Lower Duwamish Waterway Group

Port of Seattle / City of Seattle / King County / The Boeing Company

APPENDIX B PHASE 1 HUMAN HEALTH RISK ASSESSMENT

FINAL

For submittal to

The US Environmental Protection Agency
Region 10
Seattle, WA

The Washington State Department of Ecology
Northwest Regional Office
Bellevue, WA

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List of Acronyms

Acronym	definition
2,3,7,8-TCDD	2,3,7,8-tetrachlorodibenzo-*p*-dioxin
ABS	dermal absorption factor
ANOVA	analysis of variance
API	Asian and Pacific Islanders
ATSDR	Agency for Toxic Substance and Disease Registry
BCEE	Bis(2-chloroethyl) ether
BEHP	Bis(2-ethylhexyl) phthalate
BMD	benchmark dose
bw	body weight
CDI	chronic daily intake
COC	chemical of concern
COPC	chemical of potential concern
cPAH	carcinogenic polycyclic aromatic hydrocarbon
CSO	combined sewer overflow
CT	central tendency
dw	dry weight
EPA	US Environmental Protection Agency
EPC	exposure point concentration
ERA	ecological risk assessment
FC	fraction of dose obtained from site
GC/ECD	gas chromatography / electron capture detector
HEAST	Health Effects Assessment Summary Tables
HHRA	human health risk assessment
HI	hazard index
HPLC/PDA	high performance liquid chromatography / photodiode array detector

Acronym	definition
HQ	hazard quotient
HSDB	Hazardous Substance Data Bank
IEUBK	Integrated Exposure Uptake Biokinetic Model for Lead in Children
IRIS	Integrated Risk Information System
LDW	Lower Duwamish Waterway
LOAEL	lowest observed adverse effects level
MDL	method detection limit
MTCA	Model Toxics Control Act
MVUE	minimum variance unbiased estimate
NCEA	EPA's National Center for Exposure Assessment
NCP	EPA's National Contingency Plan
NOAA	National Oceanic and Atmospheric Administration
NOAEL	no observed adverse effects level
OGWDW	EPA's Office of Ground Water and Drinking Water
PAH	polycyclic aromatic hydrocarbon
PCB	polychlorinated biphenyl
PCT	polychlorinated terphenyl
PSDDA	Puget Sound Dredged Disposal Authority
RBC	risk-based concentration
RCRA	Resource Conservation and Recovery Act
RfD	reference dose
RI	remedial investigation
RME	reasonable maximum exposure
RPF	relative potency factor
SF	slope factor
TBT	tributyltin
TBTO	bis(tri-*n*-butyltin) oxide
TEF	toxic equivalency factor
TEQ	toxic equivalency quotient
UCL	upper confidence limit
WDFW	Washington Department of Fish and Wildlife
WQA	Water Quality Assessment
ww	wet weight

Executive Summary

The Lower Duwamish Waterway (LDW) was added to the US Environmental Protection Agency's (EPA) National Priorities List (NPL, also known as Superfund) on September 13, 2001. Under Superfund regulations, EPA requires that a remedial investigation and feasibility study (RI/FS) be conducted for all listed sites. A Remedial Investigation identifies areas that should be cleaned up because they pose an unacceptable risk to human health or the environment. A Feasibility Study proposes a number of alternative approaches to cleaning up the areas with unacceptable risk, and analyzes and compares these alternatives.

The key parties involved in the Duwamish RI/FS are the City of Seattle, King County, the Port of Seattle, and The Boeing Company, working together for this project as the Lower Duwamish Waterway Group (LDWG), plus EPA and the Washington Department of Ecology. These parties agreed (in an Administrative Order on Consent or AOC) to conduct the RI/FS for the LDW in two phases. Phase 1 is a thorough exploration of what is already known from previous studies of contamination in the LDW, aimed at answering two questions:

1. Based on our understanding of current conditions, are there areas within the LDW that we already know might be candidates for early remediation?
2. What are the data gaps? What more do we need to know to better understand risks to human health and the environment?

Early cleanup is of great interest because the Superfund cleanup process can take many years. This two-phase design will provide preliminary risk estimates to aid in cleaning up those portions of the LDW that pose the highest risks to human health or the environment. A large part of Phase 1 of this RI therefore consisted of a preliminary ecological risk assessment (ERA) and a preliminary human health risk assessment (HHRA).

The Phase 2 RI, which will start in 2003, will include collection of additional data to fill the data gaps identified in Phase 1. The risk assessments done in Phase 1 will be revised in Phase 2 to include the new data. The Phase 2 risk assessments will also estimate risks to human health and the environment left over from the early cleanups.

This appendix describes the HHRA, which includes the data evaluation, conceptual site model and exposure assessment, toxicity assessment, and risk characterization and quantitative uncertainty assessment.

ES.1 DATA EVALUATION

People may be exposed to chemicals found in LDW sediments either through direct exposure to sediment or indirectly through the consumption of fish and shellfish. Accordingly, both tissue and sediment chemistry data are relevant for this HHRA.

Only surface sediment chemistry data were used in the HHRA because people are unlikely to be exposed to deeper sediments. The analysis of sediment fate and transport presented in the RI report (see Section 4.4.2), suggests that erosional events that could remove surface sediment and expose the subsurface sediment are likely to be limited in extent. The likelihood of erosion will be further investigated in Phase 2. Deeper sediments could be contacted during digging in sediments (e.g., clamming) and this issue may need to be further evaluated in Phase 2 if clams are found to be present in harvestable amounts. Tissue chemistry data have been collected for chinook and coho salmon, English sole, crabs, mussels, and perch. Only data from composite samples for crab (edible meat only), English sole filets, perch filets, and mussels were used in this HHRA. Adult salmon data were not utilized in the HHRA because there is unlikely to be a relationship between site-related contamination and chemical concentrations in adult salmon tissue. Salmon feed very little as adults once they enter rivers and streams, and diet is probably the primary exposure pathway to sediment-related chemicals. Because salmon returning to the Duwamish estuary were exposed to site-related chemicals only very briefly as juveniles, the contribution of this exposure to adult body burdens is likely to be insignificant (O'Neill et al. 1998).

ES.2 EXPOSURE ASSESSMENT

The exposure assessment describes potential scenarios in which people may come in contact with chemicals of potential concern (COPCs) associated with sediment within the LDW and provides equations and parameters so that such exposure can be quantified. Direct contact with sediments during commercial netfishing or beach play in the LDW and consumption of seafood from the LDW were identified as primary exposure scenarios through input from site users including the Muckleshoot and Suquamish Tribes, and through review of prior risk assessments of the LDW. Risks associated with surface water contact were considered in this assessment. Specifically, risk estimates for swimming presented in the King County Water Quality Assessment HHRA were summarized and included in the risk characterization section. The King County assessment suggested that risks from these scenarios were well within acceptable levels identified by EPA.[1]

Risk-based screening was conducted using EPA guidance to identify COPCs to be evaluated in the Phase 1 HHRA. Forty-three chemicals were identified as COPCs for one or more scenarios; ten chemicals[2] were identified for all three scenarios. Of these COPCs, 22 were never detected in either sediment or tissue (or both) and were included because detection limits were above risk-based concentrations (RBCs). These COPCs were evaluated in the uncertainty assessment. The exposure assessment

[1] The highest excess cancer risk estimate from incidental ingestion and direct contact with water due to swimming in the LDW was 4E-6 including estimates for both adults and children. All hazard quotients were less than 1 for both adults and children (King County 1999b).

[2] arsenic, benzidine, cadmium, carcinogenic polcyclic aromatic hydrocarbons (PAHs), chromium, copper, dieldrin, lead, N-nitrosodimethylamine, and polychlorinated biphenyls (PCBs)

includes equations to calculate the chemical intake people might experience based on site-specific data for chemical concentrations, exposure frequency, and exposure duration. Values for the commercial netfishing scenario are based on data collected from the Muckleshoot Tribe, which operates a commercial netfishing operation within the LDW. Values for the seafood consumption scenario are based on data collected from the Suquamish Tribe (The Suquamish Tribe 2000), which utilizes the area adjacent to the LDW as part of its usual and accustomed fishing area. Specifically, a consumption rate of 84 grams of seafood per day was assumed based on species representative of resident fishes that may be consumed from the LDW (16 g/day for pelagic species and 15 g/day for benthic species) and on consumption of crabs (45 g/day) and mussels (7.8 g/day). The presence of habitat for crabs and shellfish and their harvestability in the LDW will be further evaluated during the Phase 2 RI, and consumption rates may be modified at that time. Additional seafood consumption data for Asian and Pacific Islanders were also used for quantitative risk estimates. Values for the beach play scenario are based primarily on EPA guidance and on best professional judgment regarding exposure frequencies and durations, since site-specific data for this scenario are not available. Consistent with EPA risk assessment guidance, health protective estimates were selected for all exposure scenarios to avoid underestimating risks. Consequently, risk estimates may be overestimated for many individuals.

Site-specific chemical data are used in the chemical intake equation via a parameter called the exposure point concentration (EPC). The EPC is the assumed concentration for each chemical to which all individuals in a given scenario are exposed over the assumed exposure duration. EPCs for the sediment scenarios (i.e., netfishing and beach play) were calculated for the area over which the exposure was expected to occur. People engaged in commercial netfishing might be exposed to both intertidal and subtidal sediment because their nets may extend across the entire river into the intertidal zone. EPCs for the beach play scenario were based only on intertidal sediment data because children playing on the beach are expected to have little or no exposure to subtidal sediment. EPCs for the seafood consumption scenario were calculated separately for each species in what is known as the market basket approach. This approach utilizes separate chemistry data and consumption rates for each species, such as English sole, perch, and crab. The chemical intakes associated with each species are then summed to yield an overall chemical intake for risk calculations. EPCs for the beach play and seafood consumption scenarios will be revised for the Phase 2 RI following the collection of additional chemistry data.

ES.3 TOXICITY ASSESSMENT

The toxicity of each COPC has been established by EPA through a series of laboratory experiments using animals or epidemiological studies of human populations who were unintentionally exposed in the workplace or in the environment. The toxicity benchmarks are health protective in that they include uncertainty factors or

extrapolations to account for sensitive sub-populations or other limitations of toxicity study data on which they are based.

ES.4 RISK CHARACTERIZATION AND UNCERTAINTY ASSESSMENT

Carcinogenic risks and noncarcinogenic health effects are evaluated separately in HHRAs due to fundamental differences in their critical toxicity values. Carcinogenic risk probabilities are calculated by multiplying the estimated chemical intake by the critical toxicity value (called a slope factor). Cancer risk is expressed as a lifetime excess cancer risk. This concept assumes that the risk of cancer from a given chemical is in "excess" of the background risk of developing cancer (i.e., approximately 1 in 3 chances during a lifetime according to the American Cancer Society). Chemicals with noncarcinogenic health effects are generally not toxic below a certain threshold; a critical chemical dose must be exceeded before health effects are observed. The potential for noncarcinogenic health effects is represented by the ratio of the estimated chemical intake and critical chemical dose (called a reference dose) and is expressed as a hazard quotient.

Cancer risks were highest for the seafood consumption scenario; the cumulative risk for all carcinogenic chemicals was 2E-3, with the primary contributors being arsenic (1E-3), carcinogenic polycyclic aromatic hydrocarbons (cPAHs) (1E-4), and polychlorinated biphenyls (PCBs) (4E-4). Cancer risks for the netfishing scenario and the beach play scenario were much lower (i.e., all risk estimates were less than 1E-5). In evaluation of noncancer risks, arsenic, PCBs, TBT, and mercury all had hazard quotients greater than one, indicating some potential for adverse effects other than cancer. Based on the exposure scenarios evaluated in the Phase 1 HHRA, the following chemicals were identified as chemicals of concern[3] for one or more scenarios: PCBs, arsenic, cPAHs, dioxins/furans (expressed as TCDD toxic equivalents), tributyltin (TBT), and mercury.

