# EXECUTIVE SUMMARY FINAL SITE-WIDE HUMAN HEALTH RISK ASSESSMENT UPPER COLUMBIA RIVER SITE

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<sup>3</sup>; 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;<sup>4</sup>
- *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;

<sup>&</sup>lt;sup>3</sup> 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.

<sup>&</sup>lt;sup>4</sup> 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.

Receptor <sup>1</sup>	Lead <sup>2</sup>	<b>COPCS</b> other than Lead		Maian Contributors to Disk
		Non-cancer	Cancer	Major Contributors to Risk
Current resident without beach	↑ P8 <sup>3</sup>	1	$\downarrow$	<ul> <li>Lead in soil</li> <li>Methylmercury<sup>5</sup>, thallium and dioxins and dioxin-like polychlorinated biphenyls (PCBs) in fish</li> </ul>
Current resident with beach	↑ P5	Ţ	$\rightarrow$	<ul> <li>Lead in soil</li> <li>Methylmercury, thallium and dioxins and dioxin-like PCBs in fish</li> </ul>
Potential future resident	↑ P8	1	$\rightarrow$	<ul> <li>Lead in soil</li> <li>Methylmercury, thallium and dioxins and dioxin-like PCBs in fish</li> </ul>

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

<sup>&</sup>lt;sup>5</sup>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 <sup>1</sup>	Lead <sup>2</sup>	COPCS other than Lead		Maior Contributors to Disk
		Non-cancer	Cancer	Major Contributors to Risk
Recreational visitor, public beach sediment	↑ P5	Ţ	$\rightarrow$	<ul> <li>Lead in sediment</li> <li>Methylmercury, thallium and dioxins and dioxin-like PCBs in fish</li> </ul>
Recreational visitor, public beach soil	↑ P8	Ţ	$\downarrow$	<ul> <li>Lead in soil</li> <li>Methylmercury, thallium and dioxins and dioxin-like PCBs in fish</li> </ul>
Recreational visitor, relict floodplain soil	↑ P3	Ţ	$\downarrow$	<ul> <li>Lead in soil</li> <li>Methylmercury, thallium and dioxins and dioxin-like PCBs in fish</li> </ul>
Outdoor worker	↑ P3	$\downarrow$	$\rightarrow$	• Lead in beach soil and sediment

 $\uparrow$  at least one DU was above risk benchmark

 $\downarrow$  no DUs above benchmark

<sup>1</sup>Results for CHIRU and STI receptor populations are found in Appendices 1 and 2, respectively.

<sup>2</sup>All scenarios have greater than 5% probability of exceeding a blood lead level of 3 micrograms per deciliter  $(\mu g/dL)$  (P3).

<sup>3</sup>"P3," "P5," "P8:" Indicates the level of lead risk, not including fish. P3 = probability of exceeding a blood lead level of 3  $\mu$ g/dL is >5%; P5 = probability of exceeding a blood lead level of 5  $\mu$ g/dL is >5%; P8 = probability of exceeding a blood lead level of 8  $\mu$ g/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.<sup>6</sup>
- 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

<sup>&</sup>lt;sup>6</sup> 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.<sup>7</sup>
- 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.<sup>8</sup> 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

<sup>&</sup>lt;sup>7</sup> https://www.doh.wa.gov/Portals/1/Documents/Pubs/334-305.pdf.

<sup>&</sup>lt;sup>8</sup> 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  $\mu$ g/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  $\mu$ g/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.<sup>9</sup> 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  $3 \mu g/dL$  was associated with a soil concentration of approximately 50 ppm lead
- Utilizing the IEUBK model, a 5% probability of exceeding a PbB of 5  $\mu$ g/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  $\mu$ g/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

<sup>&</sup>lt;sup>9</sup> 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  $\mu$ g of lead per day<sup>10</sup> 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  $\mu$ g Pb/day. In this HHRA, risk from exposures that were predicted to contribute less than (<) 1  $\mu$ g 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<sup>-4</sup> 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

<sup>&</sup>lt;sup>10</sup> 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<sup>-4</sup>.

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.<sup>11</sup> 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.

<sup>&</sup>lt;sup>11</sup> List of TCRAs and VRAs is current as of June 20, 2019.

### <u>Lead</u>

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.

# <u>Lead</u>

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 of whitefish resulted in target organ 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<sup>-4</sup> 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.

#### <u>Lead</u>

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 noncancer 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<sup>-4</sup> 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.