and lognormal distributions did not provide a good fit for the COPCs. The UTL95-95s were estimated using a nonparametric method based on the binomial distribution (EPA, 2015d, Equation 3-10). Non-parametric estimates of the 95% of the population distribution are also presented in Table 6-12. The 95% were estimated using the ranks (*order statistics*) of the sample data, with interpolation when needed (EPA, 2015d, Equation 3-1). Antimony data were not available from the NURE database; Table 6-12 includes a background estimate reported by ECY (2019) for antimony of 2.15 mg/kg (this value is a UTL95-90).[106]

Table 6-12 shows the background estimates for 12 metal COPCs in soil compared to both Residential Screening Levels[107] and to the soil EPCs used in this HHRA. Residential Screening Levels for this evaluation were calculated following the approach described in Section 3.2.2 for the COI screen with non-lead COPC risk benchmarks of HQ >1 and excess cancer risk $>10^{-4}$ entered into the RSL Calculator (EPA, 2019a). These Residential Screening Levels are based on full-time residential exposure to soil. The same toxicity values used in the COI screen were used for this calculation except for chromium. Toxicity values for Cr(VI) were used in the COI screen, but toxicity values for trivalent chromium were used in these RSL calculations, as the medium of interest is soil and the expected form of chromium in soil is likely trivalent (ATSDR, 2012). The trivalent chromium toxicity value is for insoluble salts. Both the assumption that trivalent chromium is the valence present in soil and the toxicity value are uncertainties. The RSL for inorganic arsenic is the noncarcinogenic screening level for a resident child. The background value for lead (35 ppm) does not include an adjustment for RBA.

Table 6-12 compares the recalculated Residential Screening Levels with the background estimates for soil COPCs. None of the estimated background concentrations for soil COPCs exceeded the recalculated Residential Screening Levels. The estimated background concentration of all soil COPCs except antimony, cadmium and nickel exceed the RBSLs calculated for the COI screen. In other words, all soil COPCs that are non-lead COPCs that contributed the most to risk would have been carried through the risk assessment if estimated soil background concentrations were available during the COI screen.

[106] The NURE dataset represents approximately 95% of the individual samples evaluated to estimate background in ECY (2019). This provides support for use of this dataset to derive the background estimates used in this HHRA.
[107] Residential Screening Levels are derived from EPA's tool (EPA, 2019a). It should be noted that this screening tool does not consider additivity of non-cancer effects for COPCs, nor does it sum exposure across pathways. For lead, the PRGs for P3, P5, and P8 are shown rather than the Residential Screening Level.

Measured concentrations of cadmium and lead exceeded estimated background concentrations in 564 and 503 of 588 residential soil DUs, and 138 and 141 of 142 ADAs, respectively (Table 6-12). Measured concentrations of arsenic and antimony also exceeded estimated background concentrations in many DUs and ADAs. The COPCs that contributed the most to calculated risk from exposure to soil in addition to arsenic and lead were thallium, manganese, cobalt, and iron. While arsenic and lead concentrations were above background levels, concentrations of thallium, manganese, cobalt, and iron were below estimated background concentrations. At Superfund sites, background concentrations are calculated as the UCL on the 95th percentile. It is expected that the majority of site-specific data will fall below background, provided that the sampling area reflects background conditions for the target metals (Stifelman, 2020).

Figures 6-2 through 6-19 compare Site background concentration of lead in soil along with the P3, P5, and P8 PRGs for binned soil lead concentration by DU type or ADA, as well as other COPCs compared to their background concentrations. These figures illustrate the potential for background concentrations of COPCs in soil to influence risk results. For example, consideration of background soil lead concentration at the Site is unlikely to influence results based on using the P5 or P8 risk benchmark. Because the Site background soil lead concentration (35 ppm) is close to the P3 soil lead PRG (50 ppm) consideration of background may be important for risk management for that PbB target.

7 SUMMARY AND CONCLUSIONS

Exposure to chemicals of potential concern (COPCs) was evaluated for the following receptor populations:

1. Residents who contact soil in their yards and dust in their homes, who breathe air, who may eat fish from the Upper Columbia River (UCR), and who may contact UCR surface water and sediment if they have a beach on their property;
2. Outdoor workers who contact surface and subsurface sediment or beach soil at public beaches, who contact UCR surface water, and who breathe air; and
3. Recreational visitors who use the UCR for beach day trips, boating, camping, swimming and fishing and thereby contact surface sediment and beach soil at public beaches and relict floodplains, who contact UCR surface water, who breathe air, and who may eat fish harvested from the UCR.