There are many uncertainties associated with the risk estimates for each exposure scenario. The reasonable maximum exposure assumptions used in the Phase 1 HHRA will result in high-end estimates of the risks associated with the LDW. To be health protective, these risk estimates are intended to be greater than those faced by most individuals exposed to LDW chemicals. Despite an overall reasonable maximum exposure approach, there are some aspects of the assessment that may underestimate risks. The collection of additional data or performance of additional analyses could reduce many of the uncertainties. Depending on the direction and magnitude of the uncertainty, additional data could result in the identification of additional chemicals of concern or eliminate chemicals of concern, and refine exposure pathways (e.g., shellfish consumption) identified in the current Phase 1 assessment.

[3] As defined in this risk assessment, a chemical of concern has a cancer risk estimate greater than 1E-6 or a hazard quotient greater than 1

Because risk estimates were highest for the seafood consumption scenario, reducing uncertainties associated with this pathway will be the primary goal of additional data collection efforts. Windward (2002b) evaluates each data gap relative to the need for and feasibility of collecting additional data or performing additional analysis to fill the data gap. Uncertainties associated with the seafood consumption scenario are higher than uncertainties associated with the netfishing scenario. There are fewer site-specific data on tissue chemistry than there are for sediments and there is little information on the extent and nature of recreational fishing or shellfishing activities in the LDW. Based on a preliminary analysis of existing data, concentrations of arsenic in English sole and shellfish from non-urban areas around Puget Sound are similar to arsenic concentrations in these organisms obtained from the LDW. Thus, it is uncertain whether actions within the LDW to reduce arsenic concentrations would be effective in limiting exposure below that typically detected in Puget Sound sediments or in seafood from typical sources nationally. Moreover, because there were no site-specific data on the concentrations of cPAHs in fish, it was assumed that the concentrations of cPAHs in fish are the same as those in mussels. If the concentrations of cPAHs in fish were assumed to be negligible (as is typically assumed due to metabolism in fish), the risk estimate for cPAHs in the seafood consumption pathway would be reduced from 1E-4 to 2E-5 (i.e., estimated risks would decrease by a factor of approximately 5-fold).

The risk estimates made in this HHRA for consumption of fish and shellfish exceed levels identified by EPA as the upper end of the acceptable risk range, and thus suggest that remedial action may be warranted in the LDW. It is likely that early remedial action undertaken within the LDW will reduce this risk. A memorandum will be prepared shortly after the completion of the Phase 1 RI that proposes candidate sites for early remedial action. The Phase 2 HHRA will include an analysis that will evaluate the impact of these early actions on residual risk for the seafood consumption and direct sediment exposure scenarios. Identifying the extent of remediation that may be necessary based on the seafood consumption risk estimates will require that a linkage be derived or assumed between sediment and tissue concentrations. It is likely that some type of quantitative modeling of this linkage will be performed as part of the Phase 2 RI.

B.1 Introduction

This Phase 1 (scoping-phase) human health risk assessment (HHRA) calculates risk estimates for seafood consumption, dermal contact with sediment, and incidental ingestion of sediment for chemicals of potential concern (COPCs) found in sediments and fish tissue in the Lower Duwamish Waterway (LDW) (Map B-1)[4]. This approach is consistent with the requirements identified in the Statement of Work. The results of the Phase 1 HHRA and ecological risk assessment (ERA) will be used to identify high priority sites, which in turn will be further evaluated using management-based criteria to identify candidate sites for early action. Locations where elevated concentrations of these chemicals are found may be identified as priority sites, as described in greater detail in the technical memorandum *Identification of candidate sites for early actions – technical memorandum: Description of candidate site selection criteria* (Windward Environmental LLC [Windward] 2002).

The Phase 1 HHRA is based on existing data only. Data gaps identified in the data gaps memorandum (a subsequent deliverable) will be filled prior to conducting the baseline HHRA during the Phase 2 Remedial Investigation (RI). This Phase 1 HHRA includes the following sections: data evaluation (Section B.2), the conceptual site model and exposure assessment (Section B.3), toxicity assessment (Section B.4), risk characterization (Section B.5), and quantitative uncertainty assessment (Section B.6). Details on site background, previous investigations, and environmental setting are provided in the Phase 1 RI report and will be referenced accordingly.

B.2 Data Evaluation

This HHRA uses chemical data from previously conducted studies in a Phase 1 assessment. People may be exposed to chemicals found in LDW sediments, either through direct exposure to sediment or indirectly through the consumption of fish and shellfish (see Section 2.5.3 in the RI and Section B.3.2 of this appendix). Accordingly, both tissue and sediment chemistry data are relevant. Although water data were also available for this assessment, exposure pathways related to water are likely to result in much lower exposure than those for sediments and tissue. For example, in a recent comprehensive risk assessment conducted by King County (1999b), risk estimates for the water pathways were consistently lower than those for sediments and much lower than estimates related to seafood consumption. The representativeness of these findings for this assessment is discussed in Subappendix B.1. The following sections describe available data for tissue and sediment (Section B.2.1), data selection

[4] All maps referred to in text (e.g., Map B-1) are located in a separate section following the references (in the electronic version, maps are provided as a separate file, HHRA_maps.zip). Figures provided in the body of the document are numbered independently from maps.

(Section B.2.2), data reduction (Section B.2.3), and data reliability for risk assessment purposes (Section B.2.4).

B.2.1 DATA AVAILABILITY AND SELECTION

Many environmental investigations conducted within the LDW have included the collection of chemistry data from either fish and shellfish tissue or sediment. The data sources for each data type are described in detail in Section 2.3 of the RI and in summary below. Details on data aggregation and calculations are provided in Section B.2.3 and in applicable sections of the exposure assessment (Section B.3) where such calculations are used.

B.2.1.1 Sediment chemistry

Approximately 1,200 surface[5] sediment samples have been collected from the LDW within the last ten years. Older data exist, but the data quality objectives established in the first LDW RI Task 2 deliverable (Windward 2001a) suggest that data older than ten years are not representative of current conditions.[6] The sample collection locations are shown in Section 2.3.1 of the RI and in Map B-2.

Both intertidal and subtidal sediment chemistry are used for the Phase 1 HHRA. The elevation dividing intertidal and subtidal locations was –2 ft mean lower low water. This elevation corresponds to the shoreline (i.e., land / water interface) defined by the aerial photos taken by the US Fish and Wildlife Service in 1999 (Genwest Systems Inc. 2000). Approximately 400 surface sediment samples (i.e., 15 cm or less) were collected from intertidal locations; the remainder were collected from subtidal locations (Map B-3).

Section 2.3 of the RI describes all surface sediment samples collected for each event. As described in the sediment data quality objective memorandum (Windward 2001a), some of the surface sediment samples may not reflect current conditions in surface sediment because the sediment previously characterized has been remediated or dredged. Table 3-2 in the RI lists the surface sediment samples that will not be included in the Phase 1 RA for this reason. These same samples are excluded from the Phase 1 ERA (see Appendix A) and Section 4.2 of the RI (nature and extent of contamination).

[5] For the purposes of this risk assessment, surface sediment samples are those collected from the top 15 cm of the sediment horizon. Sediment samples that include less than 15 cm of sediment are included; samples that include the top 15 cm, but also include deeper sediment in the same sample are not included here because analyses were not performed separately on the two horizons (<15 cm and >15 cm).

[6] Data from the Harbor Island Remedial Investigation were collected more than 10 years ago. For the sake of continuity throughout the project, they are still being used in the risk assessment because the data set was identified as a suitable data source at the beginning of the project.

A summary of LDW sediment chemistry data is provided in Subappendix B.2 of the HHRA and Appendix D of the RI. Appendix D of the RI also describes a CD that accompanies the RI report that contains all the raw data used in the RI and RAs.

B.2.1.2 Fish and shellfish tissue chemistry

Tissue chemistry data for the study area are available from five projects (see Section 2.3.5 of the RI). All collection locations for LDW tissue samples are identified in Map B-4. Polychlorinated biphenyls (PCBs), as Aroclors, were analyzed in most samples. Pesticides and semivolatile organic compounds were also analyzed frequently. Mercury, arsenic, lead, copper, and tributyltin (TBT) were analyzed in fewer samples. Chemicals that have not been analyzed in LDW tissue samples, such as dioxins and furans, will be discussed in the uncertainty analysis (Section B.6).

Available data are from several different tissue types, not all of which are suitable for the various analyses done in the Phase 1 risk assessment. Site-specific tissue chemistry data are available for the following species: chinook salmon, coho salmon, English sole, Dungeness crab, red rock crab, mussels, shiner perch, and striped perch. Humans may consume all these species. There may be other species found in the LDW that are also consumed by humans, but there are no available tissue chemistry data for these other species. Table B-1 lists the fish species that have been found in the LDW and indicates available information regarding the degree to which these species are consumed from the LDW and Elliott Bay. Many of the species that may be consumed from Elliott Bay, but for which no LDW tissue chemistry data are available (e.g., speckled sanddab, Pacific cod, rockfish, spiny dogfish, walleye pollock), are rarely found in the LDW.

Table B-1. Fish species in the LDW

Common Name	Abundance	Citation	Environment	Habitat	Citation	Available Chemistry Data?	Evidence of Consumption
Bay goby	r	2, 3, 6	marine (estuary)	benthic (mud bottom)	8	no	none
Bay pipefish	r	6	marine	demersal (associated with eel grass in the intertidal areas)	10	no	none
Big skate	r	7	marine	benthic (sandy and gravelly bottoms)	11	no	none
Buffalo sculpin	r	1, 2, 3, 4, 7	marine (estuary)	benthic (inshore rocky and sandy areas)	8	no	<0.3% (28), <1% (30,31)
Bull trout	r	6	anadromous	benthopelagic (near shore)	16	no	none
Butter sole	c, (r)	6, (7)	marine (estuary)	benthic (sandy bottom)	8	no	<3.1% (28), <1.2% (29), <6% (30)

Common Name	Abundance	Citation	Environment	Habitat	Citation	Available Chemistry Data?	Evidence of Consumption
Chinook salmon	a, (r)	1, 4, 5, 6, (2)	anadromous	benthopelagic	23	yes	42% (28), <64% (31)
Chum salmon	r (a)	1, 4, (5, 6)	anadromous	benthopelagic	23	no	0.2% (28), <64% (31)
Coho salmon	r, (c), [a]	1, 2, (4), [6]	anadromous	benthopelagic	23	yes	20% (28), <64% (31)
Crescent gunnel	r	6	marine (estuary)	demersal (intertidal areas, under rocks)	8	no	none
Cutthroat trout	r	1, 4, 5, 6	anadromous	benthopelagic	17	no	none
Dolly Varden	r	1, 4	fresh water	benthopelagic	16	no	none
Dover sole	c, (r)	2, (3)	marine	benthic (mud bottom)	8	no	<3.1% (28), <1.2% (29), <6% (31)
English sole	a, (r)	2, 3, 4, 7 (1,6)	marine (estuary)	benthic (sand and mud bottoms)	13	yes	0.6% (29), 1% (30,31)
Eulachon	i	3	anadromous	pelagic	8	no	none
Flathead sole	i	2	marine	benthic (soft mud bottom, adults below 180m)	8	no	<3.1% (28), <1.2% (29), <6% (30)
Hybrid sole	r	1	marine (estuary)	benthic	8	no	<3.1% (28), <1.2% (29), <6% (30)
Largescale sucker	i (r)	1, 2, 4, (6)	fresh water	demersal	16	no	none
Longfin smelt	a, (r)	1, 2, (7)	anadromous	benthopelagic (close to shore, in bays and estuaries)	16	no	none
Longnose dace	i	6	fresh water	demersal	16	no	none
Mountain whitefish	i	1, 6	fresh water	benthopelagic	9	no	none
Northern pikeminnow	i	1, 6	fresh water	benthopelagic	15	no	none
Northern sculpin	r	6	marine	demersal	8	no	<0.3% (28), <1% (30,31)
Pacific cod	r	2, 3, 4	marine	(demersal, continental shelf and upper slopes)	18	no	6.5% (29), 9.2% (28), 10% (30)
Pacific herring	c, (a), [r]	1, 2, 7, (4), [6]	marine	benthopelagic (coastal, 1st yr in bays)	9	no	1% (31)
Pacific sandlance	c, (r), [a]	4, (1), [6]	marine (brackish)	benthopelagic (surface or burrowed in sand)	8	no	none
Pacific staghorn sculpin	a, (c)	1, 2, 3, 4, 6, (7)	Marine (lower estuary, offshore)	benthic (sandy bottom)	8	no	<0.3% (28), <1% (30,31)
Pacific tomcod	r, (c), [a juvi]	1, 4, (2, 3), [7]	marine (brackish)	benthic (over sand)	18	no	<1% (30), 1% (29)

