

Health Consultation

LAKE ROOSEVELT BEACHES SEDIMENT

NON-TRIBAL EXPOSURE

NORTHEAST, WASHINGTON

EPA FACILITY ID: WA0000530279

**Prepared by the
Washington State Department of Health**

March 9, 2010

Prepared under a Cooperative Agreement with the
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

A health consultation is a verbal or written response from ATSDR or ATSDR's Cooperative Agreement Partners to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR or ATSDR's Cooperative Agreement Partner which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

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Foreword

The Washington State Department of Health (DOH) has prepared this health consultation in cooperation with the Agency for Toxic Substances and Disease Registry (ATSDR). ATSDR is part of the U.S. Department of Health and Human Services and is the principal federal public health agency responsible for health issues related to hazardous wastes. This health consultation was prepared in accordance with methodologies and guidelines developed by ATSDR.

The purpose of this health consultation is to identify and prevent harmful human health effects resulting from exposure to hazardous substances in the environment. Health consultations focus on specific health issues so that DOH can respond to requests from concerned residents or agencies for health information on hazardous substances. DOH evaluates sampling data collected from a hazardous waste site, determines whether exposures have occurred or could occur, reports any potential harmful effects, and recommends actions to protect public health. The findings in this report are relevant to conditions at the site during the time of this health consultation, and should not necessarily be relied upon if site conditions or land use changes in the future.

For additional information or questions regarding DOH or the contents of this health consultation, please call the health advisor who prepared this document:

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For persons with disabilities this document is available on request in other formats. To submit a request, please call 1-800-525-0127 (voice) or 1-800-833-6388 (TTY/TDD).

For more information about ATSDR, contact the ATSDR Information Center at 1-888-422-8737 or visit the agency's Web site: www.atsdr.cdc.gov/.

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Glossary

Agency for Toxic Substances and Disease Registry (ATSDR)	The principal federal public health agency involved with hazardous waste issues, responsible for preventing or reducing the harmful effects of exposure to hazardous substances on human health and quality of life. ATSDR is part of the U.S. Department of Health and Human Services.
Cancer Risk	A theoretical risk for developing cancer if exposed to a substance every day for 70 years (a lifetime exposure). The true risk might be lower.
Cancer Risk Evaluation Guide (CREG)	The concentration of a chemical in air, soil or water that is expected to cause no more than one excess cancer in a million persons exposed over a lifetime. The CREG is a <i>comparison value</i> used to select contaminants of potential health concern and is based on the <i>cancer slope factor</i> (CSF).
Cancer Slope Factor	A number assigned to a cancer-causing chemical that is used to estimate its ability to cause cancer in humans.
Carcinogen	Any substance that causes cancer.
Comparison value	Calculated concentration of a substance in air, water, food, or soil that is unlikely to cause harmful (adverse) health effects in exposed people. The CV is used as a screening level during the public health assessment process. Substances found in amounts greater than their CVs might be selected for further evaluation in the public health assessment process.
Contaminant	A substance that is either present in an environment where it does not belong or is present at levels that might cause harmful (adverse) health effects.
Dermal Contact	Contact with (touching) the skin (see route of exposure).
Dose (for chemicals that are not radioactive)	The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An “exposure dose” is how much of a substance is encountered in the environment. An “absorbed dose” is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.
Environmental Media Evaluation Guide (EMEG)	A concentration in air, soil, or water below which adverse non-cancer health effects are not expected to occur. The EMEG is a <i>comparison value</i> used to select contaminants of potential health concern and is based on ATSDR’s <i>minimal risk level</i> (MRL).

Environmental Protection Agency (EPA)	United States Environmental Protection Agency.
Exposure	Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [acute exposure], of intermediate duration, or long-term [chronic exposure].
Groundwater	Water beneath the earth's surface in the spaces between soil particles and between rock surfaces [compare with surface water].
Hazardous substance	Any material that poses a threat to public health and/or the environment. Typical hazardous substances are materials that are toxic, corrosive, ignitable, explosive, or chemically reactive.
Ingestion	The act of swallowing something through eating, drinking, or mouthing objects. A hazardous substance can enter the body this way [see route of exposure].
Ingestion rate	The amount of an environmental medium that could be ingested typically on a daily basis. Units for IR are usually liter/day for water, and mg/day for soil.
Inhalation	The act of breathing. A hazardous substance can enter the body this way [see route of exposure].
Inorganic	Compounds composed of mineral materials, including elemental salts and metals such as iron, aluminum, mercury, and zinc.
Lowest Observed Adverse Effect Level (LOAEL)	The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.
Maximum Contaminant Level (MCL)	A drinking water regulation established by the federal Safe Drinking Water Act. It is the maximum permissible concentration of a contaminant in water that is delivered to the free flowing outlet of the ultimate user of a public water system. MCLs are enforceable standards.
Media	Soil, water, air, plants, animals, or any other part of the environment that can contain contaminants.

<p>Minimal Risk Level (MRL)</p>	<p>An ATSDR estimate of daily human exposure to a hazardous substance at or below which that substance is unlikely to pose a measurable risk of harmful (adverse), noncancerous effects. MRLs are calculated for a route of exposure (inhalation or oral) over a specified time period (acute, intermediate, or chronic). MRLs should not be used as predictors of harmful (adverse) health effects [see oral reference dose].</p>
<p>Model Toxics Control Act (MTCA)</p>	<p>The hazardous waste cleanup law for Washington State.</p>
<p>No apparent public health hazard</p>	<p>A category used in ATSDR’s public health assessments for sites where human exposure to contaminated media might be occurring, might have occurred in the past, or might occur in the future, but where the exposure is not expected to cause any harmful health effects.</p>
<p>No Observed Adverse Effect Level (NOAEL)</p>	<p>The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.</p>
<p>Oral Reference Dose (RfD)</p>	<p>An amount of chemical ingested into the body (i.e., dose) below which health effects are not expected. RfDs are published by EPA.</p>
<p>Organic</p>	<p>Compounds composed of carbon, including materials such as solvents, oils, and pesticides that are not easily dissolved in water.</p>
<p>Parts per billion (ppb)/Parts per million (ppm)</p>	<p>Units commonly used to express low concentrations of contaminants. For example, 1 ounce of trichloroethylene (TCE) in 1 million ounces of water is 1 ppm. 1 ounce of TCE in 1 billion ounces of water is 1 ppb. If one drop of TCE is mixed in a competition size swimming pool, the water will contain about 1 ppb of TCE.</p>
<p>Plume</p>	<p>A volume of a substance that moves from its source to places farther away from the source. Plumes can be described by the volume of air or water they occupy and the direction they move. For example, a plume can be a column of smoke from a chimney or a substance moving with groundwater.</p>
<p>Reference Dose Media Evaluation Guide (RMEG)</p>	<p>A concentration in air, soil, or water below which adverse non-cancer health effects are not expected to occur. The RMEG is a <i>comparison value</i> used to select contaminants of potential health concern and is based on EPA’s oral reference dose (RfD).</p>
<p>Route of exposure</p>	<p>The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].</p>

<p>Surface Water</p>	<p>Water on the surface of the earth, such as in lakes, rivers, streams, ponds, and springs [compare with groundwater].</p>
<p>Time Weighted Approach (TWA)</p>	<p>The exposure concentration of a contaminant during a given period.</p>
<p>Volatile organic compound (VOC)</p>	<p>Organic compounds that evaporate readily into the air. VOCs include substances such as benzene, toluene, methylene chloride, and methyl chloroform.</p>

Summary and Statement of Issues

Introduction:

The northern reach of the Columbia River (Upper Columbia River) includes Franklin D. Roosevelt Lake (Lake Roosevelt) which for the purpose of this health consultation, Lake Roosevelt and the Upper Columbia River is treated as a contiguous site (Lake Roosevelt). In the Lake Roosevelt community, Washington State Department of Health's (DOH) top priority is to ensure that the community has the best information possible to safeguard its health. The Washington State Department of Health (DOH) has prepared this health consultation at the request of the U.S. Environmental Protection Agency (EPA). The purpose of this health consultation is to evaluate the potential human health hazard posed by contaminants in sediments along the beaches of Lake Roosevelt in northeast Washington. Smelting related contaminants (slag and heavy metals (inorganic compounds)) and other contaminants (organic compounds) were found in beach sediments taken between the U.S./Canadian border and Grand Coulee Dam. DOH prepares health consultations under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR).

Overview:

DOH reached three important conclusions about Lake Roosevelt beach sediment in northeast Washington.

Conclusion 1:

DOH concludes that touching, breathing, or accidentally eating sediment exposed in a two-days-per-week for four months or 35-days-per-year (area residents) scenario is not expected to harm people's health.

Basis for decision:

Although certain contaminants maximum values are above screening and background levels, exposure to average contaminants levels are below levels known to result in non-cancer harmful health effects. In addition, the exposure scenario does not present an elevated theoretical cancer risk.

Conclusion 2:

DOH concludes that touching, breathing, or accidentally eating sediment exposed in a 14-days-per-year (2 weeks per year) vacationer scenario is not expected to harm people's health.

Basis for decision:

Although certain contaminants' maximum values are above screening and background levels, exposure to average contaminant levels are below levels known to result in non-cancer harmful health effects. In addition, the exposure scenario does not present an elevated theoretical cancer risk.

Conclusion 3:

DOH concludes, since fugitive dust will be evaluated in the future, it is unknown if fugitive dust will harm people's health.

Basis for decision:

The fugitive dust pathway generated by the lowering of Lake Roosevelt reservoir was not evaluated.

Next Steps:

1. DOH will hold a series of availability sessions around Lake Roosevelt to address community health concerns related to exposure to Lake Roosevelt sediment, fish, and water.
2. DOH will provide fact sheets to communities indicating ways to reduce exposure to contaminants in beach sediments.
3. DOH will coordinate with EPA's RI/FS work in developing health messages.
4. DOH will explore the feasibility and appropriateness of signs posted at Lake Roosevelt beaches.
5. Currently, DOH is working on a health consultation for fish caught in Lake Roosevelt.
6. DOH will establish community repositories for the public health consultation and related fact sheets.

For More Information:

Please feel free to contact Lenford O'Garro at (360) 236-3376 or 1-877-485-7316 if you have any questions about this health consultation.

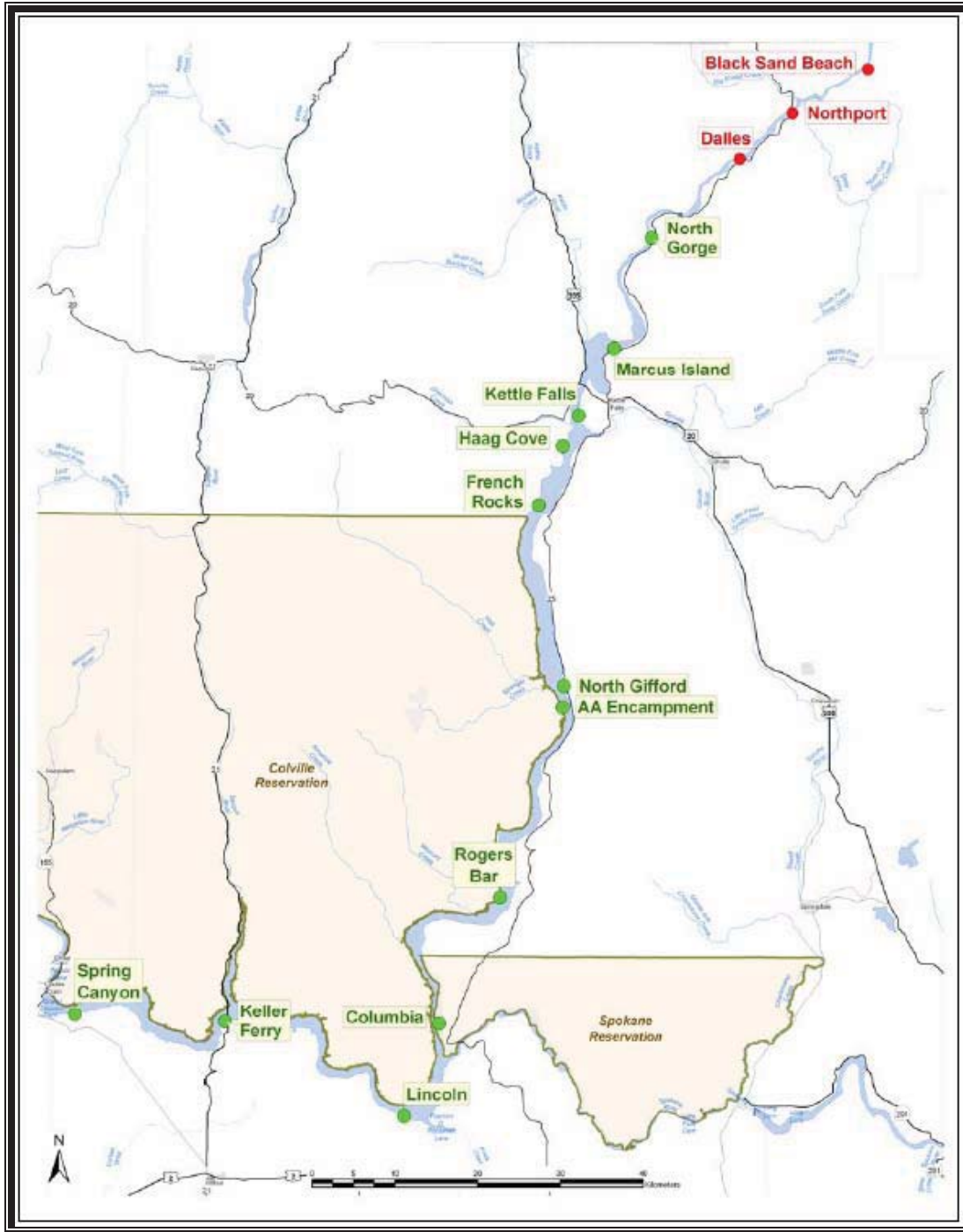
Background

The Columbia River flows from British Columbia, Canada southwards through eastern Washington, and west to the Pacific Ocean. The construction of the Grand Coulee Dam and reservoir on the Columbia River created Lake Roosevelt which is about 135 miles long [1]. The Columbia River contributes about 90% of the water flowing into Lake Roosevelt [1].

Smelting and mining activities in British Columbia and northeast Washington have left a legacy of contaminated byproduct (slag) along the beaches and in Lake Roosevelt. In August 1999, the Confederated Tribes of the Colville Indian Reservation (Colville Tribes) petitioned the U.S. Environmental Protection Agency (EPA) to assess human health and environmental risk of the Upper Columbia River [2]. In 2001, EPA conducted an expanded site inspection. EPA determined a Remedial Investigation/Feasibility Study (RI/FS) was necessary to evaluate human health and environmental risk of the Upper Columbia River due to widespread contamination in the lake and sediments [3]. Over the years, a number of studies were conducted on Lake Roosevelt's water, sediments, and fish. These studies showed various contaminants including heavy metals, dioxins/furans, and polychlorinated biphenyls (PCBs) [4, 5, 6, 7, 8, 9, 10].