The evaluations for the Colville High Intensity Resource User (CHIRU) and Spokane Tribe of Indians (STI) populations are found in Appendices 1 and 2, respectively.

Receptor populations are hypothetical because they are restricted to exposures to selected media and defined media intakes; therefore, they may not represent any single individual or real population, whose members engage in a broader diversity of activities and intensities (duration, frequency) than assumed for the receptor population. For example, the outdoor worker receptor is assumed to be exposed exclusively during occupational activities. However, workers may live in the area and may recreate at the UCR. The recreational visitor receptor is assumed to be exposed exclusively during intermittent visits to the Site. However, some recreators may also be residents or outdoor workers. Risks of a combined residential, occupational, and/or recreational exposure were not evaluated in this human health risk assessment (HHRA). Although a worker-resident-recreator receptor population has not been directly assessed in this HHRA, the risks for contributing exposures (work, or residence, or recreation) provide some information about risks of combined exposures. For example, if risks from residential exposure exceed risk benchmarks, then risks for the resident who works at the Site are also likely to be of concern, but not necessarily from exposures at work. If a hypothetical resident has estimated risk above benchmarks, then the risk for a hypothetical resident who works at the Site may be greater than for a resident who does not work at the Site. Risks for individual receptors should not be summed to estimate risks for “hybrid” receptors (e.g., resident-recreator-worker) because exposures from each exposure scenario do not necessarily sum (e.g., a person at work is not at home or recreating).

Risks from exposure to lead were assessed using the Integrated Exposure Uptake Biokinetic (IEUBK) Model for Lead in Children for residential and recreational exposures, the Adult Lead Methodology (ALM) for outdoor worker exposures, and the All Ages Lead Model (AALM). Risks for exposures to other COPCs were estimated using exposure pathways and parameters based on Environmental Protection Agency (EPA) guidance and Site-specific information obtained from the RecUse and CCT Tribal Surveys. Some exposure factors for the CHIRU and STI populations evaluated in Appendices 1 and 2 differed from those used in the body of this HHRA.

For this HHRA, the risk evaluation benchmarks selected for elevated lead risk were defined as greater than (>) 5% probability of exceeding a blood lead concentration (PbB) of 3, 5, or 8 µg/dL (referred to as "P3," "P5," and "P8," respectively). The IEUBK model cannot be used with a risk benchmark below P3 because risk goal would be exceeded even if the soil lead concentration is 0 ppm due to dietary lead exposure. P3 was selected as a lead risk benchmark to quantitatively evaluate the low end of the risk range of child PbBs associated with adverse health effects and P8 was selected as a less protective benchmark. The estimated background soil lead concentration for this Site is approximately 35 ppm (Appendix 4).

The IEUBK model was used to derive Preliminary Remediation Goals (PRGs) for residential soil lead exposures for each of the three risk benchmarks as follows:

- Utilizing the IEUBK model, a 5% probability of exceeding a PbB of 3 µg/dL was associated with a soil concentration of approximately 50 ppm lead
- Utilizing the IEUBK model, a 5% probability of exceeding a PbB of 5 µg/dL was associated with a soil concentration of approximately 200 ppm lead
- Utilizing the IEUBK model, a 5% probability of exceeding a PbB of 8 µg/dL was associated with a soil concentration of approximately 400 ppm lead

The PRGs associated with each of these risk benchmarks is based on an assumed default relative bioavailability (RBA) of 60% for lead. Site-specific *in vitro* bioavailability (IVBA) information was used to derive RBA-adjusted lead concentrations for samples collected on-Site, which allows direct comparison with these PRGs.

Because the IEUBK model requires a complete exposure scenario, the user must input a residential soil concentration even when the exposure pathway of interest (i.e., exposure to beach sediment) is not residential. Exposure of children to lead in sediment at residential beaches, public beaches, and relict floodplains (beach soil) was therefore assessed using the time-weighted approach recommended by EPA (2003a). This approach used Site-specific exposure frequency information to apportion exposure between the beach sediment/soil or relict floodplain soil and the soil at the "residence." The residential soil exposure point concentration (EPC) used for lead in this approach was either the average of the lead concentration in the house decision unit (DU) (or the nearest appropriate DU or DUs) on that property (for residential beaches), or the average residential soil EPC for the study area (129 mg/kg; for residential beaches with no associated "house" DU, for public beaches, and for relict floodplains). Surface water exposure (i.e., incidental ingestion of surface water while swimming) and exposure from consuming fish harvested from the UCR were assessed as additional exposures to lead that would occur while recreating at a public or private beach.