Common Name	Abundance	Citation	Environment	Habitat	Citation	Available Chemistry Data?	Evidence of Consumption
Padded sculpin	c, (r)	2, 3, (7)	marine	benthic	8	no	<0.3% (28), <1% (30,31)
Penpoint gunnel	r	5, 6	marine (estuary)	demersal (intertidal-tidepools)	8	no	none
Pile perch	r, (c)	1, 2, 3, 6, (4, 7)	marine	demersal (rocky shores; near kelp, pilings, underwater structures)	8	no	<1% (30), 4.5% (28), <11% (31)
Pink salmon	r	6	anadromous	benthopelagic	23	no	0.2% (28), <64% (31)
Plainfin midshipman	i	2	marine	benthic (nearshore shelf, sand/mud bottom)	13	no	none
Prickly sculpin	r	1, 2, 3, 4, 6	marine	benthic	8	no	<0.3% (28), <1% (30,31)
Pygmy poacher	i, (r)	2, 3, (7)	marine	demersal (soft bottoms)	8	no	none
Ratfish	r	2, 7	marine	demersal (sandy bottom)	8	no	none
Redsided shiner	c	6	fresh water	demersal	15	no	none
River lamprey	r	1, 4, 6	anadromous	demersal	9	no	none
Rock sole	c, (a)	2, 3, (7)	marine (estuary)	benthic (more pebbly bottom than most other flatfish)	8	no	0.7% (29), 1% (30), 1.4% (28)
Rockfish	r	1	marine	demersal (near structure)	20	no	2% (31), 2.3% (29), 6.5% (28)
Roughback sculpin	i, (r)	2, (3, 7)	marine	benthic (sand/mud bottom)	8	no	<0.3% (28), <1% (30,31)
Saddleback gunnel	r	3, 5, 6	marine (estuary)	demersal (sandy bottom)	8	no	none
Sand sole	c, (r)	1, 2, 3, 7, (1)	marine, estuary	benthic (sandy bottom)	9	no	<3.1% (28), <1.2% (29), <6% (31)
Sharpnose sculpin	i	6	marine	benthic (sand/vegetation)	8	no	<0.3% (28), <1% (30,31)
Shiner surfperch	a, (c)	1, 4, 5, 6, 7, (2, 3)	marine (estuary)	demersal (in shallow water, around eelgrass beds, piers and pilings commonly in bays and quiet back waters)	8	yes	<1% (30), <11% (31)
Slender sole	i	3	marine	benthic (>200m depth)	8	no	<3.1% (28), <1.2% (29), <6% (30)
Snake prickleback	a, (r)	1, 2, 3, 4, 6, (7)	marine	benthopelagic (shallow bays and offshore waters)	8	no	none

Common Name	Abundance	Citation	Environment	Habitat	Citation	Available Chemistry Data?	Evidence of Consumption
Sockeye salmon	i		anadromous	benthopelagic	23	no	0.2% (28), <64% (31)
Soft sculpin	r	4	marine	demersal	8	no	<0.3% (28), <1% (30,31)
Speckled sanddab	r	7	marine	benthic (sandy bottom)	8	no	<3.1% (28), <1.2% (29), <6% (30)
Spiny dogfish	i	2	marine	benthopelagic	21	no	0.9% (29), 1.6% (28)
Starry flounder	a, (c)	1, 2, 3, 4, 6, 7, (5)	marine (estuary, brackish)	benthic	17	no	<3.1% (28), <1.2% (29), <6% (30)
Steelhead	r	1, 4, 5, 6	anadromous	benthopelagic		no	0.5% (29)
Striped seaperch	r, (c)	2, 3, 5, 6, 7, (1, 4)	marine	demersal	8	yes	2% (30), 2.5 (28), <11% (31)
Sturgeon poacher	i	3	marine	demersal (soft bottom)	8	no	none
Surf smelt	c	1, 4, 6, 7	marine (brackish)	benthopelagic	17	no	none
Three-spine stickleback	c, (r)	1, 5, 6 (4)	marine, anadromous	benthopelagic (in/near vegetation)	16	no	none
Tubesnout poacher	i	3	marine	demersal (eelgrass & seaweeds)	8	no	none
Walleye pollock	r	1, 2, 4	fresh water	benthopelagic	18	no	3.1% (29), 10% (28,30)
White-spotted greenling	i, (c)	2, (7)	marine (intertidal)	demersal (nearshore, near rocks, pilings and eelgrass beds)	18	no	none

Abundance: a-abundant (numerically dominant), c-common (occurs in most samples), r-rare (occurs regularly in a few samples), i-incidental (not usually found in LDW). Letters in parentheses relate distinct abundance classification to citation; numbers in parentheses indicate the source of the distinct data.

Abundance citations: 1-Matsuda et al (1968), 2-Miller et al. (1975), 3-Miller et al. (1977a), 4-Weitkamp and Campbell (1980), 5-Taylor et al. (1999), 6-Warner and Fritz (1995), 7–West et al. (2001)

Biology citations: 8-Eschmeyer et al. (1983), 9-Hart (1973), 10-Dawson (1985), 11-McEachran and Dunn (1998), 12-Armstrong (1996), 13-Clemens and Wilbey (1961), 14-Fitch and Lavenberg (1975), 15-Scott and Crossman (1973), 16-Page and Burr (1991), 17-Morrow (1980), 18-Cohen et al. (1990), 19-Pearcy and Hancock (1978), 20-Lamb and Edgel (1986), 21-Cox and Francis (1997), 22-Compagno (1985), 23-Groot and Margolis (1991), 24-Grossman (1979), 25-Miller et al. (1977b), 26-Cordell et al. (2001), 27-Rieman and McIntyre (1993)

Evidence of consumption – numbers given are percentages of total catch by weight from the studies cited in parentheses: 28 – Landolt et al. (1987), 29 – Landolt et al. (1985), 30 – McCallum (1985), 31 – King County Water Quality Assessment (1999)

Data from four of the five tissue chemistry studies presented in Section 2.3.5 of the RI were used in the HHRA. Data from Varanasi et al. (1993) were not used in the HHRA because this study sampled only juvenile chinook salmon, which are not representative of the sizes of fish consumed by humans (see below). The tissue samples used in the Phase 1 RA are summarized in Table B-2. Only data from composite samples for crab, English sole filets, perch filets, and mussels were used in

this HHRA (Table B-2). Some site-specific tissue chemistry data were excluded from the HHRA because of the lack of relevance to site-specific contamination (e.g., salmon) or because the sample type does not reflect consumption patterns of the majority of the target population (e.g., English sole and shiner perch whole bodies). Data from whole-body samples may be relevant for characterizing exposure to specific populations (e.g., Asian and Pacific Islanders). The Phase 2 HHRA will include additional whole-body data for characterizing exposure to these populations.

Composite tissue samples included in the HHRA consisted of 3 to 20 individuals. Samples of individual fish or shellfish were not collected for any of the species listed on Table B-2. While composite samples are less representative of potential variability in concentrations, they are considered representative of human exposure over time. All concentrations qualified as estimates (i.e., J-flagged data) were assumed to be positive identifications and were used without modification in subsequent calculations. Non-detected values were assigned a value equal to one-half the identified detection limits for data aggregation purposes, such as the calculation of exposure point concentrations (see Section B.3.4.3).

Salmon are potentially exposed to sediment-related chemicals indirectly through their diet only as outmigrating juveniles. There is unlikely to be any relationship between site-related contamination and chemical concentrations in adult salmon tissue, because adult salmon generally do not feed once they enter rivers and streams, and diet is probably the primary exposure pathway to sediment-related chemicals. Salmon returning to the Duwamish estuary are exposed to site-related chemicals very briefly as juveniles, but the contribution of this exposure to adult body burdens is likely to be insignificant (O'Neill et al. 1998). For example, a 10-g juvenile chinook salmon with a total PCB concentration of 140 µg/kg, the mean concentration reported by Varanasi et al. (1993) contains 1.4 µg of PCBs. A 15-kg returning adult chinook salmon captured in the Duwamish River with a total PCB concentration of 56 µg/kg, the mean concentration reported by West et al. (2001), contains 840 µg of PCBs, almost all of which is derived from ingestion of food in Puget Sound and the Pacific Ocean. Based on these data and the analysis presented by O'Neill et al. (1998), less than 1% of the PCB body burden contained in adult salmon that may potentially be consumed by humans could have been obtained from prey items from the Duwamish. Therefore, because this assessment is focused on evaluation of risks related to the Duwamish system, adult salmon were not included in the quantitative risk assessment.

Table B-2. Tissue chemistry samples collected from the LDW that were used in the Phase 1 HHRA

Title	Year	Species	Number of Samples	Number of Individuals per Sample	Sample type	Chemicals
Waterway Sediment Operable Unit Harbor Island Superfund Site - Assessing human health risks from the consumption of seafood (Environmental Solutions Group 1999)	1998	English sole	3	5	skinless filet	Hg, TBT, PCBs
		red rock crab	2	5	edible meat	
		Dungeness crab	1	1	edible meat	
		striped perch	3	1-5	skinless filet	
King County Combined Sewer Overflow Water Quality Assessment for the Duwamish River and Elliott Bay (King County 1999a) [a]	1996-1997	Dungeness crab	2	3	edible meat	metals, TBT, semivolatiles, PCBs
			1	3	hepatopancreas	
		English sole	3	20	skinless filet	
		mussels	22	50-100	whole body	
Puget Sound Ambient Monitoring Program – annual sampling (West 2001) [b]	1992	English sole	3	5-20	skinless filet	semivolatiles, pesticides, PCBs, As, Cu, Pb, Hg
	1995	English sole	3	5-20	skinless filet	pesticides, PCBs, As, Cu, Pb, Hg
	1997	English sole	3	5-20	skinless filet	Hg, pesticides
Elliott Bay/Duwamish River Fish Tissue Investigation (Battelle Marine Research Laboratory 1996, EVS 1995, Frontier Geosciences 1996)	1995	English sole	3	6	skinless filet	PCBs, Hg, Methylmercury, TBT

[a] Additional samples of cooked crab and English sole were collected during the King County Water Quality Assessment and were used in that assessment (King County 1999a), but were not used in the main risk assessment (see uncertainty assessment). Approximately 30 additional mussel samples, beyond those indicated in the table, were analyzed as part of the caged mussel deployment designed to assess impacts from the combined sewer overflows. These data are not included in this HHRA because they are not representative of concentrations in mussels that people could collect. Four amphipod samples were also collected in support of the ERA.

[b] Approximately 140 samples of chinook and coho salmon filets (both composites and individuals) were collected from 1992 to 1998. Data from these samples were not included in the HHRA because the chemical concentrations in these fish are unrelated to site-specific contamination.

Samples of whole crab bodies collected for the King County Water Quality Assessment (King County 1999b) were excluded from the HHRA because most area anglers do not consume the entire crab body (Environmental Solutions Group 1999). Tissue chemistry data from crab hepatopancreas samples were used for some exposure scenarios because this tissue is consumed by some populations. Additional details on the sample types included for each fish and shellfish exposure scenario are provided in Section B.3.4. A few samples of cooked edible portions were also available, but were excluded because the highly variable nature of cooking methods and equipment would make comparison to other data sets difficult. Available data suggest that cooking alters the concentration of PCBs (Skea et al. 1979; Zabik et al. 1979, 1982) and mercury (Morgan et al. 1997) in tissues on a wet weight basis. Data from cooked samples are relevant for the evaluation of human health, since most people cook seafood before eating it. However using a combination of data for cooked and uncooked fish will not provide a consistent means to evaluate risks. The site-specific data on cooked seafood and consumption of crab hepatopancreas are evaluated in the uncertainty analysis (Section B.6).

