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 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 on 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.

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 from the United States (U.S.) – Canadian border south to the Grand Coulee Dam. 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 Resources Limited ("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 (U.S. Environmental Protection Agency [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 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[2]; 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;
- *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;
- *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.

[2] 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.

A Site-specific Recreational Consumption and Resource Use Survey ("RecUse Survey") (Industrial Economics, Inc. [IEc], 2012; SRC, 2019a) was conducted and provided estimates of frequency of use and of fish consumption that were incorporated into this HHRA. The HHRA for the CHIRU population of the Confederated Tribes of the Colville Reservation (CCT) is presented in Appendix 1, and the HHRA for the STI population is presented in Appendix 2. A Site-specific Tribal Consumption and Resource Use Survey ("Tribal Survey") (Westat, 2012; SRC, 2019b) was conducted and provided exposure parameters that were used to evaluate the CHIRU population. Exposure parameters provided by the STI from the 2006 STI Hazardous Substances Control Act (HSCA) 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.

The following is a general 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[3], thallium and dioxins and dioxin-like 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
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

[3]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	

↓ 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 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%.

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.[4] 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 from 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).

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 to distinguish risks that are a potential concern from risks that are below the level of concern. The Office of Solid Waste and Emergency Response (OSWER) (EPA, 1994a) identified a risk reduction goal of limiting

[4] 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.

exposure to lead such that children would have an estimated risk of no more than 5 percent (%) probability of exceeding a blood lead concentration (PbB) of 10 micrograms (µg) per deciliter (dL). The Office of Land and Emergency Management (OLEM) (EPA, 2016a) highlighted the current science and risk assessment tools that EPA Regions may consider when implementing the 1994 OSWER Directive 9355.4-12. EPA's Office of Research and Development (ORD) reviewed the health effects evidence 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 to 11 years old) with mean or group PbBs between 2 and 8 µg/dL (measured at various life stages and time periods)" (EPA, 2013a). 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.

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). These benchmarks are within the risk range of child PbBs associated with adverse health effects (2-8 µg/dL) cited in the 2016 OLEM Soil Lead Directive (EPA, 2016a). The IEUBK model cannot be used with a risk benchmark of 2 µg/dL because with default parameter values and assuming no lead in soil (0 parts per million [ppm]), the IEUBK model will calculate more than 5% probability that PbB will be >2 µg/dL (that is to say that the P2 risk goal is exceeded if the soil lead concentration is 0 ppm). The estimated background soil lead concentration for this Site is approximately 35 ppm (Appendix 4). 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.

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 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[5] 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

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

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 $>10^{-4}$.

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.[6] Those DUs were included in the evaluations in this report using their post-removal soil concentrations to represent current exposure conditions.

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.

[6] 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.

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 5-10). 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 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 5-15)

- 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 5-16)
- 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 5-11)
- 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 5-13)
- 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 5-12)
- Consumption of sturgeon: 588 DUs had HI >1 for nervous system; 18 DUs had HI >1 for skin/hair/nails target organ system (see Figure 5-14)

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, 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 5-17). 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 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).

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 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 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).

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 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 5-21)
- 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 5-18)
- 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 5-19)
- Consumption of sturgeon: 142 ADAs had HI >1 for nervous system; 21 ADAs had HI >1 for skin/hair/nails target organ system (see Figure 5-20)

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, 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 5-22). 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. Lead 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. 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 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 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 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. The last air data were collected in 2009 at one location 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).