For media other than soil, sediment, and surface water, lead risk may be considered in terms of how exposure to the medium (i.e., fish consumption) contributes to lead intake given a selected concentration of lead in residential soil. The IEUBK model predicts that lead intakes of at least 1 µg Pb/day[108] are needed to decrease the soil PRG by 10%. For example, the P5 soil PRG would decrease from approximately 200 ppm to approximately 180 ppm when lead intake from

[108] Assuming 30% absolute bioavailability (ABA).

fish consumption increases from 0 to 1 µg Pb/day. In this HHRA, risk from exposures that were predicted to contribute <1 µg lead intake per day (i.e., fish consumption), which would change the PRG by <10%, were classified as minimal.

Risks from exposure to non-lead COPCs were estimated using exposure pathways and parameters based on EPA guidance and Site-specific information. Risks were estimated two ways for each receptor: using high-end exposure parameters (termed the "Reasonable Maximum Exposure" [RME]) and using mean or average values for exposure parameters (termed the "Central Tendency Exposure" [CTE]). Risk benchmarks used in this HHRA for non-lead COPCs were as follows: a non-cancer hazard quotient (HQ) >1 or an excess cancer risk $>10^{-4}$ for individual COPCs (EPA, 1997). HQs for individual COPCs were also summed across COPCs within an exposure pathway, and across exposure pathways for a specific exposure scenario, to calculate a hazard index (HI). The risk benchmark for non-cancer hazard based on the HI was HI >1. As recommended in Exhibits 8-2 and 8-3 of EPA's *Risk Assessment Guidance for Superfund Volume I Human Health Evaluation Manual, Part A* (EPA, 1989), final risk results were presented as one significant digit and compared to benchmarks. Intermediate calculations retained additional digits to minimize rounding errors. The following are examples of how calculated results were reported as one significant digit and then compared to the non-cancer benchmark of 1:

- Calculated result of 0.92 was reported as 0.9 – it does not exceed benchmark
- Calculated result of 0.96 was reported as 1 – it does not exceed benchmark
- Calculated result of 1.3 was reported as 1 – it does not exceed benchmark
- Calculated result of 1.5 was reported as 2 – it exceeds benchmark

The assumption of dose additivity in the HI approach is most relevant to chemicals that induce the same effect by the same mode of action. If an HI >1 because of summing several HQs across pathways, then the chemicals can be segregated by effect and mode of action, and an HI can be calculated for each target organ group (EPA, 1989). Target organ HIs were calculated in this HHRA for receptors and pathways where the HI >1 if summed across COPCs within an exposure pathway, or across exposure pathways for a specific exposure scenario. The risk benchmark for non-cancer hazard based on target organ HIs was HI >1. Cancer risks were summed across a lifetime to calculate a time-weighted average (TWA) cancer risk. The risk benchmark for TWA excess cancer risk was $>10^{-4}$.

Risks for the resident, recreational visitor, and outdoor worker populations are summarized below.[109]

Current Resident Population (Not Beach DUs)

The current resident population was evaluated for exposure to outdoor soil and indoor dust, outdoor and indoor air, and consumption of fish caught from the UCR (evaluated for each species individually). This was done on a DU-by-DU basis (i.e., conservatively assuming that the

[109] Risks from exposure to lead and non-lead COPCs by the CHIRU and STI populations may be found in Appendices 1 and 2, respectively.

resident lived full-time on that DU). Table 7-1 summarizes the number of DUs that exceeded risk benchmarks for both non-lead and lead COPCs for the current resident population without a beach on the property. As noted previously, while Appendix 1 evaluates the high-intensity resource users within the CCT population, the non-subsistence CCT population is represented by the residential population evaluated in this HHRA.