In April and May 2005, EPA field crews collected sediment samples from Lake Roosevelt as part of the RI/FS phase 1 sampling. About 66 sediment samples were taken from 15 beaches along Lake Roosevelt and analyzed for total metals and organic compounds (See Figure 1). This health consultation covers only potential exposure to contaminants in sediment on Lake Roosevelt beaches. However, there are potential exposure pathways to metals and other contaminants through recreational water contact, drinking Lake Roosevelt water, and eating fish caught in Lake Roosevelt. These other pathways will be evaluated as more data becomes available from the initial phase 1 investigation and phase 2 sampling anticipated for 2009. Total exposures from all pathways may be addressed in future documents because data from all pathways are not available and those that are available (sediment and fish) are dominated by different types of contaminant of concern.

Figure 1. Upper Columbia River relief map (red dots – beaches with contaminants of concern) showing the sediment sample areas from beaches along Lake Roosevelt in northeast Washington.



Discussion

Environmental Sampling Data

Table 1 is a summary of the maximum level of each inorganic contaminant found along the 15 beaches. Appendix A, Table A1 is a summary of the maximum level of each organic contaminant found along the 15 beaches. None of the organic contaminants were found to be at levels of possible concern. Table 2 represents the maximum concentration of inorganic contaminants of concern (COC) detected in samples taken from each of the 15 beaches along Lake Roosevelt.

Uncertainty Non-detect Results

Some uncertainty is associated with any approach dealing with non-detected chemical samples (U). Non-detect results do not indicate whether the contaminant is present at a concentration just below the detection limit, present at a concentration just above zero, or absent from the sample. Therefore, contaminants that were evaluated as non-detects can lead to an overestimation of risk if the actual concentrations are just above zero, or absent from the sample. One-half the reported detection limit for non-detect samples (U) were used in the evaluation data set.

Table 1. Maximum detected or non-detected value of inorganic contaminants in beach sediment along Lake Roosevelt in northeast Washington.

Compounds	Maximum detected or non-detected value (ppm)	Comparison Value (ppm)	EPA Cancer Class	Comparison Value Reference	Contaminant of Concern
Aluminum	23,100	76,000		Region 9	No
Antimony	53J	20	D	RMEG	Yes
Arsenic	36	0.5 20	A	CREG EMEG	Yes (cancer) Yes (non-cancer)
Barium	2,250	10,000	D	RMEG	No
Beryllium	1.7	100	B1	EMEG	No
Cadmium	7.8	5	B1	EMEG	Yes
Calcium	80,300	NA		NA	No
Chromium	145	200 ^a	A	RMEG	No
Cobalt	57	500		IM EMEG	No
Copper	3,290	500	D	IM EMEG	Yes
Iron	254,000	23,000		Region 9	Yes
Lead	535	250	B2	MTCA	Yes
Magnesium	16,400				No
Manganese	4,780	3,000	D	RMEG	Yes
Mercury	0.81	1	D	MTCA	No
Nickel	30	1,000		RMEG	No
Potassium	4,730	NA		NA	No
Selenium	4.3	300	D	EMEG	No
Silver	1.5 UJ	300	D	RMEG	No
Sodium	2,780	NA		NA	No
Thallium	1.3 J / 3.8 U	5.2		Region 9	No
Uranium	84 J	100*		IM EMEG	No
Vanadium	50	200		IM EMEG	No
Zinc	22,200	20,000	D	EMEG	Yes

RMEG - ATSDR's Reference Dose Media Evaluation Guide (child)

CREG - Cancer Risk Evaluation Guide for 1×10^{-6} excess cancer risk

EMEG - ATSDR's Environmental Media Evaluation Guide (child)

IM EMEG - ATSDR's Intermediate Environmental Media Evaluation Guide (child)

J- data qualifier: (reported concentration is an estimated value).

U- data qualifier: The analyte was not detected at this level.

UJ- data qualifier: (reported concentration is an estimated value).

A - EPA: Human carcinogen

B1 - EPA: Probable human carcinogen (limited human, sufficient animal studies)

B2 - EPA: Probable human carcinogen (inadequate human, sufficient animal studies)

D - EPA: Not classifiable as to health carcinogenicity

Region 9 – EPA: Preliminary Remediation Goals

MTCA – Washington State Department of Ecology: Model Toxics Control Act

^a - chromium hexavalent RMEG value was used as a surrogate

* Highly Soluble Uranium Salts, IM EMEG value was used as a surrogate

NA – Not available

ppm -parts per million

Bold values exceed comparison values

Table 2: Maximum concentration of inorganic contaminants of concern in sediment detected on each beach sampled along Lake Roosevelt in northeast Washington.

Beaches	Antimony (ppm)	Arsenic (ppm)	Cadmium (ppm)	Copper (ppm)	Iron (ppm)	Lead (ppm)	Manganese (ppm)	Zinc (ppm)
Black Sand Beach	52 J	27	1.4	2,350	211,000	276	3,680	16,900
Northport Boat Ramp	53 J	36	4.8	3,290	254,000	535	4,780	22,200
Dalles Orchard	32 J	23	2.5	1,380	110,000	214	2,200	8,700
North Gorge Campground	6.4 J	11	4.2	216	29,500	223	434	17,000
Marcus Island Campground	8.1 J	8.6	7.3	58	23,400	297	246	915
Kettle Falls Swim Beach	6.4 UJ	2.3	0.36 J	27	26,000	11	605	74
Haag Cove	1.8 J	2.3	7.8	34	18,100	222	267	700
French Rocks Boat Ramp	1.0 UJ	2.6	0.51	15	12,000	22	260	97
North Gifford	1.6 J	7.0	3.1	29	22,600	102	526	295
A A Campground	1.5 J	5.3	1.0	10	21,100	34	383	158
Roger's Bar	1.2 UR	2.2	0.22 J	9.0	9,800	5.4	157	47
Columbia Campground	1.8 UJ	6.4	2.0	23	18,700	119	340	366
Lincoln Mills Boat Ramp	1.3 UR	6.7	0.47 U	12	14,200	6.2	334	36
Keller Ferry No.2	0.95 UR	4.9	0.50 U	9.1	15,600	6.3	248	44
Spring Canyon Campground	1.1 UJ	10	0.52 U	7.1	15,400	7.1	227	55
Comparison Value (ppm)	20	0.5 (cancer) 20 (noncancer)	5	500	23,000	250	3,000	20,000

J- data qualifier: (reported concentration is an estimated value).

U- data qualifier: The analyte was not detected at this level.

UJ- data qualifier: (reported concentration is an estimated value).

UR- data qualifier: (reported concentration is an estimated value).

ppm -parts per million

Bold values exceed comparison values

Contaminants of Concern

Contaminants of concern in sediments were determined by employing a screening process. Maximum sediment contaminant levels from each beach location were screened against health-based comparison values. Several types of health-based comparison or screening values are used during this process [see the glossary for descriptions of “comparison value,” “cancer risk evaluation guide (CREG),” “environmental media evaluation guide (EMEG),” and “reference dose media evaluation guide (RMEG)”]. Comparison values such as the CREG and EMEG offer a high degree of protection and assurance that people are unlikely to be harmed by contaminants in the environment. For chemicals that cause cancer, the comparison values represent levels that are calculated to increase the risk of cancer by about one in a million. With the exception of lead, the comparison values for chemicals that do not cause cancer represent levels that are not expected to cause any health problems. For lead, comparison values are usually based on the goal of keeping blood lead levels in most children below 10 micrograms per deciliter ($\mu\text{g}/\text{dl}$). In general, if a contaminant’s maximum concentration is greater than its comparison value, then the contaminant is evaluated further.

Comparisons may also be made with legal standards such as the cleanup levels specified in the Washington State hazardous waste cleanup law, the Model Toxics Control Act (MTCA). Legal standards may be strictly health-based or they may incorporate non-health considerations such as the cost, the practicality of attainment, or natural background levels.

The following evaluation addresses arsenic, cadmium, antimony, copper, iron, lead, manganese, and zinc as contaminants of concern in sediments along Lake Roosevelt beaches. In order for any contaminant to be a health concern, the contaminant must be present at a high enough concentration to cause potential harm, and there must be a completed route of exposure to people. However, some of these contaminants are present in food and water at low levels also and humans may be exposed to these contaminants through other pathways unrelated to Lake Roosevelt.

Human use patterns and site-specific conditions are considered in the evaluation of exposure to arsenic, antimony, copper, iron, lead, manganese, and zinc through the following pathways and routes:

- Inadvertent sediment ingestion, dust particles inhalation, and dermal absorption of contaminants in sediment during beach play.

Beach Play Scenario

Although contact with sediments at the beaches may be an infrequent or seasonal exposure pathway, there is concern because some beaches had elevated levels of contaminants (see Table 2). Exposure to contaminants in sediment can occur by swallowing it (ingestion exposure), breathing it (inhalation exposure), or getting it on the skin (dermal exposure). During recreational activities at the beaches, people are likely to be exposed to contaminants in sediments.

Ingestion exposure (swallowing)

People may inadvertently swallow small amounts of sediments, soil, and dust (and any contaminants they contain). Young children often put hands, toys, pacifiers, and other things in their mouths, and these may have dirt or dust on them that can be swallowed. Adults may ingest sediments, soil, and dust through activities such as gardening, mowing, construction work, dusting, and in this case, recreational activities.

Pica behavior is a persistent eating of non-food substances (such as dirt or paper). In a small percentage of children, pica behavior has been found to result in the ingestion of relatively large amounts of soil (one or more grams per day). Compared to typical children, those who swallow large amounts of contaminated soil may have added risks from short-term exposure. Some adults may also exhibit pica behavior.

Inhalation exposure (breathing)

Although people can inhale suspended sediment or dust, airborne sediment usually consists of relatively large particles that are trapped in the nose, mouth, and throat and are then swallowed rather than breathed into the lungs.

Skin exposure (dermal)

Dirt particles that can adhere to the skin may cause additional exposure to contaminants through dermal absorption. Although human skin is an effective barrier for many environmental contaminants, some chemicals can move easily through the skin. Metals, such as those contaminants of concern on Lake Roosevelt beaches, do not pass easily through the skin.

Fugitive Dust Scenario

As part of the operational approach of Lake Roosevelt, the Grand Coulee Dam reservoir is lowered to make room for winter snowmelt in the spring and in the fall to support fish management. During those periods, the depth of the lake can decrease in excess of 60 feet exposing many square miles of potentially contaminated sediments along the lake. As the sediments dry out, there is a potential for them to become airborne during typical ambient wind conditions and during sustained strong wind events (4 or more hours with winds > 5.14 meters per second (m/s)). Thus, the potential for human exposure to airborne contaminants can occur during times of reservoir drawdown.

In January 2002, the U.S. Geological Survey (USGS) began a five-year air monitoring study of trace elements associated with slag and metallurgical waste. Air monitoring stations were set up in three areas (Seven Bays, Inchelium, and Kettle Falls, WA). Sampling was on a regular schedule from January through June and for one month in the fall. In 2003, the Kettle Falls sampling site was replaced with a sampling site at Marcus, WA. Extra samples were taken during high wind events.

Although people can inhale suspended soil or dust, airborne soil usually consists of relatively large particles that are trapped in the nose, mouth, and throat and are then swallowed rather than breathed into the lungs. DOH has obtained data for ambient windblown sediments or during wind events at Lake Roosevelt. DOH also understands that actual data is the best way to evaluate a pathway. DOH will evaluate fugitive dust scenario in a future health consultation.

Chemical Specific Toxicity

Below are general summaries of health effects of the COCs. The public health implications of exposure to these COCs from the beaches are discussed later.

Antimony

Antimony is a naturally occurring element in the earth's soil. Background soil antimony concentration ranges between 3.1 and 7.6 ppm in Washington [11]. The main routes of exposure to antimony are from inhaling contaminated soil or dust particles, and ingesting contaminated water or food. Antimony contaminated soil can accidentally be ingested by hand-to-mouth activity that could increase exposure. EPA established an RfD for antimony of 0.0004 mg/kg/day based on animal studies that showed it can cause decrease in blood glucose levels and altered cholesterol levels [12]. EPA has not classified antimony as to human health carcinogenicity.

Arsenic

Arsenic is a naturally occurring element in the earth's soil. Background soil arsenic concentrations in Eastern Washington range from about 0.5 to 10.3 ppm [11]. However, the widespread use of arsenic-containing pesticides and the emissions from certain smelters has resulted in significantly higher levels of arsenic on many properties in the state. There are two forms of arsenic: organic and inorganic. The EPA established reference dose (RfD) for arsenic is 0.0003 mg/kg/day based on skin color changes and excessive growth of tissue (human data) [13]. EPA classifies the inorganic form of arsenic as a human carcinogen. DOH is not using the slope factor of 1.5 per mg/kg/day due to the arsenic weight of evidence approach. The EPA Integrated Risk Information System (IRIS) review draft for the Science Advisory Board presented a slope factor for combined lung and bladder cancer of 5.7 per mg/kg/day [14]. The slope factor calculated from the work by the National Research Council is about 21 per mg/kg/day [15]. These slope factors could be higher if the combined risk for all arsenic-associated cancers (bladder, lung, skin, kidney, liver, etc.) were evaluated. For this or any other health consultation, DOH uses a slope factor of 5.7 per mg/kg/day which appears to reflect EPA's most recent assessment.

Cadmium

Cadmium is a naturally occurring element in the earth's soil. Background soil cadmium concentration ranges between 0.1 and 5.0 ppm, statewide in Washington State [11]. The EPA classified cadmium as a probable human carcinogen based on animal studies. The main routes of exposure to cadmium are from inhaling contaminated soil or dust particles, and by

ingesting contaminated water or food. Cadmium contaminated soil can accidentally be ingested by hand to mouth activity that could increase exposure. Cadmium is stored in the liver and kidneys and slowly leaves the body in the urine and feces [16]. Cadmium absorption through the skin is not normally an important pathway, very little enters through the skin. The EPA assumes a value of 2.5% for gastrointestinal absorption of cadmium in food. The EPA established RfD for cadmium in food is 0.001 mg/kg/day.

Copper

Copper is a naturally occurring element in the earth's soil. Background soil copper concentrations in Eastern Washington range from about 4 to 53 ppm [11]. Copper is an essential element for good health. Copper rapidly enters the bloodstream and is distributed throughout the body after ingestion. Copper combines with protein and iron to make hemoglobin, which transports oxygen in the blood from the lungs to other parts of the body. Copper usually takes several days to leave the body in feces and urine. However, exposure to very high doses of copper can cause liver and kidney damage and even death [17]. Water containing high levels of copper may cause nausea, vomiting, stomach cramps, or diarrhea when ingested. In addition, long-term exposure to copper dust can irritate the nose, mouth, and eyes and also cause headaches, dizziness, nausea, and diarrhea. The Health Effects Assessment Summary Tables (HEAST) established RfD for copper is 0.04 mg/kg/day.

Iron

Iron is a naturally occurring element in the earth's soil. Background soil iron concentrations in Eastern Washington range from about 9670 to 30000 ppm [11]. Iron is essential in the maintenance and production of hemoglobin and myoglobin without which the body cannot sustain basic life functions. Iron combines with protein and copper to make hemoglobin, which transports oxygen in the blood from the lungs to other parts of the body. Generally, acute iron poisoning is the result of children accidentally overdosing on iron-containing supplements for adults and not from incidentally ingesting iron in soil or sediment. The EPA provisional RfD for iron has been revised to 0.7 mg/kg/day [18].