For the 1996 Elliott Bay / Duwamish River fish tissue study, both total mercury and methylmercury were analyzed in three English sole composite samples. Toxicity values (RfDs) for oral exposure are available for mercuric chloride and methylmercury, as described in Subappendix B.5. Since the majority of mercury in fish tissue samples is in the form of methylmercury (EPA 2000b), total mercury concentrations are often used as a surrogate for methylmercury concentrations. This represents a health-protective approach because methylmercury is the most toxic form via the oral route. For this HHRA, only total mercury data are included for purposes of consistency with the other datasets that did not include methylmercury. No samples were analyzed for methylmercury without also being analyzed for total mercury. Where both methylmercury and total mercury measurements were available, concentrations were very similar.

A summary of LDW tissue chemistry data is provided in Subappendix B.2 of the HHRA and Appendix D of the RI. Appendix D of the RI also describes a CD that accompanies the RI report that contains all the raw data used in the RI and RAs.

B.2.2 DATA REDUCTION

Data reduction refers to computational methods used to aggregate data. Data selected according to the description in Section B.2.2 were utilized in subsequent analyses on a dry weight basis for sediment chemistry and on a wet weight basis for tissue chemistry, with the exception of analytes reported as undetected and arsenic in tissue samples. Concentrations generated by the laboratory through duplicate analyses were treated as quality control samples and averaged for use in subsequent calculations.

A concentration equal to one-half the sample-specific detection limit (as reported by the laboratory) was used for undetected analytes. Concentrations for several analyte sums were calculated as follows:

- **Total PCBs** were calculated using only detected values for 7 Aroclor mixtures in accordance with Ecology's Sediment Management Standards (WAC 173-204). For individual samples in which none of the 7 Aroclor mixtures were detected, total PCBs were given a value equal to the highest detection limit of the 7 Aroclors and assigned a "U" qualifier indicating the lack of detected concentrations.
- **Toxic equivalency quotients (TEQs) for 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) and carcinogenic polycyclic aromatic hydrocarbons (cPAHs)** were calculated by summing the products of concentrations and compound-specific toxic equivalency factors (TEFs), including TEFs for polychlorinated dibenzo-p-dioxins or furans (PCDD/Fs) or relative potency factors (RPFs) for cPAHs, as shown in Table B-3. Compounds that were undetected for a given sample were assigned a value equal to one-half the sample-specific detection limit for use in the TEQ calculation.
- **Total DDTs** were calculated from detected concentrations of three isomers: 4,4'-DDD, 4,4'-DDE, and 4,4'-DDT. For samples in which all individual isomers were undetected, the single highest detection limit for that sample was assigned to represent the sum of the three isomers.

Table B-3. Toxic equivalency factors for dioxins/furans and cPAHs

COMPOUND	TOXIC EQUIVALENCY FACTOR
Dioxins and furans	
2,3,7,8-TCDD	1
1,2,3,7,8-PeCDD	1
1,2,3,6,7,8-HxCDD	0.1
1,2,3,4,7,8-HxCDD	0.1
1,2,3,7,8,9-HxCDD	0.1
1,2,3,4,6,7,8-HpCDD	0.01
OCDD	0.0001
2,3,7,8-TCDF	0.1
1,2,3,7,8-PeCDF	0.05
2,3,4,7,8-PeCDF	0.5
1,2,3,6,7,8-HxCDF	0.1
1,2,3,7,8,9-HxCDF	0.1
1,2,3,4,7,8-HxCDF	0.1
2,3,4,6,7,8-HxCDF	0.1
1,2,3,4,6,7,8-HpCDF	0.01
1,2,3,4,7,8,9-HpCDF	0.01
OCDF	0.0001
cPAHs	
Benzo[a]pyrene	1
Benz[a]anthracene	0.1
Benzo[b]fluoranthene	0.1
Benzo[k]fluoranthene	0.1
Chrysene	0.01
Dibenz[a,h]anthracene [a]	0.4
Indeno[1,2,3-cd]pyrene	0.1

Sources:

Dioxin/furan TEFs – World Health Organization (Van den Berg et al. 1998)

cPAHs – carcinogenic polycyclic aromatic hydrocarbons, as defined by California EPA, Office of Environmental Health Hazard Assessment (OEHHA 1999); TEFs for PAHs not analyzed in LDW sediments are not shown

[a] The TEF was determined by OEHHA by dividing the inhalation unit risk factor for this compound by the inhalation unit risk factor for benzo[a]pyrene

Arsenic in fish tissue is predominantly in nontoxic organic arsenic forms (EPA 1997b, Schoof et al. 1999a). EPA (1997b) recommends adjusting fish tissue concentrations to account for the reduced toxicity related to arsenic in fish. EPA Region 10 guidance (currently under development) recommends 10% as an appropriate fraction of inorganic arsenic for aquatic species. This fraction may be high, but is an upper bound estimate used by NRC (1999) in their evaluation of arsenic intake from fish and shellfish. All total arsenic concentrations in seafood were multiplied by 0.1 (i.e., 10%) for use in the exposure assessment.

B.2.3 SUITABILITY OF DATA FOR RISK ASSESSMENT

There are several factors to consider in assessing the suitability of environmental data for risk assessments (EPA 1989, 1992c). Of primary importance is the degree to which the data adequately represent site-related contamination and the expected human exposures at the site. Also important to consider are the data quality criteria goals, and the source, documentation, analytical methods / detection limits, and level of review associated with the data. Since data from many different investigations were available for the LDW, the factors described above had to be evaluated for each data set to determine whether it was reasonable to combine all data for use in this RA.

B.2.3.1 Representativeness to site-related contamination

B.2.3.1.1 Sediment

Many environmental sampling events have included the collection of potentially contaminated sediment (see Section 2.3 of the RI). The studies have been designed for both reconnaissance (e.g., Boeing SiteChar, EPA SI, and NOAA SiteChar) and focused (e.g., Boeing RFI, Rhône-Poulenc RFI) investigation on suspected areas of contamination. Most events focused on subtidal sediments, although intertidal sediments have also been collected. The extensive coverage of the reconnaissance surveys and the focused intensity of the facility investigations indicate that the available sediment chemistry data are representative of the general range of environmental conditions within the LDW. Far more samples have been collected in areas where chemical concentrations were high (near known sources). Standard statistical measures (e.g., mean, median) may therefore not be representative of the overall distribution of chemicals in the LDW. However, because a good spatial distribution of samples is available for most chemicals, spatially-weighted averages (SWAs) are likely to be fairly representative of overall conditions. SWAs were calculated over areas likely to be traversed by the receptor. SWAs may be less representative of persons visiting intertidal areas because there are relatively few sediment samples from those areas likely to be visited by recreational visitors. Additional research on site usage and additional data collection in intertidal areas will be conducted in Phase 2 so that less uncertain SWAs can be calculated for those areas. A discussion of the distribution of sediment chemistry data and the manner in which they will be used in the HHRA to calculate exposure point concentrations (EPCs) is provided in Section B.3.4.3.

B.2.3.1.2 Tissue

Representativeness was evaluated by looking at the migratory behavior of the target species and by reviewing the collection locations with respect to the location of the study area. The tissue samples analyzed since 1992 and summarized in Table B-2 were collected during the spring and fall.

Although site-specific studies of migration behavior are not available for English sole and crab, available data on the life history of these species in other regions suggest

that during the spring and fall, the individuals are resident in the waterbody in which they were captured (Lassay 1989; Miller et al. 1975; Pauley et al. 1988). Perch were captured only in the fall, when they are abundant in nearshore environments (Fritzsche and Hassler 1989), although they are also present during other seasons. Thus, each of the resident fish and shellfish from the studies summarized in Table B-2 were apparently exposed to the chemical environment in the vicinity of where they were captured for at least several months of the year.

The size of the home range of each resident species (i.e., perch, English sole, and crab) within the entire LDW is unclear, since no site-specific research of home ranges has been conducted. The degree to which harvestable shellfish exist in the LDW will be further characterized in Phase 2. For purposes of this risk assessment, it was assumed that perch, English sole, and crab are present, and home range estimates for these species were developed using best professional judgment. The unconstrained average home range of English sole, as reported by Puget Sound Dredged Disposal Authority (PSDDA 1988) is 9 km^2. The spatial variability of certain fish tissue abnormalities observed in other areas of the Sound (e.g., Hylebos Waterway [Myers et al. 1998]) is consistent with this value. Similarly, the unconstrained home range of Dungeness crab has been reported to range from 0.1 to 1 km per day (Breen 1985; Waldron 1958), and Ecology has used an area of 10 km^2 in crab-based risk assessments performed elsewhere in Puget Sound (e.g., Bellingham Bay). The resident species to be characterized in this HHRA are mobile but they also demonstrate some site fidelity (Lassay 1989, Pauley et al. 1988, Fritzsche and Hassler 1989), indicating they may have spent more time in the LDW than outside the LDW.

Within the LDW, samples of perch, crab, and English sole were collected from several locations. Given the variety of collection locations, the individuals within each composite sample likely represent exposure to a relatively wide range of chemical regimes. These samples, therefore, also reasonably approximate exposure of anglers who consume these organisms from the LDW. The fact that the different fish and shellfish species evaluated here may range throughout the LDW imparts some uncertainty to risk estimates based on chemical concentrations in tissues.

B.2.3.2 Representativeness to expected human exposure

B.2.3.2.1 Sediment

As described in Section B.2.4.1, the extensive coverage of several large sampling events overlaps any expected human exposure to subtidal sediments in the LDW (see Section B.3.2). The overall distribution of sampling appears to reflect the expected human exposure to the subtidal portion of the site.

The areal extent of intertidal sediment sampling is less extensive compared to subtidal sampling. Intertidal sampling density is high in some areas where focused investigations have occurred. Areas with high sampling density do not necessarily correspond with areas where human exposure is expected to occur. For example,

many intertidal sediment samples were collected as part of the Boeing Plant 2 RFI, but general public access to this site is strictly controlled. Sampling density in other areas was very low. For example, few intertidal sediment samples have been collected between Slip 4 and Kellogg Island. There are several public access sites located in this reach of the river. Also, there are a number of riverfront homes located in South Park. Available intertidal sediment data do not appear to adequately characterize the expected human exposure in these reaches. The need for additional data on this topic will be discussed in the data gaps memorandum (an upcoming deliverable).

B.2.3.2.2 Tissue

Representativeness of the tissue data to expected human exposure was evaluated by reviewing the time of sample collection (i.e., does it coincide with a time during which harvest normally occurs?) and the size range of the samples collected (is this size range normally consumed?).

An extensive review conducted by Environmental Solutions Group (1999) of existing seafood consumption surveys indicated that all the species listed on Table B-2 are potentially consumed by anglers in the LDW and Elliott Bay. Washington State sportfishing regulations (2000-2001) specify year-round seasons for English sole, perch, and crab, excluding pot gear (WDFW 2000). Therefore, all sampling dates are within the potential harvest window allowed by state regulations.

Size restrictions have been established for crab, but not for perch and English sole (WDFW 2000). Crab samples collected for the Port of Seattle (Environmental Solutions Group 1999) and King County (1999b) were all of legal size (at least 6.25 in carapace width) and sex (male). Within each composite sample of English sole and perch, the length of the smallest fish was not less than 75 percent of the length of the largest fish (Environmental Solutions Group 1999), and the average lengths within a composite were similar between composites. There are no available data that suggest that the minimum size fish from that study (250 mm total length [9.8 in] for English sole and 120 mm total length [4.7 in] for striped seaperch) captured would not be consumed.

B.2.3.4 QA/QC results

All data sets used in the HHRA have been validated by the original authors of the individual studies or by outside third parties, although the documentation of such QA/QC validation is sometimes minimal. No additional data validation is planned for this HHRA. The data validation results were summarized in Windward (2001b). Some results were qualified as unusable[7] by the data validators. Data qualified as unusable were not used in this HHRA. Additional data validation may occur during the Phase 2 RI, at which time the suitability of historical data for use in Phase 2 will be determined in consultation with the agencies.

[7] Approximately 1,000 results were qualified as unusable out of more than 140,000 analytical results

B.2.3.5 Other factors

Documenting field and laboratory procedures makes it possible to assess the impact of any deviation from these procedures on data usability. As described in Windward (2001a), such procedures were documented during the verification process that was conducted during database construction. A thorough review of the documentation provided (e.g., method descriptions, quality control results) for the various studies did not reveal any issues that would adversely affect the usability of the data for risk assessment purposes.