Lead

Of 588 residential DUs evaluated, 389 DUs exceeded the lead benchmark of P3 (66%), 87 DUs exceeded P5 (15%), and 12 exceeded P8 (2%), not including the consumption of fish from the UCR (see Figures 5-1 through 5-3). Consuming fish from the UCR may pose an additional lead risk to current residents (see Appendix 5 for batch mode lead results). As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

COPCs Other Than Lead

For the non-lead evaluation, exposures from each pathway were summed to evaluate both cancer and non-cancer effects to the current residential population (without a beach) as a whole. When cancer risk was summed across a lifetime and across exposure pathways for the current residential population (without beaches on their property), no DUs exceeded the cancer risk benchmark of 10^{-4} for either the RME or CTE scenario.

For the non-cancer evaluation for the current adult resident population, no exposure pathway on its own resulted in exceedances of target organ risk benchmarks under either the RME or CTE scenario. When these pathways were summed to look at total exposure to current adult residents without beaches on their property, one house DU had a target organ HI >1 for the skin/hair/nails system when the fish species consumed was Walleye (this DU also exceeded the lead risk benchmark of P8), and all 588 DUs had non-cancer target organ HIs >1 for the nervous system when the fish consumed was sucker (see Figure 5-11). These exceedances occurred under the RME exposure scenario; consumption of all other fish species evaluated did not result in non-cancer benchmark exceedances. When CTE exposure conditions were assumed, no DUs exceeded non-cancer benchmarks for current adult residents (without beaches).

For the non-cancer evaluation for the current child resident population, inhalation of outdoor and indoor air and consumption of Northern Pike or White Sturgeon, as individual exposure pathways, did not result in exceedance of non-cancer benchmarks at any residential DUs under either RME or CTE scenarios. For the exposure to soil/dust pathway, one house DU had a target organ HI >1 for the skin/hair/nails system with RME exposure assumptions but did not exceed benchmarks with CTE exposure assumptions. This house DU exceeds the lead risk benchmark of P8 as well, as shown in Figure ES-1.

Under the RME scenario for the fish consumption pathway for the current child resident population, consumption of Burbot resulted in target organ HIs >1 for the developmental and nervous systems; consumption of kokanee or Rainbow Trout resulted in the skin/hair/nails target organ system HI >1; consumption of Smallmouth Bass or Walleye resulted in target organ HIs >1 for the developmental, nervous, and skin/hair/nails systems; sucker consumption resulted in developmental, nervous, and reproductive target organ system HIs >1; and consumption of whitefish resulted in target organ HIs >1 for the reproductive and skin/hair/nails systems. None of these fish consumption exposure pathways resulted in exceedance of non-cancer benchmarks

under CTE exposure assumptions. When the exposure pathways were summed together under the RME scenario for the current child resident population, at least some residential DUs had non-cancer target organ HIs >1 for each fish species consumed:

- Consumption of sucker: 588 DUs had HI >1 for developmental, nervous, and reproductive target organ systems; 578 DUs had HI >1 for skin/hair/nails target organ system (see Figure 5-16)
- Consumption of Walleye: 588 DUs had HI >1 for developmental, nervous, and skin/hair/nails target organ systems; 9 DUs had HI >1 for endocrine system; 2 DUs had HI >1 for cardiovascular system (see Figure 5-17)
- Consumption of Smallmouth Bass: 588 DUs had HI >1 for developmental, nervous, and skin/hair/nails target organ systems
- Consumption of whitefish: 588 DUs had HI >1 for nervous, reproductive, and skin/hair/nails target organ systems
- Consumption of Burbot: 588 DUs had HI >1 for developmental and nervous target organ systems; 43 DUs had HI >1 for skin/hair/nails target organ system (see Figure 5-12)
- Consumption of Northern Pike: 588 DUs had HI >1 for nervous and skin/hair/nails target organ systems
- Consumption of Rainbow Trout: 588 DUs had HI >1 for skin/hair/nails target organ system; 577 DUs had HI >1 for nervous system (see Figure 5-14)
- Consumption of kokanee: 588 DUs had HI >1 for skin/hair/nails target organ system; 219 DUs had HI >1 for nervous system (see Figure 5-13)
- Consumption of White Sturgeon: 588 DUs had HI >1 for nervous system; 18 DUs had HI >1 for skin/hair/nails target organ system (see Figure 5-15)

The major contributor to skin/hair/nails target organ risk was thallium, and the major contributor to developmental and nervous system target organ risk was methylmercury.

Under CTE exposure assumptions, when all exposure pathways were summed, no target organ HIs exceeded 1 when the fish species consumed was Burbot, Northern Pike, Rainbow Trout, Smallmouth Bass, White Sturgeon, sucker, or whitefish. Target organ HIs were >1 at one house DU for the skin/hair/nails system when the fish consumed was kokanee or Walleye (see Figure 5-18). This DU exceeded the lead risk benchmark of P8.