Lead – Occurrence, Health Concerns, and Risks

Lead is a naturally occurring chemical element that is normally found in soil. In Washington, normal background concentrations rarely exceed 20 ppm [11]. However, the widespread use of certain products (such as leaded gasoline, lead-containing pesticides, and lead-based paint) and the emissions from certain industrial operations (such as smelters) has resulted in significantly higher levels of lead in many areas of the state.

Elimination of lead in gasoline and solder used in food and beverage cans has greatly reduced exposure to lead. Currently, the main pathways of lead exposure in children are ingestion of paint chips, contaminated soil and house dust, and drinking water in homes with old plumbing.

Children seven years old and under are particularly vulnerable to the effects of lead. Compared to older children and adults, they tend to ingest more dust and soil, absorb significantly more of

the lead that they swallow, and more of the lead that they absorb can enter their developing brain. Pregnant women and women of childbearing age should also be aware of lead in their environment because lead ingested by a mother can affect the unborn fetus.

Health effects

Exposure to lead can be monitored by measuring the level of lead in the blood. In general, blood lead rises 1-5 micrograms per deciliter ($\mu\text{g}/\text{dl}$) for every 1,000 ppm increase in soil lead concentration [19]. For children, the Centers for Disease Control and Prevention (CDC) has defined an elevated blood lead level (BLL) as greater than or equal to 10 $\mu\text{g}/\text{dl}$ [20]. However, there is growing evidence that damage to the central nervous system resulting in learning problems can occur at blood lead levels less than 10 $\mu\text{g}/\text{dl}$. About 2.2 percent of children in the U.S. have blood lead levels greater than 10 $\mu\text{g}/\text{dl}$. Lead poisoning can affect almost every system of the body and often occurs with no obvious or distinctive symptoms. Depending on the amount of exposure a child has, lead can cause behavior and learning problems, central nervous system damage, kidney damage, reduced growth, hearing impairment, and anemia [21].

In adults, lead can cause health problems such as high blood pressure, kidney damage, nerve disorders, memory and concentration problems, difficulties during pregnancy, digestive problems, and pain in the muscles and joints [21]. These have usually been associated with blood lead levels greater than 30 $\mu\text{g}/\text{dl}$.

Because of chemical similarities to calcium, lead can be stored in bone for many years. Even after exposure to environmental lead has been reduced, lead stored in bone can be released back into the blood where it can have harmful effects. Normally, this release occurs relatively slowly. However, certain conditions such as pregnancy, lactation, menopause, and hyperthyroidism can cause more rapid release of the lead, which could lead to a significant rise in blood lead levels [22].

Manganese

Manganese is a naturally occurring metal that is found in many types of rocks. Background soil manganese concentrations in Eastern Washington range from about 233 to 769 ppm [11]. It is an essential trace element, is necessary for good health, and can be found in several food items including grains, cereals, and tea. Manganese is an essential trace element and is required by the body to break down amino acids, and produce energy. Incidental ingestion of soil containing manganese can result in increased manganese in the body; however, most manganese is excreted in feces. Only about 3 to 5% of manganese ingested is absorbed [23]. Manganism (mental and emotional disturbances or body movements that are slow and clumsy) is a condition that is typically the result of inhaling high levels of manganese dust in the air. It is uncertain whether eating or drinking too much manganese can cause symptoms of manganism. The current EPA established RfD in IRIS for food manganese is 0.14 mg/kg/day. EPA Regions 3 and 9 used a modifying factor of six to the RfD for food manganese to establish a RfD for environmental manganese of 0.024 mg/kg/day. However, ATSDR used a modifying factor of three for a minimum risk levels (MRL) of 0.05 mg/kg/day, which is basis for the RMEG of 3000 mg/kg.

Zinc

Zinc is a naturally occurring element in the earth's soil. Background soil zinc concentrations in Eastern Washington range from about 26 to 82 ppm [11]. Zinc compounds are used as ingredients in many common products such as vitamin supplements, sun blocks, diaper rash ointments, deodorants, athlete's foot preparations, acne and poison ivy preparations, and antidandruff shampoos [24]. Ingesting high levels of zinc for short periods may cause stomach cramps, nausea, and vomiting. Ingesting high levels of zinc for long periods may cause anemia, damage the pancreas, and decrease levels of high-density lipoprotein (HDL) cholesterol [24]. The EPA established RfD for zinc is 0.3 mg/kg/day.

Evaluating Non-cancer Hazards

Exposure assumptions for estimating contaminant doses from sediment exposure are found in Appendix B, Table B1. In order to evaluate the potential for non-cancer adverse health effects that may result from exposure to contaminated media (i.e., air, water, soil, and sediment), a dose is estimated for each contaminant of concern. These doses are calculated for situations (scenarios) in which area residents or vacationers might be exposed to the contaminated media. The estimated dose for each contaminant under each scenario is then compared to EPA's oral reference dose (RfD). RfDs are doses below which non-cancer adverse health effects are not expected to occur (considered "safe" doses). They are derived from toxic effect levels obtained from human population and laboratory animal studies. These toxic effect levels can be either the lowest-observed adverse effect level (LOAEL) or a no-observed adverse effect level (NOAEL). In human or animal studies, the LOAEL is the lowest dose at which an adverse health effect is seen, while the NOAEL is the highest dose that did not result in any adverse health effects.

Because of uncertainty in these data, the toxic effect level is divided by "safety factors" to produce the lower and more protective RfD. If a dose exceeds the RfD, this indicates only the potential for adverse health effects. The magnitude of this potential can be inferred from the degree to which this value is exceeded. If the estimated exposure dose is only slightly above the RfD, then that dose will fall well below the observed toxic effect level. The higher the estimated dose is above the RfD, the closer it will be to the actual observed toxic effect level. This comparison is called a hazard quotient (HQ) and is given by the equation below:

$$HQ = \frac{\text{Estimated Dose (mg/kg-day)}}{\text{RfD (mg/kg-day)}}$$

Estimated exposure doses, exposure assumptions, and hazard quotients are presented in Appendix B for COCs found in sediments including arsenic, antimony, cadmium, copper, iron, manganese and zinc. *Estimated doses from exposure to contaminants in sediments along Lake Roosevelt beaches do not result in hazard quotients in excess of one for any chemical.* However, as mentioned above, if the estimated exposure dose is only slightly above the RfD, then that dose will fall well below the toxic effect level. The higher the estimated dose is above the RfD, the closer it will be to the actual toxic effect level. In addition, the maximum concentration is a conservative or high estimate of beach sediment concentration. Therefore, this indicates that non-

cancer adverse health effects are not likely to result from exposure to these COCs (lead is not discussed here) in sediment at the beaches.

The maximum concentration of arsenic and lead was found in sediment at elevation 1274.6 feet above mean sea level (ft). During the period from 1996 to 2006, Lake Roosevelt pool averages 127 days below elevation 1274.6 ft. The days below elevation 1274.6 ft occurred only during the winter and spring months of December to June. Therefore, a conservative exposure scenario of 120 days was used in the health consultation evaluation (See Appendix C).

Evaluating exposure to lead

The biokinetics of lead are different from most toxicants because it is stored in bone and remains in the body long after it is ingested. Children's exposure to lead is evaluated through the use of the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) developed by the EPA. The IEUBK predicts blood lead levels in a distribution of exposed children based on the amount of lead that is in the environmental media (e.g. sediment) [25]. It is important to note that the IEUBK model is not expected to accurately predict the blood lead level of an individual child (or a small group of children) at a specific point in time. In part, this is because an individual (or group of children) may behave differently, and therefore have different amounts of exposure to contaminated soil and dust than the average group of children used by the model to calculate blood lead levels. For example, the model does not take into account reductions in exposure that could result from community education programs. Despite this limitation, the IEUBK model is a useful tool to help prevent lead poisoning because of the information it can provide about the hazards of environmental lead exposure. For children who are regularly exposed to lead-contaminated soil, the IEUBK model can estimate the percentage of young children who are likely to have blood lead concentrations that exceed a level that may be associated with health problems (usually 10 µg/dl). The EPA also has an adult lead model used to predict adolescents and adults blood lead. However, only the IEUBK model will be used in the evaluation of lead because children are the most susceptible population to lead.

Sediment lead concentrations and estimated blood lead levels

The IEUBK model was used to estimate the percentage of children that could have elevated blood lead levels if they play frequently in areas with lead contamination and exhibit typical behaviors that result in ingestion of sediment. The maximum sediment lead concentration (535 ppm) was used as a screening value to estimate children's exposure to lead in beach sediments. The recreational and trespassing exposure scenario, which employs the additional soil ingestion (contact-intensive scenario) and some aspect of the time-weighted average approach (excluding the waking hour's part), was used in this analysis [26]. This assumes sediment ingestion may be greater than the default levels for children 0 to 84 months old (Table 3). It is assumed that the ingestion rate is based on 200 mg per day due to actual soil ingestion studies in children using trace elements [27, 28, 29, 30]. The age group ingestion works out to be the values in the IEUBK model defaults (Table 3).

Table 3: IEUBK soil/dust ingestion defaults by age.

Age Group (years)	IEUBK Model Defaults (g/day)
0-1	0.085
1-2	0.135
2-3	0.135
3-4	0.135
4-5	0.100
5-6	0.090
6-7	0.085

For exposures at beaches, children are assumed to potentially ingest greater amounts of sediment than they would at home; consequently, the sediment ingestion rate selected for this health consultation is 300 mg/day, rather than 200 mg/day [30]. This works out to be the default value plus 100 mg per day for all age groups except 0 – 1 years old, since they are not likely to have significant additional exposure to the beaches (Table 4).

Table 4: Total soil/dust ingestion 100 mg/day plus default value by age.

Age Group (years)	Total = 0.100 g/day + Default
0-1	0.085
1-2	0.235
2-3	0.235
3-4	0.235
4-5	0.200
5-6	0.190
6-7	0.185

There are several other assumptions being made in running the IEUBK model:

1. 30 percent of the exposure is from the beach.
2. 70 percent of the exposure is from the residential default of 100 ppm soil lead.
3. All other model inputs were default values.

The calculated weighted sediment lead concentration (PbSw - 231 ppm) and weighted dust lead concentration (PbDw - 161 ppm) are then used in the model based on the screening scenario (see Appendix D - Lead Exposure scenario used in the IEUBK model - win Version 1.0 build 255). Based on this scenario, the model indicates that about 7.2 % will have blood lead levels greater than 10 µg/dl and predicts a geometric mean blood lead level of 5.0 µg/dl for children ages seven years and under. However, this result is based on the maximum level found at the Northport beach and not on the average concentration (at Northport beach) as the model requires. Therefore, this would result in an overestimation of blood lead levels.

A more realistic exposure approach would be to use the average concentration (250 ppm at Northport beach) of sediment lead in the IEUBK model. This exposure scenario for a child yielded a calculated PbSw of 145 ppm and a calculated PbDw of 102 ppm for use in the model (see Appendix D - Lead Exposure scenario used in the IEUBK model - win Version 1.0 build 255). Based on this scenario, the model indicates that about 1.9 % will have blood lead levels greater than 10 µg/dl and predicts a geometric mean blood lead level of 3.8 µg/dl for children ages seven years and under.

The exposure assumption for vacationers was two weeks per year. However, since a vacationer would spend such a short time in the area, it is highly unlikely that the current concentration of contaminants would result in acute toxicity for vacationers.

Multiple Chemical Exposures

A person can be exposed to more than one chemical through more than one pathway. Exposure to a chemical through multiple pathways occurs if a contaminant is present in more than one medium (i.e., air, soil, surface water, groundwater, and sediment). For example, the dose of a contaminant received from drinking water might be combined with the dose received from contact with the same contaminant in soil.

For many chemicals, much information is available on how the individual chemical produces effects. However, it is much more difficult to assess exposure to multiple chemicals. Due to the large number of chemicals in the environment, it is impossible to measure all of the possible interactions between these chemicals. The potential exists for these chemicals to interact in the body and increase or decrease the potential for adverse health effects. Individual cancer risk estimates can be added since they are measures of probability. However, when estimating non-cancer risk, similarities must exist between the chemicals if the doses are to be added. Groups of chemicals that have similar toxic effects can be added, such as volatile organic compounds (VOCs) which cause liver toxicity. Polycyclic aromatic hydrocarbons (PAHs) are another group of compounds that can be assessed as one combined dose based on similarities in chemical structure and metabolites.

The ATSDR Interaction Profile for arsenic, cadmium, chromium, and lead evaluates the possibility of interactive effects from exposure to several metals including arsenic and lead. It states that if the combined exposure to arsenic and lead are high enough, there might be a greater potential for causing neurological effects than exposure to lead or arsenic alone [31]. However, the gastrointestinal absorption of lead and sensitivity to its effects are affected by the adequacy of essential metals such as calcium, zinc, iron, selenium, and other nutrients in the diet. This interdependence (gastrointestinal absorption of lead and sensitivity to its effects) seems to be true for zinc and copper, and may also hold true for manganese [32]. In general, from scientific literature related to chemical interactions, if the estimated exposure doses for individual contaminants are well below doses shown to cause adverse effects, then the combined effect of multiple chemicals is not expected to result in adverse health effects. Although some chemicals can interact to cause a toxic effect that is greater than the added effect, there is little evidence demonstrating this at concentrations commonly found in the environment. Therefore,

combined exposures to COCs at Lake Roosevelt beaches (arsenic, antimony, cadmium, copper, iron, lead, manganese, and zinc) are not expected to result in adverse health effects.

Evaluating Theoretical Cancer Risk

Some chemicals have the ability to cause cancer. Theoretical cancer risk is estimated by calculating a dose similar to that described above and multiplying it by a cancer potency factor, also known as the cancer slope factor. Some cancer potency factors are derived from human population data. Others are derived from laboratory animal studies involving doses much higher than are encountered in the environment. Use of animal data requires extrapolation of the cancer potency obtained from these high dose studies down to real-world exposures. This process involves much uncertainty.

Current regulatory practice suggests there is no “safe dose” of a carcinogen and that a very small dose of a carcinogen will result in a very small cancer risk. Theoretical cancer risk estimates are, therefore, not yes/no answers but measures of chance (probability). Such measures, however uncertain, are useful in determining the magnitude of a cancer threat because any level of a carcinogenic contaminant carries an associated risk. The validity of the “no safe dose” assumption for all cancer-causing chemicals is not clear. Some evidence suggests that certain chemicals considered carcinogenic must exceed a threshold of tolerance before initiating cancer. For such chemicals, risk estimates are not appropriate. More recent guidelines on cancer risk from EPA reflect the potential that thresholds for some carcinogenesis exist. However, EPA still assumes no threshold unless sufficient data indicate otherwise [33].

This document describes theoretical cancer risk that is attributable to site-related contaminants in qualitative terms like low, very low, slight, and no significant increase in theoretical cancer risk. These terms can be better understood by considering the population size required for such an estimate to result in a single cancer case. For example, a low increase in cancer risk indicates an estimate in the range of one cancer case per ten thousand persons exposed over a lifetime. A very low estimate might result in one cancer case per several tens of thousands exposed over a lifetime and a slight estimate would require an exposed population of several hundreds of thousands to result in a single case. DOH considers cancer risk insignificant when the estimate results in less than one cancer per one million exposed over a lifetime. The reader should note that these estimates are for excess cancers that might result in addition to those normally expected in an unexposed population.