The level of analytical data review can also affect data usability. All data used in this risk assessment were subjected to a thorough data reduction and validation process. Other factors that could potentially impact data usability for specific data types are described below.

B.2.3.5.1 Sediment

The sediment surveys summarized in Section 3.4 of the RI utilized similar or identical analytical methods for most analytes, with one notable exception. PCB analyses for NOAA SiteChar were conducted by high performance liquid chromatography and a photodiode array detector (HPLC/PDA), in contrast to PCB analyses for all the other events, which were conducted by gas chromatography and an electron capture detector (GC/ECD). NOAA data for total PCBs are based on a nonstandard analytical method and may not be quantitatively comparable to other data generated using standard analytical techniques. The NOAA laboratory data for total PCBs reflect the difference between the results of one analysis for the sum of PCBs and polychlorinated terphenyls (PCTs) and the results of a separate analysis for PCTs alone.

Krahn et al. (1998) reported the results for 30 samples that were analyzed by both HPLC/PDA and GC/ECD methods by two different laboratories.[8] The two laboratories calculated total PCBs for each sample, which were then compared to each other. Total PCB concentrations between the two laboratories varied by as much as a factor of six (Krahn et al. 1998). Regression analyses conducted for the two sets of results indicate that the GC/ECD results were lower than the HPLC/PDA results at high PCB concentrations, and higher than the HPLC/PDA results at low PCB concentrations (Krahn et al. 1998). The regression coefficient (R^2) between the two sets of analyses was 0.92. The differences between the total PCB concentrations calculated by the two laboratories are not surprising given the differences between the two methods, including 1) different ranges of linear response for the two detectors, 2) differences in methods for calculating total PCBs, 3) differences in methods of quantifying and/or removing analytical interferences, and 4) differences in detection limits.

[8] HPLC/PDA analyses were done by the NMFS laboratory in Seattle; GC/ECD analyses were done by Analytical Resources Inc., Seattle.

Despite the differences between the two analytical methods, data from both methods were used in this risk assessment, although the uncertainty associated with total PCB concentrations may be significant in some areas. Alternate risk calculations were presented in the uncertainty analysis section of the ERA and HHRA using total PCB data derived only from summing Aroclor concentrations analyzed using GC/ECD.

B.2.3.5.2 Tissue

The source of analytical data used in risk assessments can be an issue if data from different investigations are used. Although different laboratories and in some cases different methods were used for the various surveys, inter-survey consistency in sample types (e.g., skinless filets) and species selection indicates that combining data from various sources is acceptable.

Detection limits can affect data usability if they are higher than risk-based screening concentrations (RBCs). Detection limits higher than the corresponding RBCs were noted for several chemicals (see Section B.3.3.2), which were subsequently identified as COPCs based solely on this observation. The uncertainty associated with the risk characterization for these chemicals is high. A quantitative analysis of this uncertainty is provided in Section B.6.

Analytical methods were generally consistent among studies, but some variations were noted. PCBs were quantified in all studies except Environmental Solutions Group (1999) using an electron capture detector (i.e., EPA Method 8081). In the latter study, PCBs were quantified with a low-resolution mass spectrometer. The two types of detectors should give similar results, and there should be little if any impact on data comparability and usability. All analyses quantified individual Aroclors, which were then summed in an identical manner.[9]

B.3 Exposure Assessment

The exposure assessment describes scenarios in which people may come in contact with COPCs from sediment and provides equations and parameters so that potential exposures can be quantified. Section B.3.1 summarizes previous exposure assessments. Section B.3.2 presents the conceptual site model that introduces the exposure scenarios that were evaluated in this HHRA. Section B.3.3 describes a risk-based screening procedure to identify which chemicals were evaluated in detail in the HHRA. Section B.3.4 describes how the exposure scenarios were quantified. Section B.3.5 presents chronic daily intake calculations for all chemicals evaluated in this HHRA using the equations presented in Section B.3.4.

[9] Total PCBs concentrations derived from Aroclor data are presented as the sum of detected values only. In cases where all Aroclors were undetected, the total PCB concentration was assumed to be equal to the highest detection limit from among all the individual Aroclors.

B.3.1 SUMMARY OF PREVIOUS EXPOSURE ASSESSMENTS

The Phase 1 HHRA is based on existing data only, including study designs from previously conducted risk assessments. The first step in the exposure assessment is to select exposure scenarios to evaluate quantitatively. Exposure scenarios evaluated previously for the LDW, including Harbor Island, are summarized in Table B-4. Subappendix B.1 provides a summary of the risk assessment conducted by King County and evaluates the importance of the surface water pathway, which is not quantified in this assessment for the LDW.

Two exposure scenarios, consumption of fish / shellfish by recreational anglers and exposure to sediment by commercial fishermen, were evaluated in more than one risk assessment (Table B-4). The risk assessment conducted by King County (1999b) evaluated two pathways, SCUBA diving and windsurfing, that were not linked to sediment. King County (1999b) also evaluated a swimming scenario, which was based on exposure to water and sediment. None of the previous assessments quantified the potential direct exposure to intertidal sediments through activities focused on the LDW shoreline.

Table B-4 Exposure scenarios evaluated in previous risk assessments

SITE/PROJECT	REFERENCE	ACTIVITY	ROUTE/EXPOSURE MEDIUM	GROUP
Waterway Sediment Operable Unit Harbor Island Superfund Site	Environmental Solutions Group (1999)	recreational fishing	consumption of fish/shellfish	adults
LDW and Elliott Bay Water Quality Assessment	King County (1999b)	swimming	incidental ingestion of water	adults/children
			dermal contact with water	adults/children
			incidental ingestion of sediment	adults/children
			dermal contact with sediment	adults/children
		SCUBA diving	incidental ingestion of water	adults
			dermal contact with water	adults
		windsurfing	incidental ingestion of water	adults
			dermal contact with water	adults
		commercial fishing	incidental ingestion of water	adults
			dermal contact with water	adults
			incidental ingestion of sediment	adults
			dermal contact with sediment	adults
		recreational fishing	consumption of fish and shellfish	adults/children
Boeing Plant 2 RCRA facility investigation (LDW)	Weston (1998)	commercial fishing	incidental ingestion of sediment	adults
			dermal contact with sediment	adults
		recreational fishing	consumption of fish and shellfish	adults
Harbor Island RI	Weston (1993)	commercial fishing	incidental ingestion of sediment	adults
			dermal contact with sediment	adults

B.3.2 CONCEPTUAL SITE MODEL

The conceptual site model is a graphical representation of chemical sources, transport mechanisms, exposure pathways, exposure routes, and potentially exposed populations. It provides the basis for developing exposure scenarios to be evaluated in the exposure assessment component of the HHRA.

The conceptual site model is presented in Figure B-1. For the purposes of this HHRA, sediments are the only chemical source quantitatively evaluated. Although other chemical sources exist, the exposure assessment is only focused on scenarios that include a direct (i.e., ingestion or dermal contact) or indirect (i.e., consumption of fish or shellfish) pathway from potentially contaminated sediments. Additional discussion of chemical sources is provided in Section 4.3 of the Phase 1 RI report.

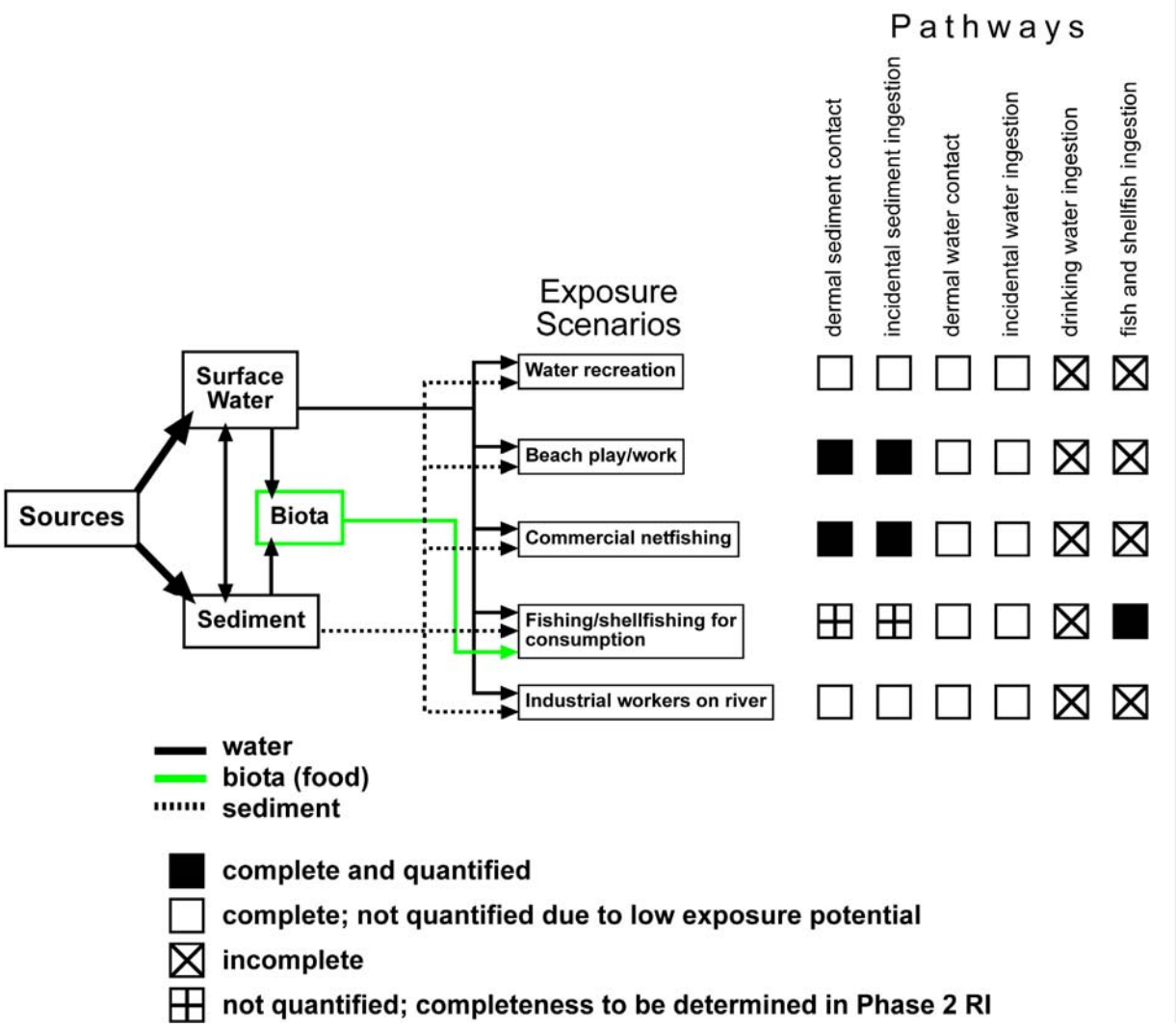


Figure B-1. Conceptual site model for Phase 1 human health risk assessment

Five exposure scenarios are represented in Figure B-1, corresponding to potentially exposed populations described in Section 2.5 of the Phase 1 RI report. Each *exposure scenario* (e.g., beach play / work) includes a potential *exposure pathway* to contaminated sediments (e.g., dermal contact with sediments; incidental ingestion of sediments) and a potential *exposure route* through which contaminants can enter the body of an exposed individual (e.g., dermal absorption of contaminants through exposed skin surfaces; gastrointestinal absorption of ingested contaminants), although the importance of some combinations of pathway and route is minor for some scenarios (e.g., ingestion of drinking water was considered to be an incomplete pathway for all scenarios considered). Each scenario shown in Figure B-1 is discussed below.

B.3.2.1 Water recreation

The water recreation scenario includes kayaking / canoeing, swimming, SCUBA diving, and windsurfing. Although the primary exposure medium for these activities

is water, individuals may come in contact with contaminated sediments that have been resuspended in the water column or as they enter the water from the shore. King County, in their issue paper on human site use in the Duwamish River and Elliott Bay (King County 1999a), concluded that the frequency of these recreational activities would be low in the LDW as compared to Elliott Bay, due to the industrial nature of the waterway and limited access to the water. Future remedial and restoration actions could increase the frequency of these recreational activities; however, the future use of the LDW is likely to remain largely industrial and therefore recreational opportunities are likely to remain limited.