Current Resident Population (Beach DUs)

The current resident-with-beach population was evaluated at 21 residential beaches sampled in 2014 and 2016 for exposure to outdoor soil and indoor dust, surface sediment, UCR surface water, outdoor and indoor air, and consumption of fish caught from the UCR (by individual fish species). Table 7-2 summarizes the number of DUs that exceeded risk benchmarks for both non-lead and lead COPCs for the current resident population with a beach on the property without including consumption of fish. For COPCs other than lead, fish consumption drives the non-

cancer risk results, and the major contributors to the increased risk from fish ingestion were methylmercury, thallium, and total TEQ. Beach sediment is less contaminated than soil.

Lead

Of 21 residential beach DUs evaluated, 19 exceeded the lead risk benchmark of P3, and 5 exceeded P5 not including consumption of local fish (see Figure 5-7). No residential beach DUs exceeded P8. Consuming fish from the UCR may pose an additional risk to current residents with beaches (see Appendix 5 for lead batch mode results). As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

COPCs Other Than Lead

For the non-lead evaluation, exposures from each pathway were summed to evaluate both cancer and non-cancer effects to the current residential population (with a beach) as a whole, by beach DU. When cancer risk was summed across a lifetime and across exposure pathways for the residential population, no residential beach DUs exceeded the cancer risk benchmark for either the RME or CTE scenario.

For the non-cancer evaluation for the current adult resident population (with residential beaches), no exposure pathway on its own resulted in exceedances of target organ risk benchmarks under either the RME or CTE scenario. When these pathways were summed to look at total exposure to current adult residents with beaches on their property, all residential beach DUs had non-cancer target organ HIs >1 for the nervous system when the fish consumed was sucker. These exceedances occurred under the RME exposure scenario, and the major contributor to risk was methylmercury. Consumption of all other fish species evaluated did not result in non-cancer benchmark exceedances. When CTE exposure conditions were assumed, no DUs exceeded non-cancer benchmarks at any residential beach DU.

For non-cancer evaluation of the current child resident-with-beach population, the only individual exposure pathways with target organ HIs >1 under the RME scenario were consumption of individual fish species other than Northern Pike and White Sturgeon. Consumption of Burbot resulted in target organ HIs >1 for the developmental and nervous systems; consumption of kokanee or Rainbow Trout resulted in the skin/hair/nails target organ system HI >1; consumption of Smallmouth Bass or Walleye resulted in target organ HIs >1 for the developmental, nervous, and skin/hair/nails systems; sucker consumption resulted in developmental, nervous, and reproductive target organ system HIs >1; and consumption of whitefish resulted in target organ HIs >1 for the reproductive and skin/hair/nails systems. None of these fish consumption exposure pathways resulted in exceedance of non-cancer benchmarks under CTE exposure assumptions. When all exposure pathways (air, water, sediment, soil/dust, and fish) were summed together for the child resident with a beach, all 21 residential beach DUs had non-cancer target organ HIs >1 under RME assumptions for:

- The nervous system for consumption of all individual fish species except kokanee and Rainbow Trout. Twenty residential beach DUs had HIs >1 for consumption of Rainbow Trout.
- The developmental system for consumption of Burbot, Smallmouth Bass, sucker, and Walleye

- The reproductive system for consumption of sucker and whitefish
- The skin/hair/nails system for consumption of kokanee, Northern Pike, Rainbow Trout, Smallmouth Bass, sucker, Walleye, and whitefish. One residential beach DU also had HI >1 for the skin/hair/nails system for consumption of Burbot and White Sturgeon.

For the CTE scenario, no target organ HIs were >1 for children at residential beach DUs when exposure pathways were summed.

Potential Future Resident Population

The potential future resident population was evaluated for exposure to outdoor soil and indoor dust, outdoor and indoor air, and consumption of fish caught from the UCR (evaluated as individual fish species). This was done on an aerial deposition area (ADA)-by-ADA basis (i.e., conservatively assuming that the potential future resident lived full-time on that ADA). Table 7-3 summarizes the number of ADAs that exceeded risk benchmarks for both non-lead and lead COPCs for the potential future resident population.