Theoretical Cancer Risk		
Theoretical cancer risk estimates do not reach zero no matter how low the level of exposure to a carcinogen. Terms used to describe this risk are defined below as the number of excess cancers expected in a lifetime:		
<u>Term</u>		<u># of Excess Cancers</u>
low	is approximately equal to	1 in 10,000
very low	is approximately equal to	1 in 100,000
slight	is approximately equal to	1 in 1,000,000
insignificant	is less than	1 in 1,000,000

Cancer is a common illness and its occurrence in a population increases with age. Depending on the type of cancer, a population with no known environmental exposure could be expected to

have a substantial number of cancer cases. There are many different forms of cancer that result from a variety of causes; not all are fatal. Approximately 1/4 to 1/3 of people living in the United States will develop cancer at some point in their lives [34].

Theoretical cancer risk from exposure to sediments was calculated for arsenic only as no other carcinogenic COC was identified in sediments (see Appendix B - Table B4 and B5 and Appendix C - Table C3). The recent EPA IRIS review draft presented a slope factor for combined lung and bladder cancer of 5.7 per mg/kg-day [14]. The slope factor calculated from the work by the National Research Council is about 21 per mg/kg/day [15]. These slope factors could be higher if the combined risk for all arsenic-associated cancers (bladder, lung, skin, kidney, liver, etc.) were evaluated. For this health consultation, DOH used a slope factor of 5.7 per mg/kg-day which appears to reflect EPA's most recent assessment.

The lifetime increase of theoretical cancer risk associated with exposure to arsenic found in sediments at the maximum concentration is low to very low (2×10^{-5} or 2 in 100,000) for a 14-day vacationer scenario and low to very low (4×10^{-5} or 4 in 100,000) for a 35-day area resident scenario. Also, low (1×10^{-4} or 1 in 10,000) for a 120-day area resident scenario, which is an overestimation of theoretical cancer risk. The legacy of arsenic and lead in the state of Washington was addressed by the Area Wide Task Force which provided recommendations for action to the Departments of Ecology and Health [35].

Children's Health Concerns

The potential for exposure and subsequent adverse health effects often increases for younger children compared with older children or adults. ATSDR and DOH recognize that children are susceptible to developmental toxicity that can occur at levels much lower than those causing other types of toxicity. The following factors contribute to this vulnerability:

- Children are more likely to play outdoors in contaminated areas by disregarding signs and wandering onto restricted locations.
- Children often bring food into contaminated areas resulting in hand-to-mouth activities.
- Children are smaller and receive higher doses of lead exposure per body weight.
- Children are shorter than adults; therefore, they have a higher possibility of breathing in dust and soil.
- Fetal and child exposure to contaminants such as lead can cause permanent damage during critical growth stages.

These unique vulnerabilities of infants and children demand special attention in communities that have contamination of their water, food, soil, or air. Children's health was considered in the writing of this health consultation and the exposure scenarios treated children as the most sensitive population being exposed.

Conclusions

Based on the information provided, DOH concludes the following:

1. DOH concludes that touching, breathing or accidentally eating sediment 35-days-per-year (two-days-per-week for four months) area resident's exposure scenario from Lake Roosevelt beaches is not expected to harm people's health. The maximum levels of contaminants of concern in this exposure scenario are below levels known to result in non-cancer harmful health effects. In addition, the exposure scenario does not present an elevated theoretical cancer risk.
2. DOH concludes that touching, breathing or accidentally eating sediment 14-days-per-year (2 weeks per year) vacationer exposure scenario from Lake Roosevelt beaches is not expected to harm people's health. The maximum levels of contaminants of concern in this exposure scenario are below levels known to result in non-cancer harmful health effects. In addition, the exposure scenario does not present an elevated theoretical cancer risk.
3. DOH concludes that human health effects from breathing fugitive dust from Lake Roosevelt beaches are unknown.

Recommendations

1. Since the screening level based on the maximum concentration for lead exceeded the 5 percent threshold for blood lead levels and the risk for arsenic falls within the low to very low range for 120 days of exposure. DOH recommends that EPA require additional beach sediments sampling at Black sand, Northport boat ramp, and Marcus Island campground beaches in order to obtain a statistically valid number to use in the model as the central tendencies.
2. Although touching, breathing or accidentally eating contaminated sediment from Lake Roosevelt beaches is not likely to harm health, people may be exposed to these contaminants through other pathways unrelated to Lake Roosevelt (e.g. food and drinking water). Since some of these contaminants are found above background and screening levels, DOH recommends that people take steps to reduce exposure (washing hands after playing at the beach and children avoid putting beach sand in their mouth). Therefore, efforts should be made to educate residents of the hazards posed by exposure (specifically to lead and arsenic in the sediments).

Public Health Action Plan

Actions Planned

1. DOH will hold a series of availability sessions around Lake Roosevelt to address community health concerns related to exposure to Lake Roosevelt sediment, fish, and water.
2. DOH will provide fact sheets to communities indicating ways to reduce exposure to contaminants in beach sediments.
3. DOH will coordinate with EPA's RI/FS work in developing health messages.
4. DOH will explore the feasibility and appropriateness of signs posted at Lake Roosevelt beaches.
5. Currently, DOH is working on a health consultation for fish caught in Lake Roosevelt.
6. Community repositories for the public health consultation and related fact sheets will be established at the following:
 - Northport: Northport Town Hall, 315 Summit St., (509) 732-4450
 - Colville: Colville Public Library, 195 S. Oak Street, (509) 684-6620
 - Inchelium: Inchelium Tribal Resource Center, 12 Community Loop, (509) 634-2791
 - Nespalem: Office of Environmental Trust, Bldg. #2, Colville Confederated Tribes, 1 Colville, (509) 634-2413
 - Grand Coulee: Grand Coulee Library, 225 Federal Street, (509) 633-0972
 - Wellpinit: Spokane Tribe Department of Natural Resources, 6290 B Ford-Wellpinit Road, (509) 258-7709 ext. 13
 - Spokane: Spokane Library, 906 W. Main, (509) 444-5336

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References

1. U.S. Geological Survey (USGS). 1994. *Sediment Quality Assessment of Franklin Roosevelt Lake and the Upstream Reach of the Columbia River*. October 1992 investigation. October 1994.
2. CH2M HILL. 2004b. *Draft Phase I Sediment Sampling Approach and Rationale – Upper Columbia River Site CERCLA RI/FS*. Prepared for USEPA Region 10, Seattle, WA. December 10, 2004.
3. Ecology and Environment (E&E). 2003. *Upper Columbia River Expanded Site Inspection Report, Northeast Washington*, prepared for U.S. Environmental Protection Agency.
4. U.S. Geological Survey (USGS). 2000. Contaminant Trends in Sport Fish From Lake Roosevelt and the Upper Columbia River, Washington, 1994-1998. Water-Resources Investigations Report 00-4024. U.S. Geological Survey, Water Resources Division, Tacoma, WA.
5. U.S. Geological Survey (USGS). 2005. Vertical Distribution of Trace Element Concentrations and Occurrence of Metallurgical Slag Particles in Accumulated Bed Sediments of Lake Roosevelt, Washington, September 2002.
6. Washington State Department of Ecology (Ecology). 1997. Polychlorinated Dibenzo-P-Dioxins and Dibenzofurans in Upper Columbia River Suspended Particulate Matter, 1990-1994.
7. Patrick, Glen, December 1993, Washington State Department of Health, Aquatic Toxicology Program Manager, Office of Toxic Substances, Environmental Health Programs, Cominco Slag in Lake Roosevelt: Review of Current Data.
8. Washington State Department of Ecology (Ecology), December 2001, Lake Roosevelt Sediment Toxicity Reassessment Report, Publication No. 01-03-043.
9. Ecology and Environment (E&E). 2000. Upper Columbia River Mines and Mills Preliminary Assessments and Site Inspections Report, Stevens County, Washington, prepared for the U.S. Environmental Protection Agency.
10. Majewski, M.S., Kahle, S.C., Ebbert, J.C. & Josberger, E.G. (2003). Concentrations and Distribution of Slag-Related Trace Elements and Mercury in Fine-Grained Beach and Bed Sediments of Lake Roosevelt, Washington, April-May 2001 pp. 29. U.S. Geological Survey: Tacoma, WA. U.S. Geological Survey Water-Resources Investigations Report 03-4170. <http://water.usgs.gov/pubs/wri/wri034170/>

11. Toxics Cleanup Program, Department of Ecology: Natural background soil metals concentrations in Washington State Publication No. 94-115. Olympia: Washington State Department of Ecology: October 1994.
12. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for antimony PB/93/110641/AS. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. December 1992.
13. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for arsenic (update) PB/2000/108021. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. September 2005.
14. U.S. Environmental Protection Agency. Toxicological Review of Inorganic Arsenic: In support of summary information on the Integrated Risk Information System, July 2005. http://www.epa.gov/waterscience/criteria/arsenic/sab/AsDraft_SAB.pdf.
15. NAS. 2001b. Arsenic in Drinking Water: 2001 Update. National Academy Press. Washington, DC. 2001. Available from URL: <http://books.nap.edu/books/0309076293/html/index.html>.
16. Agency for Toxic Substances and Disease Registry (ATSDR). 2008. Toxicological profile for Cadmium (*Draft for Public Comment*). Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service.
17. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for copper PB 2004-10733. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. September 2004.
18. U.S. Environmental Protection Agency. Air quality criteria for lead, Volume I. National Center for Environmental Assessment, Superfund Health Risk Technical Support Center: Cincinnati, OH.
19. U.S. EPA. Air Quality Criteria for Lead Volume I. U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-5/144aF, October 2006.
20. CDC. Preventing lead poisoning in young children: a statement by the Centers for Disease Control, October 1991. Atlanta, Georgia: U.S. Department of Health and Human Services, Public Health Service, CDC. 1991.
21. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry: Toxicological profile for Lead (update) PB/99/166704. Atlanta: U.S. Department of Health and Human Services. July 1999.
22. Agency for Toxic Substances and Disease Registry (ATSDR). Lead Toxicity (Case studies in environmental medicine course) SS3059. Atlanta: U.S. Department of Health and Human Services, Public Health Service. October 2000.

23. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for manganese (update) PB/2000/108025. U.S. Department of Health and Human Services; Atlanta, Georgia. September 2000.
24. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological profile for zinc (update) PB2006-100008. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service. August 2005.
25. U.S. Environmental Protection Agency. Technical Review Workgroup for Lead. *User's Guide for the Integrated Exposure Uptake Biokinetic Model for Lead in Children, (IEUBK) Windows version 1.0*, OSWER Directive No.9285.7-42. Document No. EPA 540-K-01-005 Washington, DC: May 2002.
26. U.S. Environmental Protection Agency. 2003. Assessing intermittent or variable exposures at lead sites. OSWER Directive 9285.7-76. EPA-540-R-03-008. November. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response, Washington, DC.
27. Calabrese, E., Stanek, E.J., Gilbert, C.E. 1991. Evidence of soil pica behavior and quantification of soil ingested. *Human Experi. Toxicol.* 10:245–249.
28. Clausing, P., Brunekreef, B., van Wijnen. J.H. 1987. A method for estimating soil ingestion by children. *Int. Arch. Occup. Environ. Health* 59:73–82.
29. Davis, S., Waller, P., Buschbom, R., Ballou, J., White, P. 1990. Quantitative estimates of soil ingestion in normal children between the ages of 2 and 7 years: population-based estimates using aluminum, silicon, and Titanium as Soil Tracer Elements. *Arch. Environ. Health* 45(2):112–122.
30. van Wijnen, J. H., Clausing, P., and Brunekreef, B. 1990. Estimated soil ingestion by children. *Environ Res.* 51:147-162.
31. Agency for Toxic Substances and Disease Registry (ATSDR). Interaction Profile for: Arsenic, Cadmium, Chromium, and Lead. U.S. Department of Health and Human Services, Public Health Service. May 2004.
32. Agency for Toxic Substances and Disease Registry (ATSDR). Interaction Profile for: Lead, Manganese, Zinc, and Copper. U.S. Department of Health and Human Services, Public Health Service. May 2004.
33. U.S. Environmental Protection Agency. Guidelines for Carcinogen Risk Assessment (Review Draft). NCEA-F-0644 July 1999. Available at internet: <http://www.epa.gov/NCEA/raf/cancer.htm>.

34. ATSDR Agency for Toxic Substances and Disease Registry. ATSDR Fact Sheet: Cancer. Updated August 30, 2002. Atlanta: U.S. Department of Health and Human Services. Available at internet: <http://www.atsdr.cdc.gov/COM/cancer-fs.html>.
35. Area-wide soil contamination Task Force Report: June 30, 2003. Available at internet: http://www.ecy.wa.gov/programs/tcp/area_wide/Final-Report/index.htm.
36. National Center for Environmental Assessment. Exposure Factors Handbook Volume 1 – General Factors EPA/600/P-95/002Fa: U.S. Environmental Protection Agency; August 1997.
37. Kissel, J.C., Richter, K.Y. & Fenske, R.A. (1996). Factors affecting soil adherence to skin in hand-press trials. *Bull Environ Contam Toxicol*, **56**, 722-8.
38. Cowherd, C., G. Muleski, P. Engelhart, and D. Gillete. 1985. Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination. EPA/600/8-85/002. Prepared for EPA Office of Health and Environmental Assessment, Washington, D.C.
39. U.S. Food and Drug Administration. 1997. Preventing iron poisoning in children. FDA backgrounder. January 15, 1997. Available at: <http://www.cfsan.fda.gov/~dms/bqiron.html>.
40. Institute of Medicine (2001) Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc 2001 National Academy Press Washington, D.C.
41. ATSDR. 2009. Public health assessment guidance manual. Available at: www.atsdr.cdc.gov/HAC/PHAManual/index.html. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, Atlanta, GA.
42. USEPA. 2009. Integrated risk information system (IRIS) home page. Available at: <http://www.epa.gov/iris>. U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment, Washington, DC.
43. USEPA. 2006b. Consumer factsheet on: copper. Available at http://www.epa.gov/safewater/contaminants/dw_contamfs/copper.html. U.S. Environmental Protection Agency, Office of Ground Water and Drinking Water, Washington, DC.
44. Stanek, E.J., and E.J. Calabrese. 2000. Daily soil ingestion estimates for children at a Superfund site. *Risk Anal.* 20(5):627–635.
45. Stanek, E.J., E.J. Calabrese, and M. Zorn. 2001. Biasing factors for simple soil ingestion estimates in mass balance studies of soil ingestion. *Hum. Ecol. Risk Assess.* 7:329–355.

Appendix A - Organic contaminants

Table A1. Maximum detected or non-detected values of organic contaminants in beach sediment along Lake Roosevelt in northeast Washington.