King County (1999b) concluded that the risk from these activities in both Elliott Bay and the LDW was generally within the range of risks considered to be acceptable by EPA. Excess cancer risks were highest for arsenic and PCBs, ranging from 2E-7 for adults exposed to PCBs to 4E-6 for young children exposed to arsenic. All hazard indices were less than one. The risks associated with the water component of the swimming scenario were small (25% or less) compared to risks associated with the sediment component. Subappendix B.1 summarizes the methods and results from the King County HHRA for three of the direct exposure scenarios: swimming, SCUBA diving, and windsurfing. King County assumed that recreational SCUBA diving and windsurfing would not occur in the LDW (King County 1999b).

B.3.2.2 Beach play/work

The beach play / work scenario pertains to the following groups: 1) children and adults that recreate at LDW waterfront (now and in the future), 2) individuals that trespass at intertidal areas within private property boundaries, 3) individuals that live in homes bordering the LDW, and 4) volunteers and public sector employees responsible for habitat restoration within intertidal areas. This scenario includes two pathways: incidental ingestion of intertidal sediment or dermal contact with intertidal sediment. Direct contact with surface water may occur in some scenarios, but the contact frequency and magnitude is likely to be very low compared to the contact frequency and magnitude for sediments, since most of the activities in intertidal regions of the LDW probably occur on land. Subappendix B.1 provides a comparison of the relative importance of sediment surface water pathways in the risk assessment conducted by King County (1999b).

B.3.2.3 Commercial netfishing

Individuals from the Muckleshoot Tribe participate annually in a commercial netfishing operation in the LDW. The gillnet lead lines may come in contact with sediments during normal operations. The fishermen may contact this sediment incidentally upon net retrieval. The fishermen may also make incidental contact with surface water and sediment suspended in surface water.

B.3.2.4 Fishing/shellfishing for consumption

Fish and shellfish consumed by people fishing in the LDW may have acquired chemicals from their association with contaminated sediments in the LDW. These individuals may also come in direct contact with surface water and sediment. Contact with these media is likely only incidental for fishermen, but sediment contact would be common for individuals harvesting clams. Incidental ingestion and dermal contact with sediment will be added to this scenario if clam harvest is added to the baseline risk assessment to be conducted in Phase 2. Available data suggest that current clam harvest from the LDW is very rare due both to the current LDW conditions (e.g., high level of industrial activities, few areas with exposed sediments, advisories against shellfish consumption because of fecal coliform bacteria contamination) and to the natural conditions at the site limiting suitable habitat. Additional data will be collected during the Phase 2 RI to estimate the current harvestable clam population in the LDW and the potential for increased harvest of clams that could occur subsequent to remediation and/or restoration activities within the LDW. Fecal coliform bacterial concentrations would not be a factor in developing shellfish consumption rates for a risk assessment that considers future use.

B.3.2.5 Industrial workers on river

Many of the facilities adjacent to the LDW rely on vessel traffic on the waterway. Workers on these vessels could potentially come in contact with sediment and surface water on rare occasions. The expected contact frequency and magnitude is likely to be much lower compared to the other exposure scenarios shown in Figure B-1 because these workers are typically on board vessels well above the water surface. Industrial workers not associated with vessels, such as workers at cement plants and shipyards, are not expected to come in contact with surface water and sediment on a regular basis because they work at land-based facilities.

B.3.2.6 Selection of exposure scenarios for quantification

Specific exposure assumptions were developed to quantify some exposure pathways for the scenarios shown in Figure B-1. A complete exposure pathway includes an exposure medium and exposure point, a potentially exposed population (see Section 2.5.3 of Phase 1 RI report) including receptor age (i.e., adult vs. child) and an exposure route (see Figure B-1). The exposure parameters (see Section B.3.4), and the likelihood of exposure under both current and future land use at the site are discussed for all exposure pathways quantified.

EPA (1989) states "actions at Superfund sites should be based on an estimate of the reasonable maximum exposure (RME) expected to occur under both current and future land-use conditions." EPA defines the RME as "the highest exposure that is reasonably expected to occur at a site." The scenarios evaluated in this HHRA were consistent with RME guidelines. Separate scenarios for current and future land use were not evaluated for the following reasons:

- Future land use within the LDW is not expected to differ greatly from current land use. The use of the waterway for commercial and industrial purposes is expected to continue into the foreseeable future, although certain recreational activities that are consistent with these land uses may be more common in the future.
- Because site-specific parameters based on current land use practices are unavailable for many exposure parameters (see Section B.3.4), reasonable maximum values were selected. These values likely overestimate current exposure and can be assumed to represent future land use practices if the activities represented by the selected exposure scenarios (Table B-5) become more common in the future.

EPA (1989) also describes a central tendency (CT) scenario, which is intended to represent a more typical or likely scenario compared to the RME scenario. CT scenarios will be used only to a limited extent in this HHRA because the main objective of the scoping phase is to identify potential sites for early remedial action, which will be defined by risk quantification of the RME scenarios. In addition to the RME scenarios, a CT scenario was developed for the netfishing exposure pathway.

Summing risks from multiple exposure pathways may be reasonable if multiple pathways are relevant to the same receptor. EPA (1989) suggests that summing risks from multiple RME scenarios that do not occur simultaneously could be overly conservative. Consequently, risk estimates for the netfishing CT scenario were combined with risk estimates for the seafood consumption RME scenario (see Section B.5).

Table B-5 documents the decision process for selecting exposure pathways for quantification. Risk estimates could be made for the water recreation and industrial worker scenarios, but these estimates are likely to be much lower than estimates for the other scenarios (see Subappendix B.1). Consequently, the information gained from such quantification is not likely to be helpful in identifying potential early action sites, nor would quantification of these pathways be useful for the baseline RA since it is highly unlikely that they would represent the risk drivers upon which remedial decisions would be made. Nonetheless, risk estimates associated with swimming, as quantified by King County (1999b), are added to risk estimates for other exposure scenarios in the risk characterization section (B.5).

Table B-5. Selection of exposure pathways

Scenario Timeframe	Source Medium	Exposure Medium	Exposure Point	Activity	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
Current/ Future	sediment	sediment	Water recreation areas in LDW	water recreation	resident	adult	dermal ingestion	on-site on-site	qual qual	Exposure via swimming less than exposure via beach play
						child	dermal ingestion	on-site on-site	qual qual	Exposure via swimming less than exposure via beach play
		surface water				adult	dermal ingestion	on-site on-site	qual qual	Exposure via swimming less than exposure via beach play because of much lower exposure frequency
						child	dermal ingestion	on-site on-site	qual qual	Exposure via swimming less than exposure via beach play because of much lower exposure frequency
Current/ Future	sediment	sediment	LDW beaches	beach play in intertidal area	resident	adult	dermal ingestion	on-site on-site	qual qual	Adult's exposure during beach play likely to be less than child's exposure on a per kilogram body weight basis
						child	dermal ingestion	on-site on-site	quant quant	Residents may play at the shoreline near or adjacent to their houses
		surface water				adult	dermal ingestion	on-site on-site	qual qual	Exposure attributable to resuspended sediment in water column is likely to be insignificant compared to that from bedded sediment
						child	dermal ingestion	on-site on-site	qual qual	Exposure attributable to resuspended sediment in water column is likely to be insignificant compared to that from bedded sediment
Current/ Future	sediment	sediment	commercial netfishing locations in LDW, which potentially includes all LDW sediments	netfishing	worker	adult	dermal ingestion	on-site on-site	quant quant	Commercial fishermen are active at the site throughout the fishing season; nets contact the sediment
		surface water				adult	dermal ingestion	on-site on-site	qual qual	Exposure attributable to resuspended sediment in water column is likely to be insignificant compared to that from bedded sediment
Current/ Future	sediment	fish and shellfish tissue	fishing locations in the LDW	consumption of seafood	resident/ visitor/ worker	adult child	ingestion	on-site on-site	quant quant	Although available data suggest current seafood consumption from LDW is low, tribal members and public have harvest rights that extend well beyond current conditions

Scenario Timeframe	Source Medium	Exposure Medium	Exposure Point	Activity	Receptor Population	Receptor Age	Exposure Route	On-Site/ Off-Site	Type of Analysis	Rationale for Selection or Exclusion of Exposure Pathway
						adult child	dermal	on-site on-site	qual qual	Exposure via dermal pathway is unlikely
		sediment		fishing/ shellfishing in intertidal area		adult	dermal ingestion	on-site on-site	qual qual	Incidental exposure during fishing likely to be less than that assumed in beach play/work scenario; potential exposure during clamming to be reevaluated during Phase 2 RI
						child	dermal ingestion	on-site on-site	qual qual	Incidental exposure during fishing likely to be less than that assumed in beach play/work scenario; potential exposure during clamming to be reevaluated during Phase 2 RI
		surface water		fishing/ shellfishing in intertidal area		adult	dermal ingestion	on-site on-site	qual qual	Incidental exposure likely to be insignificant (see Subappendix B.1)
						child	dermal ingestion	on-site on-site	qual qual	Incidental exposure likely to be insignificant (see Subappendix B.1)
Current/ Future	sediment	sediment	Industrial facilities adjacent to LDW	typical industrial practices	worker	adult	dermal ingestion	on-site on-site	qual qual	Exposure much less than that evaluated in other sediment exposure scenarios
						child	dermal ingestion	on-site on-site	qual qual	No child industrial workers
		surface water				adult	dermal ingestion	on-site on-site	qual qual	Exposure much less than that evaluated in other scenarios
						child	dermal ingestion	on-site on-site	qual qual	No child industrial workers

B.3.3 CHEMICAL SCREENING AND EVALUATION

Many different chemicals have been analyzed in both sediment and tissue collected from the LDW. In accordance with EPA (1996a) guidelines, risk-based screening was conducted to determine which chemicals should be quantitatively evaluated in the Phase 1 HHRA. The decision process for identifying COPCs is shown in Figure B-2. Chemicals without RBCs are discussed in the uncertainty assessment. For detected[10] chemicals with RBCs, the maximum detected concentration was compared to the applicable RBC (step 3a). Detection limits were also evaluated relative to the RBCs for chemicals whose maximum detected concentrations did not exceed the RBCs, as shown in Figure B-2 (steps 4a and 4b). If a chemical was detected in greater than 10% of the samples, and those detected values never exceeded the RBC, the chemical was excluded from further analysis. For those chemicals with a detection frequency less than 10%, the number of times the detection limit exceeded the RBC was determined (the right side of Figure B-2; step 4b). If detection limits exceeded the RBC with a frequency greater than 10% (step 4b), that was considered sufficient uncertainty that the RBC could have been exceeded, and the chemical was retained as a COPC. Risks related to COPCs identified based on detection limit exceedances of RBCs alone were considered in the Uncertainty Assessment.

Some chemicals (e.g., carcinogenic PAHs, and polychlorinated dibenzodioxins and dibenzofurans) were evaluated as groups, rather than individual compounds, using a Toxicity Equivalency Factor approach. Additional details on the calculations associated with this approach are provided in Section B.2.2 and Table B-3.

Screening was conducted separately with data for intertidal sediment (beach play scenario), intertidal and subtidal sediment (netfishing scenario), and tissue chemistry (seafood consumption scenario). Specific analytical steps for evaluating background concentrations are described below in the media-specific sections. Tables describing the occurrence, distribution, and selection of COPCs are provided in Subappendix B.2.

[10] The distinction between a "detected" and "non-detected" concentration is generally based on whether the concentration reported by the laboratory exceeds the sample quantitation limit calculated by the laboratory. Additional details on what is meant by the generic term "detection limit" for specific sampling events summarized in the RI and risk assessments is provided in Section 4.2.1 of the RI report.