Lead
Of 142 upland ADAs evaluated, 139 ADAs exceeded the lead benchmark of P3 (98%), 68 ADAs exceeded P5 (48%), and 15 exceeded P8 (11%) not including consumption of UCR fish (see Figures 5-4 through 5-6). Consuming fish from the UCR may pose an additional risk to potential future residents (see Appendix 5 for lead batch mode results). As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead.

COPCs Other Than Lead
For the non-lead evaluation, exposures from each pathway were summed to evaluate both cancer and non-cancer effects to the potential future residential population as a whole. When cancer risk was summed across a lifetime and across exposure pathways for the potential future residential population, no ADAs exceeded the cancer risk benchmark of 10^{-4} for either the RME or CTE scenario.

For the non-cancer evaluation for the adult future resident population, no exposure pathway on its own resulted in exceedances of target organ risk benchmarks under either the RME or CTE scenario. When these pathways were summed to look at total exposure to future adult residents, all 142 ADAs had non-cancer target organ HIs >1 for the nervous system when the fish consumed was sucker. These exceedances occurred under the RME exposure scenario; the major contributor to risk was methylmercury. Consumption of all other fish species evaluated did not result in non-cancer benchmark exceedances. When CTE exposure conditions were assumed, no DUs exceeded non-cancer benchmarks for potential future adult residents.

For the non-cancer evaluation for the future child resident population, inhalation of outdoor and indoor air and consumption of Northern Pike or White Sturgeon, as individual exposure pathways, did not result in exceedance of non-cancer benchmarks at any ADAs under either RME or CTE scenarios. For the exposure to soil/dust pathway, two ADAs had a target organ HI >1 for the skin/hair/nails system with RME exposure assumptions but did not exceed benchmarks with CTE exposure assumptions. These two ADAs exceed the lead risk benchmark of P8 as well, as shown in Figure ES-2.

Under the RME scenario for the fish consumption pathway for the future child resident population, consumption of Burbot resulted in target organ HIs >1 for the developmental and nervous systems; consumption of kokanee or Rainbow Trout resulted in the skin/hair/nails target organ system HI >1; consumption of Smallmouth Bass or Walleye resulted in target organ HIs >1 for the developmental, nervous, and skin/hair/nails systems; sucker consumption resulted in developmental, nervous, and reproductive target organ system HIs >1; and consumption of whitefish resulted in target organ HIs >1 for the reproductive and skin/hair/nails systems. None of these fish consumption exposure pathways resulted in exceedance of non-cancer benchmarks under CTE exposure assumptions. When the exposure pathways were summed together under the RME scenario, at least some ADAs had non-cancer target organ HIs >1 for each fish species consumed:

- Consumption of sucker: 142 ADAs had HI >1 for developmental, nervous, reproductive, and skin/hair/nails target organ systems
- Consumption of Walleye: 142 ADAs had HI >1 for developmental, nervous, and skin/hair/nails target organ systems; 3 ADAs had HI >1 for the endocrine target organ system; 1 ADA had HI >1 for the cardiovascular system (see Figure 5-22)
- Consumption of Smallmouth Bass: 142 ADAs had HI >1 for developmental, nervous, and skin/hair/nails target organ systems
- Consumption of whitefish: 142 ADAs had HI >1 for nervous, reproductive, and skin/hair/nails target organ systems
- Consumption of Burbot: 142 ADAs had HI >1 for developmental and nervous target organ systems; 55 ADAs had HI >1 for skin/hair/nails target organ system (see Figure 5-19)
- Consumption of Northern Pike and Rainbow Trout: 142 ADAs had HI >1 for nervous and skin/hair/nails target organ systems
- Consumption of kokanee: 142 ADAs had HI >1 for skin/hair/nails target organ system; 122 ADAs had HI >1 for nervous system (see Figure 5-20)
- Consumption of White Sturgeon: 142 ADAs had HI >1 for nervous system; 21 ADAs had HI >1 for skin/hair/nails target organ system (see Figure 5-21)

The major non-lead contributors to risk were methylmercury (developmental and nervous systems), thallium (skin/hair/nails system), and dioxins and dioxin-like PCBs (reproductive system).