Compounds	Maximum detected or non-detected value (ppm)	Comparison Value (ppm)	EPA Cancer Class	Comparison Value Reference	Contaminant of Concern
2-Methylnaphthalene	0.03	200		RMEG	No
Acenaphthene	0.003	3000		RMEG	No
Acenaphthylene	0.009	2000*	D		No
Anthracene	0.007	20000	D	RMEG	No
Benzo(a)anthracene	0.012	0.62	B2	Region 9	No
Benzo(a)pyrene	0.013	0.1	B2	CREG	No
Benzo(b)fluoranthene	0.009	0.62	B2	Region 9	No
Benzo(ghi)perylene	0.009	2000*	D		No
Benzo(k)fluoranthene	0.007	6.2	B2	Region 9	No
Chrysene	0.017	62	B2	Region 9	No
Dibenz(a,h)anthracene	0.003	0.1**		CREG	No
Dibenzofuran	0.01	290	D	Region 9	No
Fluoranthene	0.036	2000	D	RMEG	No
Fluorene	0.003	2000	D	RMEG	No
Indeno(1,2,3-cd)pyrene	0.011	0.62	B2	Region 9	No
Naphthalene	0.043	30000	C	IM EMEG	No
Phenanthrene	0.041	2000*	D		No
Pyrene	0.036 E	2000	D	RMEG	No
PCB-1016	0.0063 U	3.9		Region 9	No
PCB-1221	0.025 U	0.22		Region 9	No
PCB-1232	0.025 U	0.22		Region 9	No
PCB-1242	0.0063 U	0.22		Region 9	No
PCB-1248	0.0063 U	0.22		Region 9	No
PCB-1254	0.0063 U	0.22		Region 9	No
PCB-1260	0.0063 U	0.22		Region 9	No
2,4'-DDD	0.0051 U	3 ⁺	B2	CREG	No
2,4'-DDE	0.017	2 ⁺⁺	B2	CREG	No
2,4'-DDT	0.057	2 ⁺⁺⁺	B2	CREG	No

4,4'-DDD	0.0021 J	3	B2	CREG	No
4,4'-DDE	0.063	2	B2	CREG	No
4,4'-DDT	0.2 J	2	B2	CREG	No
Aldrin	0.0026 U	0.04	B2	CREG	No
alpha-BHC	0.0026 U	0.1	B2	CREG	No
alpha-Chlordane	0.0026 U	2 ⁺		CREG	No
beta-BHC	0.0026 U	0.4	C	CREG	No
cis-Nonachlor	0.0026 U	2 ⁺		CREG	No
delta-BHC	0.0026 U	0.1 [±]		CREG	No
Dieldrin	0.0051 U	0.04	B2	CREG	No
Endosulfan I	0.0026 U	100 ⁺⁺		EMEG	No
Endosulfan II	0.0051 U	100 ⁺⁺		EMEG	No
Endrin aldehyde	0.0051 U	20 ⁺⁺⁺	D	EMEG	No
Endrin ketone	0.0051 U	20 ⁺⁺⁺	D	EMEG	No
Endrin	0.0051 U	20	D	EMEG	No
gamma-BHC	0.0026 U	0.5	C	IM EMEG	No
gamma-Chlordane	0.0026 U	2 ⁺		CREG	No
Heptachlor epoxide	0.0026 U	0.08	B2	CREG	No
Heptachlor	0.0026 U	0.2	B2	CREG	No
Hexachlorobenzene	0.008	0.4	B2	CREG	No
Hexachlorobutadiene	0.0026 U	9	C	CREG	No
Methoxychlor	0.025 U	8000	C	RMEG	No
Oxychlordane	0.0026 U	2 ⁺		CREG	No
Toxaphene	0.25 U	0.6	B2	CREG	No
trans-Nonachlor	0.0026 U	2 ⁺		CREG	No
1,1'-Biphenyl	0.16 U	3000	D	RMEG	No
1,2,4-Trichlorobenzene	0.16 U	500	D	RMEG	No
1,2-Dichlorobenzene	0.16 U	5000	D	RMEG	No
1,3-Dichlorobenzene	0.16 U	2000	D	IM EMEG	No
1,4-Dichlorobenzene	0.16 U	4000	C	IM EMEG	No
2,2'-oxybis(1-chloropropane)	0.16 U	2000	D	RMEG	No
2,4,5-Trichlorophenol	0.4 U	5000		RMEG	No
2,4,6-Trichlorophenol	0.16 U	60	B2	CREG	No
2,4-Dichlorophenol	0.16 U	200		IM EMEG	No
2,4-Dimethylphenol	0.16 U	1000		RMEG	No
2,4-Dinitrophenol	0.4 U	100		RMEG	No
2,4-Dinitrotoluene	0.16 U	100	B2	EMEG	No

2,6-Dinitrotoluene	0.16 U	200	B2	IM EMEG	No
2-Chloronaphthalene	0.16 U	4000		RMEG	No
2-Chlorophenol	0.16 U	300	D	RMEG	No
2-Methylphenol	0.16 U	3100		Region 9	No
2-Nitroaniline	0.4 U	1.7		Region 9	No
2-Nitrophenol	0.4 U	100 [±]		RMEG	No
3,3'-Dichlorobenzidine	0.16 U	2	B2	CREG	No
3-Nitroaniline	0.4 U	1.7***		Region 9	No
4,6-Dinitro-2-methylphenol	0.4 U	200		IM EMEG	No
4-Bromophenyl-phenylether	0.16 U	NA		NA	No
4-Chloro-3-methylphenol	0.16 U	100 [±]		RMEG	No
4-Chloroaniline	0.16 U	200		RMEG	No
4-Chlorophenylphenyl ether	0.16 U	NA		NA	No
4-Methylphenol	0.16 U	310		Region 9	No
4-Nitroaniline	0.4 U	1.7***		Region 9	No
4-Nitrophenol	0.4 U	100 [±]		RMEG	No
Acetophenone	0.16 U	5000	D	RMEG	No
Atrazine	0.16 U	200		IM EMEG	No
Benzaldehyde	0.16 U	5000		RMEG	No
Benzoic acid	0.22 U	200000		RMEG	No
Benzyl alcohol	0.16 U	18000		Region 9	No
bis(2-Chloroethoxy)methane	0.16 U	NA	D	NA	No
Bis(2-chloroethyl)ether	0.16 U	0.6	B2	CREG	No
Bis(2-ethylhexyl)phthalate	0.16 U	35		Region 9	No
Butyl benzyl phthalate	0.16 U	10000	C	RMEG	No
Caprolactam	0.15	30000	D	RMEG	No
Carbazole	0.16 U	24		Region 9	No
Di-n-butyl phthalate	0.16 U	5000	D	RMEG	No
Di-n-octylphthalate	0.16 U	20000		IM EMEG	No
Diethyl phthalate	0.16 U	300000	D	IM EMEG	No
Dimethyl phthalate	0.16 U	100000	D	Region 9	No
Hexachloroethane	0.16 U	50	C	CREG	No
Isophorone	0.16 U	700	C	CREG	No
N-Nitrosodi-n-propylamine	0.16 U	0.1	B2	CREG	No
N-Nitrosodiphenylamine	0.16 U	9.9	B2	Region 9	No
Nitrobenzene	0.16 U	30	D	RMEG	No

Pentachlorophenol	0.4 U	6	B2	CREG	No
Perchlorocyclopentadiene	0.16 U	300		RMEG	No
Phenol	0.16 U	20000	D	RMEG	No
Total Dioxin TEQ	0.0000046	0.00001	B2		No

CREG - ATSDR's Cancer Risk Evaluation Guide (child)
 RMEG - ATSDR's Reference Dose Media Evaluation Guide (child)
 EMEG - ATSDR's Environmental Media Evaluation Guide (child)
 IM EMEG - ATSDR's Intermediate Environmental Media Evaluation Guide (child)
 J, E - data qualifier: The associated numerical result is an estimate
 U- data qualifier: The analyte was not detected at this level
 B2 - EPA: Probable human carcinogen (inadequate human, sufficient animal studies)
 C - EPA: Possible human carcinogen (no human, limited animal studies)
 D - EPA: Not classifiable as to health carcinogenicity
 Region 9 - EPA: Preliminary Remediation Goals
 * Fluoranthene RMEG value was used as a surrogate
 * * Benzo(a)pyrene CREG value was used as a surrogate
 * * * 2-Nitroaniline Region 9 value was used as a surrogate
 † 4,4'-DDD CREG value was used as a surrogate
 †† 4,4'-DDE CREG value was used as a surrogate
 ††† 4,4'-DDT CREG value was used as a surrogate
 ‡ Chlordane CREG value was used as a surrogate
 †† Endosulfan EMEG value was used as a surrogate
 ††† 2-Nitroaniline Region 9 value was used as a surrogate
 ‡± alpha-BHC CREG value was used as a surrogate
 ‡±± 2,4-Dinitrophenol RMEG value was used as a surrogate
 2,2'-oxybis(1-chloropropane) was formally known as bis(2-chloroisopropyl)ether
 4,6-Dinitro-2-methylphenol also known as 4,6-Dinitro-O-Cresol
 Perchlorocyclopentadiene also known as Hexachloropentadiene
 ppm -parts per million
 NA - Not available

Appendix B - Dose and Cancer Risk Calculations

This section provides calculated exposure doses and assumptions used for exposure to chemicals in sediment along Lake Roosevelt beaches. Three different exposure scenarios were developed to model exposures that might occur. These scenarios were devised to represent exposures to a child (0-5 yrs), an older child, and an adult. The following exposure parameters and dose equations were used to estimate exposure doses from direct contact with chemicals in sediment.

Exposure to chemicals in sediments via ingestion and dermal absorption.

Total dose (non-cancer) = Ingested dose + dermally absorbed dose

Ingestion Route

$$\text{Dose}_{\text{(non-cancer (mg/kg-day))}} = \frac{C \times CF \times IR \times EF \times ED}{BW \times AT_{\text{non-cancer}}}$$

$$\text{Cancer Risk} = \frac{C \times CF \times IR \times EF \times CPF \times ED}{BW \times AT_{\text{cancer}}}$$

Dermal Route

$$\text{Dermal Transfer (DT)} = \frac{C \times AF \times ABS \times AD \times CF}{ORAF}$$

$$\text{Dose}_{\text{(non-cancer (mg/kg-day))}} = \frac{DT \times SA \times EF \times ED}{BW \times AT_{\text{non-cancer}}}$$

$$\text{Cancer Risk} = \frac{DT \times SA \times EF \times CPF \times ED}{BW \times AT_{\text{cancer}}}$$

Inhalation of Particulate from Sediment Route

$$\text{Dose}_{\text{non-cancer (mg/kg-day)}} = \frac{C \times SMF \times IHR \times EF \times ED \times 1/PEF}{BW \times AT_{\text{non-cancer}}}$$

$$\text{Cancer Risk} = \frac{C \times SMF \times IHR \times EF \times ED \times CPF \times 1/PEF}{BW \times AT_{\text{cancer}}}$$

Table B1. Exposure assumptions for exposure to contaminants in sediments samples from beaches along Lake Roosevelt in northeast Washington.

Parameter	Value	Unit	Comments
Concentration (C)	Variable	mg/kg	Maximum detected value
Conversion Factor (CF)	0.000001	kg/mg	Converts contaminant concentration from milligrams (mg) to kilograms (kg)
Ingestion Rate (IR) – adult	100	mg/day	Exposure Factors [36, 37]
Ingestion Rate (IR) – older child	100		
Ingestion Rate (IR) - child	300		
Exposure Frequency (EF)	14	Days/year	Vacationer
	35		Area Resident (two days a week for four months)
Exposure Duration (Ed)	30 (5, 10,15)	years	Number of years at one residence (child, older child, adult yrs).
Body Weight (BW) - adult	72	kg	Adult mean body weight
Body Weight (BW) – older child	41		Older child mean body weight
Body Weight (BW) - child	15		0-5 year-old child average body weight
Surface area (SA) - adult	5700	cm ²	Exposure Factors [36, 37]
Surface area (SA) – older child	2900		
Surface area (SA) - child	2900		
Averaging Time _{non-cancer} (AT)	Variable	days	Equal to Exposure Duration
Averaging Time _{cancer} (AT)	27375	days	75 years
Cancer Potency Factor (CPF)	5.7	mg/kg-day ⁻¹	Source: EPA (Chemical Specific) Arsenic
24 hr. absorption factor (ABS)	0.03	unitless	Source: EPA (Chemical Specific) Arsenic
Oral route adjustment factor (ORAF)	1	unitless	Non-cancer (nc) / cancer (c) - default
Adherence duration (AD)	1	days	Source: EPA
Adherence factor (AF)	0.2	mg/cm ²	Child, older child
	0.07		Adult
Inhalation rate (IHR) - adult	15.2	m ³ /day	Exposure Factors [36, 37]
Inhalation rate (IHR) – older child	14		
Inhalation rate (IHR) – child 0-5	8.3		
Soil matrix factor (SMF)	1	unitless	Non-cancer (nc) / cancer (c) - default
Particulate emission factor (PEF)	6.00E+8	m ³ /kg	Model Parameters 0% grass cover [38]

Sediment Exposure Route–Non-cancer

Table B2. Vacationer beach use scenario hazard calculations resulting from exposure to contaminants in sediments samples from beaches along Lake Roosevelt in northeast Washington.

Contaminants	Max Concentration (ppm)	Scenarios	Estimated Dose (mg/kg/day)			Total Dose	RfD (mg/kg/day)	Hazard quotient
			Incidental Ingestion of Soil	Dermal Contact with Soil	Inhalation of Particulates			
Antimony	53 J	Child	4.07E-5	7.86E-7	9.38E-10	4.15E-5	0.0004	0.104
		Older Child	4.96E-6	2.88E-7	5.79E-10	5.25E-6		0.013
		Adult	2.82E-6	1.13E-7	3.58E-10	2.93E-6		0.007
Arsenic	36	Child	2.76E-5	1.60E-6	1.27E-9	2.92E-5	0.0003	0.097
		Older Child	3.37E-6	5.86E-7	7.86E-10	3.96E-6		0.013
		Adult	1.92E-6	2.30E-7	4.86E-10	2.15E-6		0.007
Cadmium	7.8	Child	5.98E-6	1.16E-7	2.76E-10	6.10E-6	0.001	0.006
		Older Child	7.30E-7	4.23E-8	1.70E-10	7.72E-7		0.0008
		Adult	4.16E-7	1.66E-8	1.05E-10	4.33E-7		0.0004
Copper	3,290	Child	2.52E-3	4.88E-5	1.16E-7	2.57E-3	0.04	0.064
		Older Child	3.08E-4	1.79E-5	7.19E-8	3.26E-4		0.008
		Adult	1.75E-4	6.99E-6	4.44E-8	1.82E-4		0.005
Iron	254,000	Child	1.95E-1	3.77E-3	8.99E-6	1.99E-1	0.7	0.284
		Older Child	2.38E-2	1.38E-3	5.55E-6	2.52E-2		0.036
		Adult	1.35E-2	5.40E-4	3.43E-6	1.40E-2		0.020
Manganese	4,780	Child	3.67E-3	7.09E-5	1.69E-7	3.74E-3	0.024	0.16
		Older Child	4.47E-4	2.59E-5	1.04E-7	4.73E-4		0.02
		Adult	2.55E-4	1.02E-5	6.46E-8	2.65E-4		0.01
Zinc	22,200	Child	1.70E-2	3.29E-4	7.86E-7	1.73E-2	0.3	0.058
		Older Child	2.08E-3	1.20E-4	4.85E-7	2.20E-3		0.007
		Adult	1.18E-3	4.72E-5	3.00E-7	1.23E-3		0.004

ppm -parts per million

Max –maximum

RfD - EPA oral reference dose

J- data qualifier: (reported concentration is an estimated value).