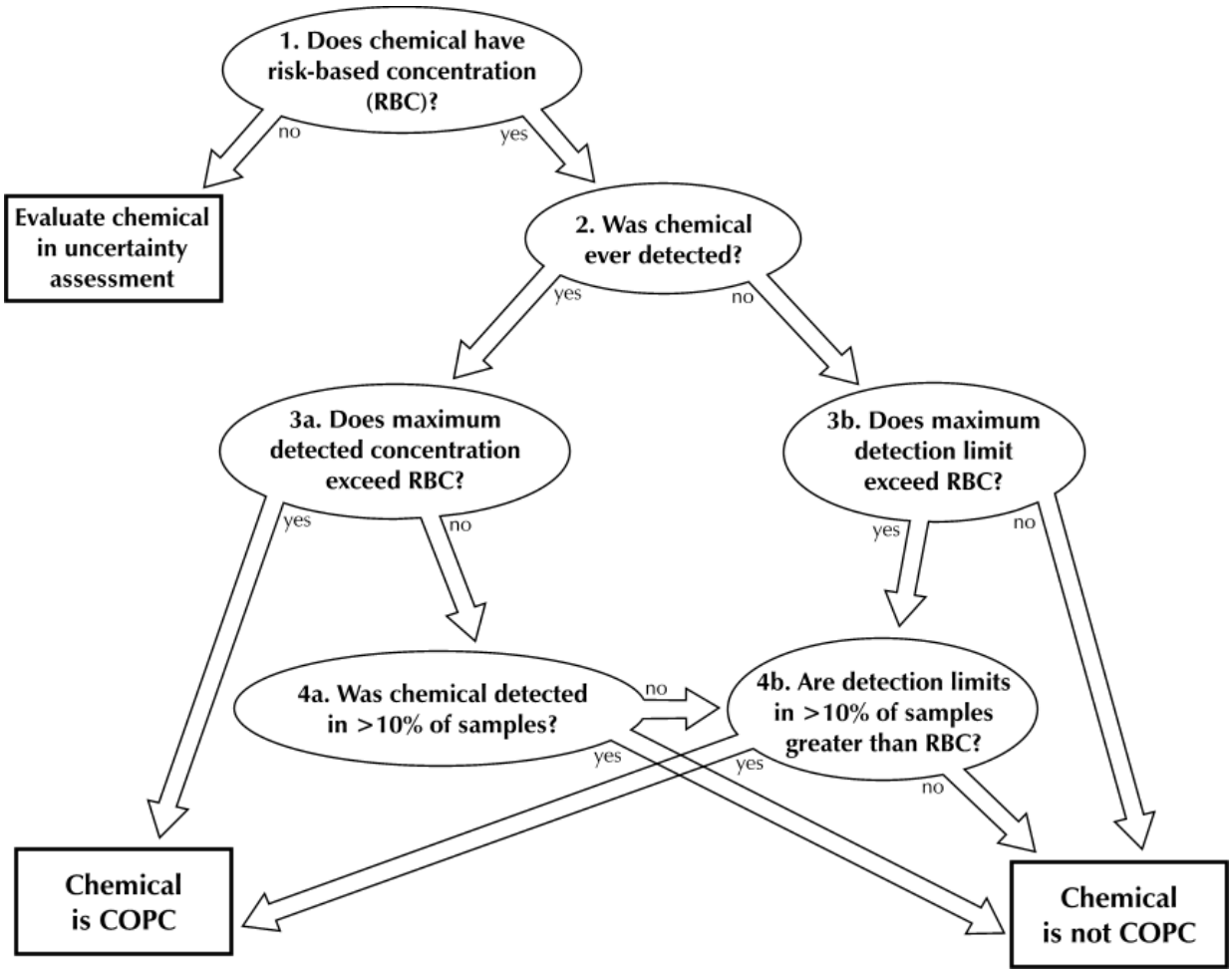


Figure B-2. COPC identification flowchart

B.3.3.1 Sediment

EPA has not developed RBCs specifically for sediment, but soil RBCs are generally applicable for scenarios which include incidental ingestion and dermal contact with sediment. The Washington State Model Toxics Control Act (MTCA) contains residential soil RBCs, but they are higher than EPA RBCs because of different exposure parameters. Consequently, EPA RBCs were used instead of MTCA RBCs because they are more health protective. RBCs[11] developed by EPA Region 9 (1999a) are widely used for screening at many locations throughout the country and will be used in this project as well.

EPA (1999a) contains soil RBCs for both industrial and residential scenarios. The equations used to calculate the RBCs incorporate exposure via ingestion, skin contact, and inhalation simultaneously. Region 9 RBCs for chemicals with noncarcinogenic effects were decreased by a factor of 10 to account for the target hazard quotients of 0.1

[11] EPA Region 9 uses the term Preliminary Remediation Goals (PRGs) for RBCs.

used in screening by EPA Region 10.[12] Both residential and industrial RBCs were utilized in the screening. Residential RBCs were applied to the beach play scenario and industrial RBCs were applied to the netfishing scenario.

Tables in Subappendix B.2 compare the maximum sediment concentrations for each chemical with the applicable RBC,[13] and include summary statistics such as detection frequency. For the netfishing scenario, data for subtidal and intertidal sediments were screened because nets may come in contact with sediments at both water depths. Only intertidal sediment chemistry data were screened for the beach play scenario.

The COPCs for the two sediment exposure scenarios are identified in Table B-6, which is excerpted from Tables 1 and 2 of Subappendix B.2.

Table B-6. Identification of COPCs for sediment exposure scenarios

Chemical	Netfishing Scenario		Beach Play Scenario	
	COPC?	Rationale	COPC?	Rationale
Detected chemicals				
2,3,7,8-TCDD TEQ	Yes	max detection > RBC	Yes	max detection > RBC
Aluminum	Yes	max detection > RBC	Yes	max detection > RBC
Antimony	Yes	max detection > RBC	Yes	max detection > RBC
Arsenic	Yes	max detection > RBC	Yes	max detection > RBC
Barium	No	max detection < RBC	Yes	max detection > RBC
Cadmium	Yes	max detection > RBC	Yes	max detection > RBC
cPAHs	Yes	max detection > RBC	Yes	max detection > RBC
Chromium	Yes	max detection > RBC	Yes	max detection > RBC
Copper	Yes	max detection > RBC	Yes	max detection > RBC
DDTs (total)	No	max detection < RBC	Yes	max detection > RBC
Dieldrin	Yes	max detection > RBC	Yes	max detection > RBC
Heptachlor epoxide	No	max detection < RBC	Yes	max detection > RBC
Hexachlorobenzene	No	max detection < RBC	Yes	max detection > RBC
Iron	Yes	max detection > RBC	Yes	max detection > RBC
Lead	Yes	max detection > RBC	Yes	max detection > RBC
Manganese	Yes	max detection > RBC	Yes	max detection > RBC
Mercury	No	max detection < RBC	Yes	max detection > RBC
Nickel	No	max detection < RBC	Yes	max detection > RBC
PCBs (total-calc'd)	Yes	max detection > RBC	Yes	max detection > RBC
Silver	No	max detection < RBC	Yes	max detection > RBC
Thallium	Yes	max detection > RBC	Yes	max detection > RBC

[12] EPA Region 10 recommends a target hazard quotient of 0.1; therefore, the EPA Region 9 PRGs (which are based on a target hazard quotient of 1) have been adjusted by dividing by 10 for this HHRA

[13] In some cases, surrogate RBCs were used if an RBC was not available for a particular COPC. For example, mercury concentrations were compared to the RBC for methyl mercury, chromium concentrations were compared to the RBC for hexavalent chromium, and thallium concentrations were compared to the RBC for thallium and compounds. All surrogate RBCs used are identified in the tables in Subappendix B.2.

Chemical	Netfishing Scenario		Beach Play Scenario	
	COPC?	Rationale	COPC?	Rationale
Vanadium	No	max detection < RBC	Yes	max detection > RBC
Zinc	No	max detection < RBC	Yes	max detection > RBC
Undetected chemicals				
1,2,3-Trichloropropane	Yes	24 of 44 detection limits > RBC	Yes	all detection limits > RBC
2-Nitroaniline	No	1 of 525 detection limits > RBC	Yes	68 of 184 detection limits > RBC
Benzidine	Yes	all detection limits > RBC	Yes	all detection limits > RBC
Bis(2-chloroethyl)ether	No	7 of 527 detection limits > RBC	Yes	29 of 186 detection limits > RBC
N-Nitrosodimethylamine	Yes	all detection limits > RBC	Yes	all detection limits > RBC
N-Nitroso-di-n-propylamine	No	12 of 527 detection limits > RBC	Yes	68 of 186 detection limits > RBC

Seventeen COPCs were identified for the netfishing scenario (Table B-6). Detection limits were generally lower than RBCs, with a few exceptions. Two chemicals (benzidine and N-nitrosodimethylamine) were identified as COPCs because all detection limits (neither chemical was ever detected) exceeded the applicable RBC. However, given the uncertainty surrounding these data, risk estimates for these two undetected chemicals are provided in the uncertainty assessment only. Additional comparisons of detection limits to RBCs are provided in Table 1 of Subappendix B.2. Table 1 also provides detail on the 287 other chemicals analyzed in subtidal and intertidal surface sediment that were not selected as COPCs. Some of these chemicals were not selected as COPCs because of the lack of toxicity data. These chemicals were qualitatively evaluated in the uncertainty assessment (Section B.6).

Twenty-nine COPCs were identified for the beach play scenario (Table B-6). Three chemicals (benzidine, N-nitrosodimethylamine, and 1,2,3-trichloropropane) were identified as COPCs because all detection limits exceeded the applicable RBC. The greater number of COPCs for the beach play scenario compared to the netfishing scenario reflects the lower RBCs for the beach play scenario, which are based on residential exposure for which more protective assumptions typically are used relative to industrial exposure scenarios. Table 2 of Subappendix B.2 provides details on the 241 other chemicals analyzed in intertidal surface sediment that were not selected as COPCs.

Because metals occur naturally in sediments in the absence of any human influence, an additional screen against data from background areas was performed (EPA 1989). Identification of specific areas that represent background has not been conducted for this project. In the absence of background samples collected for this project, sediment chemistry data for metals from non-urban areas were compiled for comparison purposes from the joint Ecology / PSAMP monitoring program for Central Puget Sound (Ecology 2000). Urban areas sampled in this study, such as Elliott Bay and Sinclair Inlet, were excluded from the data set used to estimate background concentrations. Tables 1 and 2 in Subappendix B.2 describe the locations included in the background concentration calculation. The maximum detected concentration for each metal in LDW surface sediments was compared to the maximum detected

concentration from the 52 samples collected from Central Puget Sound (Ecology 2000). In all cases, the LDW maximum was much higher than the Central Puget Sound (see Tables 1 and 2 in Subappendix B.2). Therefore, all the metals COPCs shown in Table B-6 were retained.

All COPCs identified in Table B-6 will be quantitatively evaluated in the HHRA. However, risk estimates for COPCs identified based solely on detection limits are provided in the Uncertainty Assessment.

B.3.3.2 Tissue

RBCs have been developed by EPA Region 9 (EPA 1996a) for soil and water, but not for fish tissue. MTCA, which contains risk-based cleanup levels for several media, considers uptake into fish from surface water but does not directly provide an RBC for fish tissue. EPA Region 3 (EPA 2001a) has developed RBCs for fish tissue. Exposure factors for fish RBCs include: target hazard quotient = 1, target risk = 10^{-6}, body weight = 70 kg, exposure frequency = 350 days / yr, exposure duration = 30 yr, and fish ingestion rate = 54 g / day (EPA 2001a). These exposure factors are consistent with Region 10 guidance, with the exception of the target hazard quotient. Region 10 recommends a target hazard quotient of 0.1 to account for cumulative effects from multiple chemicals and pathways (EPA 1996a). Region 3 RBCs for chemicals with noncarcinogenic effects were decreased by a factor of 10 to be consistent with guidance from EPA Region 10.

In addition to the modification described above for target hazard quotient, the Region 3 RBCs were modified to account for site-specific differences in consumption rate (84 g vs. 54 g, see Section B.3.4.1), exposure frequency (365 days vs. 350 days), body weight (79 kg vs. 70 kg), and exposure duration (55 yrs vs. 30 yrs, see Tables B-8d and B-8e). As a result of these site-specific modifications, the Region 3 RBCs based on a carcinogenic endpoint were multiplied[14] by 0.38 for screening use in this HHRA. The Region 3 RBCs based on non-carcinogenic endpoint were multiplied by 0.70 after application of the 0.1 factor described above for the hazard quotient modification. The difference between the correction factors for carcinogenic and non-carcinogenic endpoints is due to the fact that the RBC equation for carcinogenic endpoints is sensitive to exposure frequency and duration, while the RBC equation for non-carcinogenic endpoints is not.