Under CTE exposure assumptions for the future child resident, when all exposure pathways were summed, no target organ HIs were >1 when the fish species consumed was Burbot, kokanee, Northern Pike, Rainbow Trout, Smallmouth Bass, White Sturgeon, or whitefish. Target organ HIs were >1 at two ADAs for the nervous system when the fish consumed was sucker, and one ADA had a target organ HI >1 for the skin/hair/nails system when Walleye was the fish species consumed (see Figure 5-23). One of the ADAs with nervous system HI >1 when sucker was consumed exceeded the lead risk benchmark of P8, and the other ADA exceeded the lead risk benchmark of P3. The ADA with the skin/hair/nails HI >1 when Walleye was consumed

exceeded the lead risk benchmark of P8. The major non-lead contributors to risk were methylmercury (developmental, nervous system) and thallium (skin/hair/nails).

Recreational Visitor Population

The adult and child recreational visitor population was evaluated for exposure on beach day trips, boating and camping trips to UCR surface water, outdoor air, public beach surface sediment or beach surface soil, relict floodplain surface soil, and consumption of fish caught in the UCR (evaluated by individual species).

Lead
For day trips to public beaches, the lead risk benchmark of P3 was exceeded at all public beaches with or without consumption of fish. As shown in Table 5-1, consumption of sucker from the UCR would result in the highest exposure to lead. Lead batch mode results for individual fish species are presented in Appendix 5. The P5 benchmark was only exceeded at Bossburg Flat Beach (based on 2011 sampling and 2013 reanalysis data) with or without fish consumption (Figure 5-8). No public beaches exceeded P8. These lead results are the same for boating and camping trips as well. Consuming fish from the UCR may pose an additional lead risk to recreational visitors. For beach day trips, camping and boating trips to Bossburg Flat Beach and exposure to surface soil in upland DUs (UDUs), all UDUs exceeded P3 and one UDU exceeded P5 and P8 with or without consumption of UCR fish. For relict floodplains, with or without UCR fish consumption, all relict floodplain deposition areas (RFDAs) exceeded the lead risk benchmark of P3, and no RFDAs exceed P5 or P8.

COPCs Other Than Lead
For the non-lead evaluation, exposures from each pathway were summed to evaluate both cancer and non-cancer effects to the recreational visitor population as a whole. When cancer risk was summed across a lifetime and across exposure pathways for the recreational visitor population, no public beaches or relict floodplains exceeded the cancer risk benchmark regardless of trip type for both the RME and CTE scenarios.

For the non-cancer evaluation for the adult recreational visitor population, no exposure pathway on its own resulted in exceedances of target organ risk benchmarks under either the RME or CTE scenario. When these pathways were summed to look at total exposure to adult recreational visitors, there were no target organ HIs >1 at any public beach, SDU, RFDA, or UDU for the RME and CTE scenarios, regardless of trip type.

The only individual exposure pathway that exceeded non-cancer risk benchmarks under the RME scenario for target organ HIs for the child recreational visitor population was the consumption of fish species except Northern Pike and White Sturgeon. For the RME scenario, consumption of fish species resulted in non-cancer target organ HIs >1 for the following systems at each public beach, SDU, RFDA, and UDU for each trip type:

- Developmental system: Burbot, Smallmouth Bass, sucker, and Walleye
- Nervous system: Burbot, Smallmouth Bass, sucker, and Walleye
- Reproductive system: sucker and whitefish

- Skin/hair/nails system: kokanee, Rainbow Trout, Smallmouth Bass, Walleye, and whitefish

When exposure pathways were summed, there were no target organ HIs >1 at any public beach, SDU, relict floodplain, or UDU (regardless of trip type) for the child recreational visitor, under RME exposure assumptions, when the fish species consumed was Northern Pike or White Sturgeon. There were non-cancer target organ HIs >1 under the RME scenario at each public beach, SDU, relict floodplain, or UDU (regardless of trip type) for the developmental, nervous, reproductive, and skin/hair/nails system for the child recreational visitor consuming the following species:

- Developmental system: Burbot, Smallmouth Bass, sucker, and Walleye
- Nervous system: Burbot, Smallmouth Bass, sucker, and Walleye
- Reproductive system: sucker and whitefish
- Skin/hair/nails system: kokanee, Rainbow Trout, Smallmouth Bass, Walleye, and whitefish

The major non-lead contributors to risk were methylmercury (developmental and nervous systems), thallium (skin/hair/nails system), and dioxins and dioxin-like PCBs (reproductive system).

For the CTE scenario, none of the exposure pathways evaluated had target organ HIs >1 for child recreational visitors at any public beach, SDU, RFDA, or UDU (regardless of trip type). No target organ HIs >1 at any public beach, SDU, RFDA, or UDU for child recreational visitors when exposure was summed across pathways, regardless of trip type.