Table B3. Area resident beach use scenario (35 days) hazard calculations resulting from exposure to contaminants in sediments samples from beaches along Lake Roosevelt, in northeast Washington.

Contaminants	Maximum Concentration (ppm)	Scenarios	Estimated Dose (mg/kg/day)			Total Dose	RfD (mg/kg/day)	Hazard quotient
			Incidental Ingestion of Soil	Dermal Contact with Soil	Inhalation of Particulates			
Antimony	53 J	Child	1.02E-4	1.97E-6	2.35E-9	1.04E-4	0.0004	0.260
		Older Child	1.24E-5	7.19E-7	1.45E-9	1.31E-5		0.033
		Adult	7.06E-6	2.82E-7	8.95E-10	7.34E-6		0.018
Arsenic	36	Child	6.90E-5	4.00E-6	3.19E-9	7.30E-5	0.0003	0.243
		Older Child	8.42E-6	1.47E-6	1.97E-9	9.89E-6		0.033
		Adult	4.79E-6	5.74E-7	1.22E-9	5.37E-6		0.018
Cadmium	7.8	Child	1.50E-5	2.89E-7	6.90E-10	1.53E-5	0.001	0.015
		Older Child	1.82E-6	1.06E-7	4.26E-10	1.93E-6		0.0019
		Adult	1.04E-6	4.14E-8	2.63E-10	1.08E-6		0.0011
Copper	3,290	Child	6.31E-3	1.22E-4	2.91E-7	6.43E-3	0.04	0.161
		Older Child	7.69E-4	4.46E-5	1.80E-7	8.13E-4		0.020
		Adult	4.38E-4	1.75E-5	1.11E-7	4.56E-4		0.011
Iron	254,000	Child	4.87E-1	9.42E-3	2.25E-5	4.96E-1	0.7	0.709
		Older Child	5.94E-2	3.45E-3	1.39E-5	6.29E-2		0.089
		Adult	3.38E-2	1.35E-3	8.58E-6	3.52E-2		0.050
Manganese	4,780	Child	9.17E-3	1.77E-4	4.23E-7	9.35E-3	0.024	0.39
		Older Child	1.12E-3	6.48E-5	2.61E-7	1.19E-3		0.05
		Adult	6.37E-4	2.54E-5	1.61E-7	6.63E-4		0.03
Zinc	22,200	Child	4.26E-2	8.23E-4	1.96E-6	4.34E-2	0.3	0.145
		Older Child	5.19E-3	3.01E-4	1.21E-6	5.49E-3		0.018
		Adult	2.96E-3	1.18E-4	7.50E-7	3.08E-3		0.010

ppm -parts per million

RfD - EPA oral reference dose

J- data qualifier: (reported concentration is an estimated value).

Sediment Exposure Route – Cancer

Table B4. Vacationer beach use scenario cancer risk resulting from exposure to arsenic in sediment samples from beaches along Lake Roosevelt, in northeast Washington.

Contaminant	Max Concentration (ppm)	EPA cancer Group	Cancer Potency Factor (mg/kg-day ⁻¹)	Scenarios	Increased Cancer Risk			Total Cancer Risk
					Incidental Ingestion of Soil	Dermal Contact with Soil	Inhalation of Particulates	
Arsenic	36	A	5.7	Child	1.05E-5	6.09E-7	4.84E-10	1.11E-5
				Older child	2.56E-6	4.45E-7	5.98E-10	3.01E-6
				Adult	2.19E-6	2.62E-7	5.54E-10	2.45E-6

ppm - parts per million
 Max – maximum

Lifetime cancer risk: 1.11E-5 + 3.01E-6 + 2.45E-6 = 1.66E-5

Table B5. Area resident beach use scenario (35 days) cancer risk resulting from exposure to arsenic in sediment samples from beaches along Lake Roosevelt, in northeast Washington.

Contaminant	Maximum Concentration (ppm)	EPA cancer Group	Cancer Potency Factor (mg/kg-day ⁻¹)	Scenarios	Increased Cancer Risk			Total Cancer Risk
					Incidental Ingestion of Soil	Dermal Contact with Soil	Inhalation of Particulates	
Arsenic	36	A	5.7	Child	2.62E-5	1.52E-6	1.21E-9	2.77E-5
				Older child	6.40E-6	1.11E-6	1.49E-9	7.51E-6
				Adult	5.47E-6	6.54E-7	1.39E-9	6.13E-6

ppm - parts per million

Lifetime cancer risk: 2.77E-5 + 7.51E-6 + 6.13E-6 = 4.13E-5

Appendix C - Dose and Cancer Risk Calculations

This section provides calculated exposure doses and assumptions used for exposure to chemicals in sediment along Lake Roosevelt beaches for 120 days. Three different exposure scenarios were developed to model exposures that might occur. These scenarios were devised to represent exposures to a child (0-5 yrs), an older child, and an adult. The following exposure parameters and dose equations were used to estimate exposure doses from direct contact with chemicals in sediment.

Exposure to chemicals in sediments via ingestion and dermal absorption.

Total dose (non-cancer) = **Ingested dose + dermally absorbed dose**

Ingestion Route

$$\text{Dose}_{\text{(non-cancer (mg/kg-day))}} = \frac{C \times CF \times IR \times EF \times ED}{BW \times AT_{\text{non-cancer}}}$$

$$\text{Cancer Risk} = \frac{C \times CF \times IR \times EF \times CPF \times ED}{BW \times AT_{\text{cancer}}}$$

Dermal Route

$$\text{Dermal Transfer (DT)} = \frac{C \times AF \times ABS \times AD \times CF}{ORAF}$$

$$\text{Dose}_{\text{(non-cancer (mg/kg-day))}} = \frac{DT \times SA \times EF \times ED}{BW \times AT_{\text{non-cancer}}}$$

$$\text{Cancer Risk} = \frac{DT \times SA \times EF \times CPF \times ED}{BW \times AT_{\text{cancer}}}$$

Inhalation of Particulate from Sediment Route

$$\text{Dose}_{\text{non-cancer (mg/kg-day)}} = \frac{C \times SMF \times IHR \times EF \times ED \times 1/PEF}{BW \times AT_{\text{non-cancer}}}$$

$$\text{Cancer Risk} = \frac{C \times SMF \times IHR \times EF \times ED \times CPF \times 1/PEF}{BW \times AT_{\text{cancer}}}$$

Table C1. Exposure assumptions for exposure to contaminants in sediment samples from beaches along Lake Roosevelt in northeast Washington.

Parameter	Value	Unit	Comments
Concentration (C)	Variable	mg/kg	Maximum detected value
Conversion Factor (CF)	0.000001	kg/mg	Converts contaminant concentration from milligrams (mg) to kilograms (kg)
Ingestion Rate (IR) – adult	100	mg/day	Exposure Factors [36, 37]
Ingestion Rate (IR) – older child	100		
Ingestion Rate (IR) - child	300		
Exposure Frequency (EF)	120	Days/year	Area Resident everyday (four months)
Exposure Duration (Ed)	30 (5, 10,15)	years	Number of years at one residence (child, older child, adult yrs).
Body Weight (BW) - adult	72	kg	Adult mean body weight
Body Weight (BW) – older child	41		Older child mean body weight
Body Weight (BW) - child	15		0-5 year-old child average body weight
Surface area (SA) - adult	5700	cm ²	Exposure Factors [36, 37]
Surface area (SA) – older child	2900		
Surface area (SA) - child	2900		
Averaging Time _{non-cancer} (AT)	Variable	days	Equal to Exposure Duration
Averaging Time _{cancer} (AT)	27375	days	75 years
Cancer Potency Factor (CPF)	5.7	mg/kg-day ⁻¹	Source: EPA (Chemical Specific) Arsenic
24 hr. absorption factor (ABS)	0.03	unitless	Source: EPA (Chemical Specific) Arsenic
Oral route adjustment factor (ORAF)	1	unitless	Non-cancer (nc) / cancer (c) - default
Adherence duration (AD)	1	days	Source: EPA
Adherence factor (AF)	0.2	mg/cm ²	Child, older child
	0.07		Adult
Inhalation rate (IHR) - adult	15.2	m ³ /day	Exposure Factors [36, 37]
Inhalation rate (IHR) – older child	14		
Inhalation rate (IHR) – child 0-5	8.3		
Soil matrix factor (SMF)	1	unitless	Non-cancer (nc) / cancer (c) - default
Particulate emission factor (PEF)	6.00E+8	m ³ /kg	Model Parameters 0% grass cover [38]

Table C2. Area resident beach use scenario (120 days) hazard calculations resulting from exposure to contaminants in sediments samples from beaches along Lake Roosevelt in northeast Washington.

Contaminants	Maximum Concentration (ppm)	Scenarios	Estimated Dose (mg/kg/day)			Total Dose	RfD (mg/kg/day)	Hazard quotient
			Incidental Ingestion of Soil	Dermal Contact with Soil	Inhalation of Particulates			
Antimony	53 J	Child	3.48E-4	6.74E-6	8.04E-9	3.55E-4	0.0004	0.888
		Older Child	4.25E-5	2.46E-6	4.96E-9	4.50E-5		0.112
		Adult	2.42E-5	9.66E-7	3.07E-9	2.52E-5		0.063
Arsenic	36	Child	2.37E-4	1.37E-5	1.09E-8	2.51E-4	0.0003	0.837
		Older Child	2.89E-5	5.02E-6	6.74E-9	3.39E-5		0.113
		Adult	1.64E-5	1.97E-6	4.17E-9	1.84E-5		0.061
Cadmium	7.8	Child	5.13E-5	9.92E-7	2.37E-9	5.23E-5	0.001	0.05
		Older Child	6.25E-6	3.63E-7	1.46E-9	6.61E-6		0.007
		Adult	3.56E-6	1.42E-7	9.03E-10	3.70E-6		0.004
Copper	3,290	Child	2.16E-2	4.18E-4	9.98E-7	2.20E-2	0.04	0.550
		Older Child	2.64E-3	1.53E-4	6.16E-7	2.79E-3		0.070
		Adult	1.50E-3	5.99E-5	3.81E-7	1.56E-3		0.039
Iron	254,000	Child	1.65E+0	3.23E-2	7.71E-5	1.68E+0	0.7	2.40
		Older Child	2.04E-1	1.18E-2	4.76E-5	2.16E-1		0.308
		Adult	1.16E-1	4.63E-3	2.94E-5	1.21E-1		0.173
Manganese	4,780	Child	3.14E-2	6.08E-4	1.45E-6	3.20E-2	0.024	1.33
		Older Child	3.83E-3	2.22E-4	8.95E-7	4.05E-3		0.17
		Adult	2.18E-3	8.71E-5	5.53E-7	2.27E-3		0.09
Zinc	22,200	Child	1.46E-1	2.82E-3	6.74E-6	1.49E-1	0.3	0.496
		Older Child	1.78E-2	1.03E-3	4.16E-6	1.88E-2		0.063
		Adult	1.01E-2	4.04E-4	2.57E-6	1.05E-2		0.035

ppm -parts per million

RfD - EPA oral reference dose

J- data qualifier: (reported concentration is an estimated value)

According to the National Academy of Sciences, the median daily intake of dietary iron is about 11–13 mg/day for children ages 1 to 8 years old [39]. The median daily intake equate to a dose of about 0.73 – 0.87 mg/kg/day for a child. According to the FDA, doses 200 mg or greater per event could poison or kill a child [40]. 200 mg per event (1 event = 1 day) equate to a dose of about 13.3 mg/kg/day for a child. Extreme exposure dose at Lake Roosevelt is about 1.68 mg/kg/day. Non-cancer adverse health effects are not likely to result from exposure to iron in sediment at the beaches.

For manganese, as mentioned earlier, if the estimated exposure dose is only slightly above the RfD, then that dose will fall well below the toxic effect level. The higher the estimated dose is above the RfD, the closer it will be to the actual toxic effect level. In addition, the maximum concentration is a conservative or high estimate of beach sediment concentration. The dose calculated above assumes 100 % absorption. However, most manganese is excreted in feces; only about 3 to 5% of manganese ingested is absorbed. Therefore, non-cancer adverse health effects are not likely to result from exposure to manganese in sediment at the beaches.

Table C3. Area resident beach use scenario (120 days) cancer risk resulting from exposure to arsenic in sediment samples from beaches along Lake Roosevelt in northeast Washington.

Contaminant	Maximum Concentration (ppm)	EPA cancer Group	Cancer Potency Factor (mg/kg-day ⁻¹)	Scenarios	Increased Cancer Risk			Total Cancer Risk
					Incidental Ingestion of Soil	Dermal Contact with Soil	Inhalation of Particulates	
Arsenic	36	A	5.7	Child	9.00E-5	5.22E-6	4.15E-9	9.52E-5
				Older child	2.19E-5	3.82E-6	5.12E-9	2.57E-5
				Adult	1.87E-5	2.24E-6	4.75E-9	2.09E-5

ppm - parts per million

Lifetime cancer risk: $9.52E-5 + 2.57E-5 + 2.09E-5 = 1.42E-4$

The lifetime increase of theoretical cancer risk associated with exposure to arsenic found in sediment at the maximum concentration is low (1.42×10^{-4} or 1 in 10,000) for a 120-day or four month exposure scenario. However, this result is based on the maximum level found at the Northport beach and not on the average concentration. Therefore, this would result in an overestimation of the theoretical cancer risk.

Appendix D - Lead Exposure scenario used in the IEUBK model

This section provides inputs for the IEUBK model. The following inputs to the model were used to account for lead exposures on Lake Roosevelt at the Northport Boat Ramp beach in Northport, Washington where:

PbS_{beach} = Average soil lead concentration at an exposure unit on the site (**Average sediment concentration at Northport was used in the screening scenario**).

PbS_{home} = Average soil lead concentration near home (ppm). (**Default value = 100 mg/kg**)

PbS_w = Weighted sediment lead concentration (ppm). ($PbS_w = [0.3 \times PbS_{beach}] + [0.7 \times PbS_{home}]$)

PbD_w = Weighted dust lead concentration (ppm). ($PbD_w = 0.7 \times PbS_w$)

The weighted lead concentration results based on the average sediment concentration level (250 ppm) - PbS_w 145 mg/kg and PbD_w 102 mg/kg (Table D1). This number was used to run the IEUBK Model.

Table D1. IEUBK parameters used to calculate the weighted sediment lead concentration for children exposed using a contact intensive scenario to sediment at Lake Roosevelt beaches, Washington.

IEUBK input parameters	Values used for Northport Boat Ramp beach	
Derived Weight sediment concentration (PbS_w)		145 mg/kg
Derived Weight dust concentration (PbD_w)		102 mg/kg
PbS_{beach}	250 mg/kg	^a
PbS_{home}	100 mg/kg	^b
Exposure period	365 days	

^a Corresponds to the average sediment lead value.

^b Corresponds to default lead levels (constant value).

IEUBK total soil/dust ingestion rate = 100 mg/day plus default value by age based on 300 mg/day ingestion rate.

Table D2. Risk estimates for the different age ranges of children ages seven and under exposed to the average lead concentration using a contact intensive scenario to sediment at Lake Roosevelt beaches, Washington.

IEUBK Output		
Age range (months)	GM PbB	% > 10 µg/dL
0-84	3.8	1.9
6-12	3.2	0.8
12-24	4.5	4.5
24-36	4.4	4.0
36-48	4.2	3.1
48-60	3.7	1.7
60-72	3.4	1.0
72-84	3.1	0.6

GM PbB: Blood lead geometric mean
 EPA’s target cleanup goal of having no more than 5 % of the community (0-84 months) with BLLs above 10 µg/dL.

The weighted lead concentration results based on the maximum sediment concentration level (535 ppm) - PbS w 231mg/kg and PbD w 161 mg/kg (Table D3). This number was used to run the IEUBK Model.

Table D3. IEUBK parameters used to calculate the weighted sediment lead concentration for children exposed using a contact intensive scenario to sediment at Lake Roosevelt beaches, Washington.

IEUBK input parameters	Values used for Northport Boat Ramp beach	
Derived Weight sediment concentration (PbS W)		231mg/kg
Derived Weight dust concentration (PbD W)		161 mg/kg
PbS _{beach}	535 mg/kg	^a
PbS _{home}	100 mg/kg	^b
Exposure period	365 days	

^a Corresponds to the maximum sediment lead value.
^b Corresponds to default lead levels (constant value).
 IEUBK total soil/dust ingestion rate = 100 mg/day plus default value by age based on 300 mg/day ingestion rate.

Table D4. Risk estimates for the different age ranges of children ages seven and under exposed to the maximum lead concentration using a contact intensive scenario to sediment at Lake Roosevelt beaches, Washington.

IEUBK Output		
Age range (months)	GM PbB	% > 10 $\mu\text{g/dL}$
0-84	5.0	7.2
6-12	5.6	10.6
12-24	6.3	16.0
24-36	5.9	12.8
36-48	5.6	10.7
48-60	4.7	5.2
60-72	4.0	2.5
72-84	3.6	1.5

GM PbB: Blood lead geometric mean
EPA's target cleanup goal of having no more than 5 % of the community (0-84 months) with BLLs above 10 $\mu\text{g/dL}$.

Appendix E - Public Comments

DOH's responses to public comments are as follows in blue:

1. **Editorial Comment for Clarity**—In the third paragraph, DOH states: “About 66 sediment samples were taken from 15 beaches along Lake Roosevelt and analyzed for total metals.” For clarity and accuracy, it is recommended that DOH insert “and organic compounds” at the end of this statement.

A change has been made to the document to incorporate “and organic compounds”.

2. **Incorrect Concentration Data Values Reported in Referenced Tables**—Table 1 of the report and Table 1 of Appendix A include both detected and undetected concentration values. We believe it is misleading to represent maximum detection limits for undetected concentration values as “Maximum concentrations of [inorganic or organic] contaminants detected in beach sediment along Lake Roosevelt in northeast Washington” as is suggested by the titles for these tables. It is suggested that if undetected concentration values, or some surrogate adjustment of such values, are used in DOH's assessment, it should be clearly explained in the text and the tables modified accordingly (i.e., the addition of appropriate data qualifiers). For example, in Table 1, silver was not detected in any of the beach sediment samples, and yet a maximum detected concentration of 1.5 parts per million (ppm) silver without any qualifier was reported.

Changes have been made to the document to reflect U, J, UJ, etc. qualified data. However, this does not change the document's conclusion.

3. In addition and based on our review of the EPA data, it does not appear that values reported in the tables are correct for several of the analytes listed. For example, in Table 1 of the report, the maximum concentration of thallium is reported as 3.8 ppm. This value corresponds to the highest undetected concentration limit reported for EPA's 2005 primary samples. It is missing the data qualifier associated with it in the original data set and should not be reported as a detected value. For thallium, the highest concentration detected in EPA's 2005 samples was 1.3 ppm, not 3.8 ppm.

Changes have been made to the document to reflect U, J, UJ, etc. qualified data. However, this does not change the document's conclusion.

4. In Table 1 of Appendix A, concentrations reported for many of the compounds (e.g., 2-methylnaphthalene) appear to be extracted from EPA's 2005 quality control samples rather than the primary samples. Based on our professional opinion, we recommend that these errors be corrected.

Changes have been made to the document to reflect U, J, UJ, etc. qualified data. However, this does not change the document's conclusion.

5. Given the example errors described above, we recommend that DOH verify data reported in all tables of the health consultation. In addition, information regarding data selection approaches (i.e., treatment of field duplicates vs. primary samples and use/adjustment of undetected concentration values) applied by DOH should be provided in the health consultation to ensure transparency.

Changes have been made to the document to reflect U, J, UJ, etc. qualified data. However, this does not change the document's conclusion.

6. **Adjustment of J Qualified Concentration Data**—Table 1 of the report includes a footnote regarding use of half the reported concentration for J qualified (i.e., “estimated”) data. Based on our experience, this is not a standard data treatment approach and a technical rationale for DOH's adjustment of J qualified data in this way would be beneficial.

A change has been made to the document to reflect J qualified data. However, this does not change the document's conclusion.

7. **Unclear Derivation of Comparison Values Reported in Referenced Tables**—In Tables 1 and 2 of the report, DOH lists comparison values, many of which are referenced as Agency for Toxic Substances and Disease Registry (ATSDR) values. While it appears that these values were derived according to the ATSDR Public Health Guidance Manual, the specific input values used in each derivation (e.g., reference dose, minimum risk level, body weight) are not included in DOH's health consultation [41]. Absent this information, it is difficult to verify the appropriateness of the comparison values used. For example, the comparison value for manganese is reported as 3,000 ppm based on ATSDR's reference dose media evaluation guide (RMEG). According to ATSDR's guidance manual, an RMEG is derived using the chemical specific oral reference dose (RfD), along with a body weight and ingestion rate for children, “unless childhood exposures can be excluded.” The current RfD for manganese is listed in EPA's Integrated Risk Information System (IRIS) as 0.14 mg/kg/day [42]. ATSDR's guidance specifies a child body weight of 10 kg and soil ingestion rate of 200 mg/day. Based on these inputs, the resulting RMEG is 7,000 ppm, not 3,000 ppm. At this RMEG, manganese is not a contaminant of concern. The RfD for manganese reported on p. 14 of the health consultation, 0.024 mg/kg/day, is not correct, but would also not yield an RMEG of 3,000 ppm based on ATSDR's methodology. We recommend that DOH identify the specific assumptions and inputs used to derive comparison values reported in the health consultation.

The current RfD for manganese listed in IRIS of 0.14 mg/kg/day is for food manganese. The 0.024 mg/kg/day RfD is based on EPA Regions 3 and 9 RfD for environmental manganese. A modifying factor of 6 was used to establish the 0.024 mg/kg/day RfD. However, ATSDR used a modifying factor of 3 for a MRL of 0.05 mg/kg/day which is the basis for the RMEG of 3000 mg/kg.

8. The use of subchronic exposure assumptions (i.e., body weight and ingestion rate associated with a 6 year childhood exposure) in conjunction with toxicity values for noncancer effects based on chronic exposure (i.e., greater than 7 years) represents a highly conservative approach. It is suggested that DOH provide text in its health consultation acknowledging the inherent conservatism of this screening approach.

Chronic and sub-chronic exposures are associated with a time period of exposure and not body weight and ingestion rate. For example, exposure less than one year is considered sub-chronic and exposure more than one year is considered chronic. Human health and risk assessment approaches are conservative by nature. DOH's screening approach is a standard approach used in human health and risk assessment. If you are referring to the sub-chronic comparison values such as the Intermediate Environmental Media Evaluation Guide (IM EMEG) for a child, this is based on ATSDR's hierarchy level for health guidelines. When there are not chronic comparison values for a contaminant and there are sub-chronic comparison values, the sub-chronic comparison values are used. The chronic comparison values are more conservative than sub-chronic comparison values.

9. **Suggested Sentence Deletion under Contaminants of Concern**—Regarding the comparison values DOH used to screen contaminants of concern, DOH states: “These types of comparison values often form the basis for cleanup.” This statement is contrary to ATSDR guidance regarding use of minimum risk levels (MRLs), which states: “These substance specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors and other responders to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that MRLs are not intended to define clean up or action levels for ATSDR or other Agencies.” Thus, it is recommended that the statement be deleted or modified accordingly.

The report has been modified to reflect your comment.

10. **Suggested Sentence Revision under Contaminants of Concern**—Reference to contaminants in sediment exceeding health comparison values in DOH's second bulleted exposure pathway is misleading as it suggests screening of these contaminants in air. To reduce confusion introduced by the current text, we suggest replacing this bulleted text with “Inhalation of sediment derived dust particles in air during periods of reservoir drawdown.”

The report has been modified to reflect your comment.

- 11. Suggested Sentence Revision under Beach Play Scenario, Ingestion Exposure**—DOH’s statement, “Most people inadvertently swallow small amounts of sediments, soil and dust (and any contaminants they contain)” is misleading as written. An appropriate alternative would be to state: “People may inadvertently swallow small amounts of sediments, soil and dust (and any contaminants they contain).”

The report has been modified to reflect your comment.

- 12. Suggested Sentence Revision under Fugitive Dust Scenario**—DOH states, “During those periods, the depth of the lake can decrease in excess of 60 feet exposing many square miles of contaminated sediments along the lake.” For accuracy, we suggest inserting the word “potentially” before “...contaminated sediments...” in this sentence.

The report has been modified to reflect your comment.

- 13. Use of Incorrect Cancer Slope Factor for Arsenic**—DOH proposes to use a cancer slope factor (CSF) of 5.7 per mg/kg/day to estimate arsenic cancer risk due to exposure to UCR beach sediments, stating that this CSF “appears to reflect EPA’s most recent assessment,” citing EPA’s 2005 IRIS review draft in support of this selection [14]. Based on our experience, DOH’s reliance on this assessment may be premature. We would like to point out that printed on the cover page to the 2005 review draft, the “Notice” states:

This document is an IRIS review draft for the Science Advisory Board (SAB). It has not been formally released by the U.S. Environmental Protection Agency and should not at this stage be construed to represent Agency position on this chemical. It is being circulated for review of its technical accuracy and science policy implications.

The importance of this disclaimer is highlighted by an advisory report on EPA’s Assessment of Carcinogenic Effects of Organic and Inorganic Arsenic, which was released by the EPA SAB on June 28, 2007. Based on the major findings reported in the SAB’s report, implementation by EPA of the SAB’s recommendations will require substantial EPA effort before a revised dose response analysis can be issued. In the meantime, the current CSF for arsenic, 1.5 per mg/kg/day, as published in IRIS, is recommended. It should also be noted that use of the current IRIS value is consistent with the arsenic CSF proposed for use by EPA in the recent (July 7, 2008) external review draft of the Work Plan for the Human Health Risk Assessment for the Upper Columbia River Site Remedial Investigation and Feasibility Study.

The prevailing scientific evidence indicates the current cancer potency factor of 1.5 per mg/kg/day is too low. The oral slope factor for arsenic of 5.7×10^{-6} is a typographical error and instead should be 5.7 mg/kg/day. Based on the calculation using the arsenic unit risk 1.6×10^{-4} ug/L, times 70 kg body weight, times 1000 (unit conversion) divided by 2 L/day water ingestion, the cancer potency factor is 5.6 mg/kg/day. DOH has already addressed their use of the 5.7 per mg/kg/day slope factor issue with both EPA Region 10 and ATSDR.

- 14. Request Updating Statements Regarding Lead Health Effects Based on Current Information**—DOH cites a 1992 ATSDR analysis paper to support its assertion that “In general, blood lead rises 3-7 µg/dl [microgram per deciliter]) for every 1,000 ppm [parts per million] increases in soil or dust concentration.” A more recent review of the relationship between dust and soil lead and children’s blood lead levels is available in EPA’s Air Quality Criteria Document for Lead [19]. It is recommended that DOH consult this review and update relevant statements accordingly.

The report has been modified to reflect your comment.

- 15. Incorrect Toxicity Value for Manganese**—DOH states: “EPA established RfD for manganese is 0.024 mg/kg/day.” This is not correct. The current RfD for manganese, as listed in IRIS, is 0.14 mg/kg/day [42].

Manganese referenced in this report is environmental manganese. The RfD for manganese, as listed in IRIS, of 0.14 mg/kg/day is for food manganese. The 0.024 mg/kg/day RfD is based on EPA Regions 3 and 9 RfD for environmental manganese. A modifying factor of 6 was used to establish the 0.024 mg/kg/day RfD. However, ATSDR used a modifying factor of 3 to establish their MRL of 0.05 mg/kg/day which is basis for the RMEG of 3000 mg/kg.

- 16. Incorrect Toxicity Value for Copper**—DOH states: “The EPA established RfD for copper is 0.04 mg/kg/day.” This is not correct. Currently and to the best of our knowledge, there is no oral FRS for copper published in IRIS.^a

The RfD of 0.04 mg/kg/day for copper is from Health Effects Assessment Summary Tables (HEAST). The HEAST is an EPA document, which is used as tier three toxicity values. This current RfD value for copper of 0.04 mg/kg/day is used by EPA Regions 3 and 9. The current drinking water standard (MCLG) of 1.3 mg/L is converted to an RfD for chronic and sub-chronic oral exposure.

- 17. Requested Clarification of Approaches and Assumptions Employed by DOH to Evaluate Lead Exposures**—On p. 16 of the health consultation, DOH states: “The contact-intensive scenario as suggested by the Colville Tribes instead of a time-weighted average approach as was previously used in the draft.” It is recommended that the specific approaches DOH employed, as well as the rationale and assumptions associated

^a The value reported by DOH is likely derived from EPA’s maximum contaminant level goal action level for copper in drinking water, 1.3 mg/L. EPA requires water systems to control the corrosiveness of their water if more than 10 percent of the tap water samples indicate that the level of copper at home taps exceeds this action level. As described in EPA’s “Consumer Factsheet on: Copper,” the action level for copper is not based strictly on health protection. It “has also been set at 1.3 ppm because EPA believes, given present technology and resources, this is the lowest level to which water systems can reasonably be required to control this contaminant should it occur in drinking water at their customers’ home taps” [43].

with these, be clearly presented in the current consultation. If information from prior draft consultations is important to understanding their current approaches, DOH should include complete references to such documents.

DOH cannot include complete reference to an agency draft for comment document dated around November 17, 2006. However, for clarity any reference to the “contact-intensive scenario as suggested by the Colville Tribes instead of a time-weighted average approach as was previously used in the draft,” has been removed and the current approach stated.

18. Based on the information provided in DOH’s consultation, it appears that two approaches were employed in the lead exposure evaluation: a contact-intensive approach and a time-weighted approach. Assumptions associated with each approach are not fully described in the consultation. Further information is needed to evaluate the appropriateness of these approaches, used either individually or in combination, to DOH’s assessment and interpretation of results.

The approach used is based on the EPA’s guidance for assessing intermittent lead exposures [26]. In particular, the recreational and trespassing exposure scenario which employs the additional soil ingestion (contact-intensive scenario) and some aspect of the time-weighted average approach (excluding the waking hour’s part) because that will increase the soil ingestion even more.