Outdoor Worker Population

The adult outdoor worker population was evaluated for exposure to surface and subsurface sediment or soil at public beaches, UCR surface water, and outdoor air. This included each public beach sampled in 2009-2011, 2013, and 2015.

Lead
Subsurface sediment or soil exposure at public beaches exceeded P3 at three exposure areas at Bossburg Flat Beach and did not exceed P5 or P8.

COPCs Other Than Lead
Under both the RME and CTE scenario, non-cancer target organ HIs did not exceed the non-cancer benchmark of 1 at any public beach for the outdoor worker population. Cancer risks summed across exposure pathways were below the cancer risk benchmark of 10^{-4} at all public beaches evaluated.

Conclusions

This HHRA evaluated risks under the assumption that no additional steps are taken to remediate the environment or to reduce human contact with contaminated environmental media. The following conclusions are supported by this HHRA:

1. The main chemical contributing to risk in sediment was lead.
2. For lead, exposure to residential and upland soil was the dominant contributor to risk. The number of DUs and ADAs exceeding lead risk benchmarks are provided in Table 7-4.
3. For non-lead COPCs, exposure to residential and upland soil, air, and fish, summed across exposure pathways, resulted in target organ HIs >1. Fish consumption was the largest contributor to risk when aggregated by target organ. The major contributors to risk from ingestion of fish were methylmercury (developmental, nervous system), thallium (skin/hair/nails system), and dioxins and dioxin-like PCBs (reproductive system). Measured concentrations of COPCs in fish collected from the UCR are similar to concentrations measured in other locations in the northeastern portion of Washington and in Idaho. UCR fish advisories are similar to (Largemouth Bass, 2 meals per month; Northern Pikeminnow, do not eat; Washington State Department of Health [WDOH], 2020) or less restrictive than (Smallmouth Bass, 1 meal per month, WDOH, 2012) Statewide advisories.[110] When only soil/dust exposure was evaluated, no DUs or ADAs had non-cancer target organ benchmark exceedances for adult residents (current or potential future). For child residents, one house DU (current resident) and two ADAs (future resident) had HIs >1 for the skin/hair/nails target organ system.
4. Risks associated with exposure to sediment and surface water in isolation were significantly lower than for other exposure pathways.
5. For lead, the only public beach exceeding P5 was Bossburg Flat Beach. One UDU at Bossburg Flat Beach >P8 for recreational exposure.
6. For lead, consumption of fish from the UCR was not a major contributor to risk; however, consuming fish from the UCR can increase lead exposure. Avoiding consumption of sucker can reduce exposure to lead when harvesting fish from the UCR for consumption.
7. Consideration of background soil concentration lead will influence P3 results, but not P5 or P8 results. Background soil lead levels are estimated to be 35 ppm, which is 70% of (or similar to) the PRG associated with P3 (50 ppm).
8. For workers, the only public beach subsurface sediment that exceeded risk benchmarks were two Bossburg Flat Beach UDUs (these exceeded P3, but not P5 or P8).
9. The main chemical contributors to risk in soil were lead and arsenic; in many DUs and ADAs, their measured concentrations exceeded estimated background concentrations. Other COPCs that contributed to calculated risk from exposure to soil were thallium,

[110] https://www.doh.wa.gov/CommunityandEnvironment/Food/Fish/Advisories/Publications.

manganese, cobalt, and iron; they were measured below estimated background concentrations in many DUs.

10. Exposure to Site-related COPCs in air has been a public concern at the Site because of the source of contamination. However, as discussed in detail in the body of the report, exposure to COPCs via the air pathway alone did not exceed risk benchmarks for lead, non-cancer effects from other chemicals, or cancer. The concentration of lead in UCR air is an order of magnitude lower than the default air lead concentration in the IEUBK model. The last Site-specific air data were collected in 2009 at one location near Northport. However, emissions from the Trail, B.C., Canada smelter as reported to the Canadian National Pollutant Release Inventory from 2002 to 2017 show that while individual metals may be emitted at varying rates, aerial emissions of arsenic and lead are generally lower than in years prior to 2009. Exposure to airborne contaminants from the Teck smelter do not pose a substantial risk to Site residents, recreators, or workers.
11. The HHRA results are believed to be sound and appropriate to support remedy selection and risk management.

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