19. **Appropriateness of Increasing Soil/Dust Ingestion Rate Assumption**— DOH increased the default values for age-dependent soil/dust ingestion rates based on assumed increased contact rates with sediments at beaches relative to residential soils. The basis for changing the Integrated Exposure Uptake Biokinetic (IEUBK) model for lead in children default values for the age-specific total soil/dust ingestion rate inputs should be clearly documented.

The IEUBK model is designed to use central tendency values of all input parameters. The IEUBK model assumes child soil/dust ingestion rates to be 85 mg/day for 0–1 year, 135 mg/day for 1–4 years, 100 mg/day for 4–5 years, 90 mg/day for 5–6 years, and 80 mg/day for 6–7 years, which yields an average of 108 mg/day [25]. However, the model’s default values for age-dependent soil ingestion rates are based on short-term population surveys from older studies. It is of particular concern that such surveys will overestimate the distribution of long-term average daily soil ingestion across a population of children [36]. This issue was recently addressed by Stanek and Calabrese, and Stanek et al., who showed that 95th percentile intake estimates drop substantially when the distribution represents a longer time period [44, 45]. DOH should include these more recent articles in its reference list.

Stanek and Calabrese updated their earlier analyses of studies conducted in Amherst, Massachusetts, and Anaconda, Montana, to provide more reliable estimates of average

daily soil intake rates over longer time periods [44]. This analysis yielded estimated mean soil intakes of 31 and 57 mg/day over a 1-year period, for 1–4 year old children for the Anaconda and Amherst data sets, respectively. One-year average 95th percentiles were 106 and 124 mg/day, respectively. Ninety-day average intakes that may also be relevant for evaluating lead exposures are almost identical to the 1-year averages. Based on this analysis, the default values in the IEUBK model are similar to 95th percentile values for 30–365 day exposure periods, and are not representative of central tendency values. DOH should include discussion of these updated analyses.

Considering the reported mean values of 31 and 57 mg/day from the two studies, the IEUBK model would be more accurate if the default soil ingestion rates were reduced by 50 percent. Given this analysis, the basis for DOH’s use of a 150 percent increased soil/dust ingestion rate should be thoroughly described, particularly because DOH applied the increased contact rates to a weighted exposure medium concentration of lead that was based on both beach sediment and home soil concentrations. Thus, DOH’s approach implies the higher intensity contact rate is occurring both at the beach locations and at home. At a minimum, the contact rate increase should be reduced to account for the proportion of the contact interval occurring at home.

The ingestion rate for children was established by EPA as 300 mg/day for the Upper Columbia River. The IEUBK model default values are central values within the range of observational ingestion values. The contact intensive scenario calls for an addition to the default rates and can be as much as adding 200 mg/day, which is consistent with OSWER guidance on assessing risk for Reasonably Maximally Exposed (REM) individuals [26]. Therefore, 100 mg/day addition was used based on the difference between the 300 mg/day for the Upper Columbia River and EPA’s Exposure Factors Handbook 200 mg/day conservative estimate of the mean.

- 20. Requested Clarification of Impact of Intermittent or Seasonal Exposures on Interpretation of DOH’s Evaluation**— DOH appears to have assumed exposures occurring 2 days per week over an exposure period of 120 days for estimation of cancer and noncancer exposures, including exposures to lead evaluated using the IEUBK model. The IEUBK model is not intended to capture seasonal or intermittent exposures averaged over a period of time less than 1 year, and DOH does not clearly explain what is meant by a “contact-intensive scenario” vs. a “time weighted average approach” (p. 16). We presume that DOH is relying on EPA’s guidance for assessing intermittent lead exposures, but this document is not cited [26]. We suggest that DOH clarify the basis for its evaluation of intermittent or seasonal exposure.

The approach used is based on the EPA’s guidance for assessing intermittent lead exposures [26]. In particular, the recreational and trespassing exposure scenario which employs the additional soil ingestion (contact-intensive scenario) and some aspect of the time-weighted average approach (excluding the waking hour’s part) because that will increase the soil ingestion even more. The assumption is that 30 % of the sediment ingested would be from the site, (about 2 days per week spent at the beach during 120 days exposure period) and 70 % at home. There

are inherent uncertainties and limitations in the IEUBK model. The IEUBK model predictions refer to a full year of exposure regardless of the actual exposure period. Therefore, it can produce results that over-predict actual blood lead levels (BLLs) over the entire year. However, it can also produce results that under-predict actual BLLs during the period of seasonal and successive seasonal exposures. During seasonal and successive seasonal exposures, BLLs can be much higher than the average for the entire year.

- 21. Requested Clarification of Assumptions Regarding Proportion of Exposure Related to Beach vs. Home**— In calculating time-weighted average media concentrations for use in the IEUBK model, DOH assumed that 30 percent of the exposure was from the beach and 70 percent was from the home. The basis for the proportions assigned is not well-described. It appears, based on Table B1 of Appendix B, that the 30% beach exposure assumption is related to 2 days out of 7 days each week spent at the beach during the 120-day exposure period, but it is not clear what portion of the child’s waking time each of these 2 days was assumed to occur at the beach. Such information is important to appropriately assess potential exposures. For example, if only 30% of the child’s waking time was spent at the beach on each of the 2 days per week that beach visits occurred, the 30% assumption for beach exposure could significantly overestimate beach exposure relative to residential soil exposure.

The ingestion rate for children was established by EPA as 300 mg/day for the Upper Columbia River. The recreational and trespassing exposure scenario which employs the additional soil ingestion (contact-intensive scenario) and some aspect of the time-weighted average approach (excluding the waking hour’s part) because that will increase the soil ingestion even more. The assumption is that 30 % of the sediment ingested would be from the site, (about 2 days per week spent at the beach during 120 days exposure period) and 70 % at home.

- 22. Requested Clarification of Model Results’ Interpretation**—The IEUBK model does not predict “daily average” blood lead level for children as stated by DOH in the health consultation (p. 17). DOH should clarify its interpretation of the IEUBK model results as providing estimates of the geometric mean blood lead concentration for children of a given yearly age averaged over that year.

The report has been modified to reflect your comment.

23. Use of Incorrect Cancer Slope Factor for Arsenic

The prevailing scientific evidence indicates the current cancer potency factor of 1.5 per mg/kg/day is too low. The oral slope factor for arsenic of 5.7×10^{-6} is a typographical error and instead should be 5.7 mg/kg/day. Based on the calculation using the arsenic unit risk 1.6×10^{-4} ug/L, times 70 kg body weight, times 1000 (unit conversion) divided by 2 L/day water ingestion, the cancer potency factor is 5.6 mg/kg/day. DOH has already addressed their use of the 5.7 mg/kg/day slope factor issue with both EPA Region 10 and ATSDR.

24. **Reference Dose for Manganese:** The Reference Dose (RfD) used in the risk assessment appears to be incorrect. The value used is 0.024 mg/kg/day. IRIS indicates that the RfD is 0.14 mg/kg/day.

Manganese referenced in this report is environmental manganese. The RfD for manganese, as listed in IRIS, of 0.14 mg/kg/day is for food manganese. The 0.024 mg/kg/day RfD is based on EPA Regions 3 and 9 RfD for environmental manganese. A modifying factor of 6 was used to establish the 0.024 mg/kg/day RfD. However, ATSDR used a modifying factor of 3 to establish their MRL of 0.05 mg/kg/day which is basis for the RMEG of 3000 mg/kg.

25. **Surface Area Values:** The values for the surface area exposed parameter (SA) used in the risk assessment for recreational exposure (e.g., swimming, wading) are not consistent with values proposed in EPA's RAGS Part E. Values used include 2,900 cm² for children and 5,700 cm² for adults. EPA's RAGS Part E recommends using values of 6,600 cm² for children and 18,000 cm² for adults.

EPA's RAGS Part E recommends using values of 6,600 cm² for children and 18,000 cm² for bathing and swimming is for water contact exposures not sediment contact exposures.

26. The third issue was related to the cancer potency factor used by DOH to calculate cancer risks from arsenic exposure. In the previous October 2006 draft a value of 1.5 per mg/kg/day was used, though several other higher values were listed. Our comment on the previous draft pertained to the listing and use of alternative higher values. Specifically, we noted that use of higher values to assess risk under early life and adolescent exposure scenarios as per EPA's Supplemental Cancer Guidelines would be incorrect, because arsenic is not mutagenic and has not been shown to pose greater risk in early life and childhood. In the most recent draft Health Consultation, the authors now use a cancer potency factor of 5.7, apparently taken from a 2005 EPA report titled "Toxicological Review of Inorganic Arsenic: In support of summary information on the Integrated Risk Information System". First, we note that this document is labeled as "draft". Second, the factor as shown in the DOH Health Consultation does not exist in the referenced document (page 65 of the document presents an oral slope factor for arsenic of 5.7×10^{-6} per mg/kg/day). We recommend that the cancer potency factor of 1.5 per mg/kg/day presented in IRIS and used in the October 2006 version of the draft be used.

The prevailing scientific evidence indicates the current cancer potency factor of 1.5 per mg/kg/day is too low. The oral slope factor for arsenic of 5.7×10^{-6} is a typographical error and instead should be 5.7 mg/kg/day. Based on the calculation using the arsenic unit risk 1.6×10^{-4} ug/L, times 70 kg body weight, times 1000 (unit conversion) divided by 2 L/day water ingestion, the cancer potency factor is 5.6 mg/kg/day. DOH has already addressed their use of the 5.7 mg/kg/day slope factor issue with both EPA Region 10 and ATSDR.

27. First, we believe DOH should not present risk assessment results for inhalation of particulates in Appendices B and C if, as noted in the Conclusions section, the fugitive dust pathway will be re-evaluated in the future and represents an “indeterminate health hazard” at present.

The fugitive dust pathway is specific to the windblown dust that occurs only under specific condition noted in that section (USGS five-year monitoring study). It is not dust generated from day to day recreational activities on the beach such as walking, jogging, etc.

28. Second, the 120-day exposure scenario and associated results that appear in Appendix C are not adequately described in the main text of the risk assessment, nor are the implications of the scenario discussed in the Conclusions section.

The 120-day exposure scenario and associated results that appear in Appendix C was not discussed in the Conclusions section because they were based on maximum levels. For example, the maximum level of arsenic at Northport was 36 ppm however; the average of the nine samples was 13.4 ppm. This is below the State clean-up level and would not be evaluated any further. For this reason, DOH recommended EPA to carry out additional sampling to obtain a statistically valid number to use as a central tendency.

29. **Presentation of information:** Currently, technical information about the IEUBK approach to estimating risks due to lead is split between the main text of the Health Consultation and Appendix D. We recommend that this information be consolidated either in the Appendix or included in its entirety in the text.

This approach was suggested by previous reviewers to split the information between the main text and the appendix. Using the appendix to show how the weighted sediment lead concentration was derived.

30. **Calculation of weighted soil concentration:** No justification is given for the assumption that beach sediment would comprise 30 percent of a child’s exposure while residential soils would comprise the remaining 70 percent of soils exposure.

The approach used is based on the EPA’s guidance for assessing intermittent lead exposures. In particular, the recreational and trespassing exposure scenario which employs the additional soil ingestion (contact-intensive scenario) and some aspect of the time-weighted average approach (excluding the waking hour’s part) because that will increase the soil ingestion even more. The assumption is that 30% of soil is ingested at the beach (about 2 days per week spent at the beach during 120 days exposure period) and 70 % from the home. There are inherent uncertainties and limitations in the IEUBK model. The IEUBK model predictions refer to a full year of exposure regardless of the actual exposure period. Therefore, it can produce results that over-predict actual blood lead levels (BLLs) over the entire year. However, it can also produce results that under-predict actual BLLs during the period of seasonal and successive seasonal exposures. During seasonal and successive seasonal exposures, BLLs can be much higher than the average for the entire year.

31. **Calculation of age-specific ingestion rates for sediment:** No justification is given for assuming that children less than one year of age would not exhibit higher ingestion rates, as is assumed for other childhood age groups.

This is recommended in the EPA guidance, “assessing intermittent or variable exposures at lead sites”. Additional soil contact is not applicable to children <1 year old, since they are not likely to have significant additional exposure to site soil.

32. **Use of default values:** The user’s guide for the IEUBK model suggests a default residential soil lead concentration of 200 $\mu\text{g/g}$, not 100 $\mu\text{g/g}$ as was used in the analysis. This would result in a weighted soil concentration of 215 $\mu\text{g/g}$ as opposed to the value of 145 $\mu\text{g/g}$ used in the “average sediment concentration” scenario and a value of 301 $\mu\text{g/g}$ rather than the value of 231 $\mu\text{g/g}$ used in the “maximum sediment concentration” scenario. Justification for this site-specific value should be provided.

The approach used is based on the EPA’s guidance, “assessing intermittent or variable exposures at lead sites,” the recreational and trespassing exposure scenario example.

33. **Calculation of dust lead concentration:** The explanation of the derivation of dust lead concentrations is unclear. It appears that default values were applied in the IEUBK model; however, the relationship between air lead concentrations and dust lead concentrations was not fully explained so the dust lead concentrations used are not replicable.

The approach used is based on the EPA’s guidance “assessing intermittent or variable exposures at lead sites” model input parameter.

34. **Reporting of values:** In the main text, the age group for the reported the percentages of children expected to have blood lead levels in excess of the 10 $\mu\text{g/dl}$ cutoff value is not specified. Tables D-2 and D-4 say the results are for children under age 7; this should be clarified in the text as well.

The report has been modified to reflect your comment.

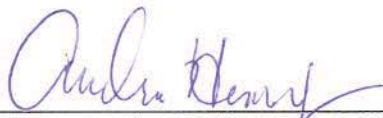
35. **Errors in computing the particle size percentages:** ASTM D-422 procedures do not support use of the hydrometer to determine the colloid fraction. The hydrometer is not a reliable instrument for this use. The error will be at least + or – 25% and likely much larger (personal communication Will Austin, Director of the Soil Science Lab at OSU). A reliable soil analysis lab will not use the hydrometer method. The pipette method is more accurate, reliable, and easier to use. The colloid fraction is determined centrifugally. A modern up-to-date lab will use a laser-diffraction grain size analyzer (notably USGS). The hydrometer will result in an error of + or – 12.5% for clay size fraction in a sample

of 50 gm. The pipette method error will be + or – 0.32%. The laser analyzer will be four times more accurate.

While EPA and its contractor have agreed that the calculation for determining fines percent is incorrect, this aspect of the data was never used in DOH's evaluation of the sediment data. Therefore, this analysis does not apply to any of DOH's evaluation of the data. For laboratory chemical analysis of metals in sediment, the method used was appropriated. Maximum levels were used in all of the evaluation of sediment data except for lead at Northport beach. However, Northport is one of the beaches that DOH recommended for additional sampling to obtain a statistically valid average for the evaluation of the data.

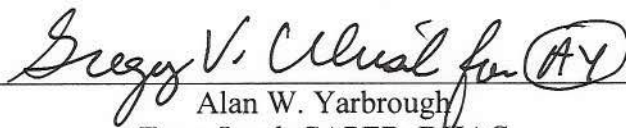
Certification

The Washington State Department of Health prepared this Health Consultation under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It was completed in accordance with approved methodology and procedures existing at the time the health consultation was initiated. Editorial review was completed by the Cooperative Agreement partner.



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The Division of Health Assessment and Consultation, ATSDR, has reviewed this public health consultation and concurs with the findings.



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