Table 3 in Subappendix B.2 compares the maximum concentration for each chemical analyzed in the samples summarized in Table B-2 with the applicable RBC, and includes summary statistics such as detection frequency. Since only a few samples were available for each species, COPC screening was performed using the combined

[14] Using the ratios of site-specific exposure factors to default exposure factors used in the Region 3 RBC equation, the correction factor of 0.38 was derived by the following equation: (79 / 70 kg) / [(365 / 350 days / yr) x (55 / 30 yrs) x (84 / 54 g / day)]. The correction factor of 0.70 was derived by the following equation: [(79 / 70 kg) x (20,075 / 10,950 days)] / (84 / 54 g / day)

data set summarized in Table 3 in Subappendix B.2 rather than by the individual market basket fraction. The COPCs for this seafood consumption scenario are identified in Table B-7, which is excerpted from Table 3 of Subappendix B.2.

Table B-7. Identification of COPCs for the seafood consumption scenario

CHEMICAL	RATIONALE
Detected chemicals	
Arsenic	max detection > RBC
BEHP	max detection > RBC
cPAHs	max detection > RBC
Cadmium	max detection > RBC
Chromium	max detection > RBC
Copper	max detection > RBC
DDTs (total)	max detection > RBC
Lead	EPA recommends use of alternate toxicity evaluation method (i.e., IEUBK model)
Mercury	max detection > RBC
PCBs (total-calc'd)	max detection > RBC
Tributyltin as ion	max detection > RBC
Zinc	max detection > RBC
Undetected chemicals	
1,2-Diphenylhydrazine	all detection limits > RBC
3,3'-Dichlorobenzidine	all detection limits > RBC
Aldrin	all detection limits > RBC
Alpha-BHC	all detection limits > RBC
Benzidine	all detection limits > RBC
Beta-BHC	all detection limits > RBC
Bis(2-chloroethyl)ether	all detection limits > RBC
Bis(2-chloroisopropyl)ether	27 of 30 detection limits > RBC
Chlordane	all detection limits > RBC
Dieldrin	all detection limits > RBC
Gamma-BHC	11 of 20 detection limits > RBC
Heptachlor	all detection limits > RBC
Heptachlor epoxide	all detection limits > RBC
Hexachlorobenzene	all detection limits > RBC
Hexachlorobutadiene	27 of 30 detection limits > RBC
Pentachlorophenol	all detection limits > RBC
N-Nitrosodimethylamine	all detection limits > RBC
N-Nitroso-di-n-propylamine	all detection limits > RBC
Toxaphene	all detection limits > RBC

Note: IEUBK – Integrated Exposure Uptake Biokinetic Model for Lead in Children

Thirty-one COPCs were identified for the seafood consumption scenario (Table B-7). Twelve chemicals [total PCBs, cPAHs, mercury, arsenic, total DDTs, lead, BEHP, zinc, chromium, copper, cadmium, and tributyltin] were identified as COPCs based on detected concentrations; the remainder were never detected, but detection limits exceeded the applicable RBC. All COPCs identified in Table B-7 will be evaluated quantitatively in the HHRA. Table 3 of Subappendix B.2 provides details on the 91 other chemicals analyzed in tissue samples that were not selected as COPCs. In addition, approximately 180 chemicals measured in sediment were never analyzed in tissue. These chemicals are shown in Table 4 of Subappendix B.2. None of the chemicals listed in Table 4 were identified as sediment COPCs and none were identified by EPA (2000a) as important bioaccumulative compounds. Some of these chemicals might have been detected if they had been analyzed in tissue. Prior to Phase 2 data collection, additional evaluation of the need for tissue chemistry data for particular chemicals will be conducted.

Many of the chemicals detected in LDW tissue samples may also be detected in samples collected from background or reference areas. English sole have been collected from many locations throughout Puget Sound since 1989 as part of the Puget Sound Ambient Monitoring Program (West et al. 2001). Chemistry data from English sole filets collected in non-urban areas were compiled as part of this HHRA for comparison to site-specific English sole chemistry data. Average concentrations were calculated for 8 chemicals (total PCBs, arsenic, benzoic acid, benzyl alcohol, BEHP, copper, mercury, and total DDTs) detected in more than 10% of the samples from non-urban areas. For each of the 8 chemicals, the LDW concentration used for screening (i.e., the maximum concentration) was greater than the average concentration from the PSAMP database (see Table 3 in Subappendix B.2). Therefore, the background screen did not affect COPC selection. Additional discussion on background concentrations of arsenic is provided in the uncertainty assessment (Section B.6).

B.3.4 SELECTION OF EXPOSURE PARAMETERS

Exposure to contaminated sediment or fish and shellfish is expressed as the chronic daily intake (CDI). The CDI for oral ingestion is estimated as:

$$CDI_o = \frac{EPC \times IR \times FI \times EF \times ED \times CF}{BW \times AT} \qquad (\textit{Equation 1})$$

where:

CDI_o = chronic daily intake from oral exposure route (mg/kg-day)
EPC = chemical-specific exposure point concentration (mg/kg)
IR = ingestion rate (g/day)
FI = fractional intake of media derived from contaminated source (unitless)
EF = exposure frequency (days/year)
ED = exposure duration (years)

CF = conversion factor (kg/g)
BW = body weight (kg)
AT = averaging time (days)

The CDI equation for dermal exposure[15] is given below:

$$CDI_d = \frac{EPC \times ABS \times SA \times AF \times FI \times EF \times ED \times CF}{BW \times AT} \quad \textbf{(Equation 2)}$$

where:

CDI_d = chronic daily intake from dermal exposure route (mg/kg-day)
EPC = chemical-specific exposure point concentration (mg/kg)
ABS = dermal absorption factor (unitless)
SA = skin surface area exposed (cm^2)
AF = sediment to skin adherence factor by event (mg/cm^2-event)
FI = fractional intake of media derived from contaminated source (unitless)
EF = exposure frequency (events/year)
ED = exposure duration (years)
CF = conversion factor (kg/mg)
BW = body weight (kg)
AT = averaging time (days)

The exposure parameters used in the risk assessment for each exposure scenario are given in Tables B-8a through B-8i. These tables indicate the source for each exposure parameter. Additional explanation is provided in separate sections below for seafood consumption rates, dermal absorption factors, and exposure point concentrations. The combination of exposure scenario, pathway, and route described in each table is listed below for both RME and CT scenarios.

Table B-8a	Netfishing, incidental sediment ingestion, adult RME
Table B-8b	Netfishing, incidental sediment ingestion, adult CT
Table B-8c	Netfishing, dermal contact, adult RME
Table B-8d	Netfishing, dermal contact, adult CT

[15] Although chronic daily intake technically refers to oral exposure only, this term is also used in the HHRA to refer to dermal exposure, which is technically an absorbed dose, not intake. For this HHRA, the adjustment between orally administered doses and dermally administered doses was made by adjusting the oral toxicological benchmarks, as appropriate, according the EPA (2001b) guidance (see Section B.3.4.2 for additional details).

Table B-8a. Values used for daily intake calculations – incidental ingestion of sediment by adult tribal members during netfishing (RME scenario)

Scenario Timeframe: Current/Future

Medium: Sediment

Exposure medium: Sediment

Exposure Route	Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference	Intake Equation/ Model Name
Ingestion	EPC	Exposure point concentration in sediment	mg/kg	Table B-15	Section B.2.4.3	Chronic Daily Intake (CDI) (mg/kg-day) = EPC x IR-s x FI x EF x ED x CF x 1/BW-a x 1/AT
	IR-s	Incidental ingestion rate	g/day	0.050	EPA 1991a	
	FI	Fractional intake derived from source	unitless	1 [a]	–	
	EF	Exposure frequency	days/yr	119 [b]	Subappendix B.3	
	ED	Exposure duration	yrs	44 [b]	Subappendix B.3	
	CF	Conversion factor	kg/g	0.001	–	
	BW-a	Body weight-adult	kg	79	Suquamish Tribe 2000	
	AT-C	Averaging time – cancer	days	25,550	EPA 1989	
	AT-N	Averaging time – noncancer	days	16,060	EPA 1989	

Source: Standard Table 4 in EPA (1998a)

Note: RME = reasonable maximum exposure

[a] No available data suggest using any value other than a default of 1

[b] Value recommended by EPA based on conversation with Muckleshoot Tribe Assistant Harvest Manager. See Subappendix B.3 for more details on the derivation of this value.

Table B-8b. Values used for daily intake calculations – incidental ingestion of sediment by adult tribal members during netfishing (CT scenario)

Scenario Timeframe: Current/Future

Medium: Sediment

Exposure medium: Sediment

Exposure Route	Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference	Intake Equation/ Model Name
Ingestion	EPC	Exposure point concentration in sediment	mg/kg	Table B-15	Section B.2.4.3	Chronic Daily Intake (CDI) (mg/kg-day) = EPC x IR-s x FI x EF x ED x CF x 1/BW-a x 1/AT
	IR-s	Incidental ingestion rate	g/day	0.050	EPA 1991a	
	FI	Fractional intake derived from source	unitless	1 [a]	–	
	EF	Exposure frequency	days/yr	63 [b]	Subappendix B.3	
	ED	Exposure duration	yrs	29 [c]	–	
	CF	Conversion factor	kg/g	0.001	–	
	BW-a	Body weight-adult	kg	79	Suquamish Tribe 2000	
	AT-C	Averaging time – cancer	days	25,550	EPA 1989	
	AT-N	Averaging time – noncancer	days	10,585	EPA 1989	

Source: Standard Table 4 in EPA (1998a)

Note: CT = central tendency

[a] No available data suggest using any value other than a default of 1

[b] Value recommended by EPA based on conversation with Muckleshoot Tribe Assistant Harvest Manager. Selected value is duration of coho fishing season (see Subappendix B.3). Most individuals fish for coho.

[c] Value recommended by EPA based on conversation with Muckleshoot Tribe Assistant Harvest Manager. Selected value is best professional judgment assuming that fishing starts at age 16 and ends at age 45.

Table B-8c. Values used for daily intake calculations – dermal contact with sediment by adult tribal members during netfishing (RME scenario)

Scenario Timeframe: Current/Future

Medium: Sediment

Exposure medium: Sediment

Exposure Route	Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference	Intake Equation/ Model Name
Dermal	EPC	Exposure point concentration in sediment	mg/kg	Table B-15	Section B.2.4.3	Chronic Daily Intake (CDI) (mg/kg-day) = EPC x ABS x SA x AF x FI x EF x ED x CF x 1/BW-a x 1/AT
	ABS	Dermal absorption factor	unitless	Table B-13	Section B.2.4.2	
	SA	Skin surface area exposed	cm^2	3,600 [a]	EPA 2001a	
	AF	Adherence factor by event	mg/cm^2-event	0.2	EPA 1999b	
	FI	Fractional intake derived from source	unitless	1 [b]	–	
	EF	Exposure frequency	event/ year	119 [c]	Subappendix B.3	
	ED	Exposure duration	years	44 [c]	Subappendix B.3	
	CF	Conversion factor	kg/mg	0.000001	–	
	BW-a	Body weight-adult	kg	79	Suquamish Tribe 2000	
	AT-C	Averaging time – cancer	days	25,550	EPA 1989	
	AT-N	Averaging time – noncancer	days	16,060	EPA 1989	

Source: Standard Table 4 in EPA (1998a)

Note: RME = reasonable maximum exposure

[a] Recommended surface area for commercial/industrial worker. Assumes that head, hands, and forearms are exposed. Selected value represents sum of 50th percentile surface areas for men for these body parts; taken from Table 6-2 in EPA (1997a). Given the higher body weight of individuals surveyed in Suquamish Tribe (2000) compared to the general US population, the surface area values selected here for commercial/industrial workers may underestimate the surface area of tribal fishermen body parts. However, no conversion can be derived at the present time.

[b] No available data suggest using any value other than a default of 1

[c] Value recommended by EPA based on conversation with Muckleshoot Tribe Assistant Harvest Manager. See Subappendix B.3 for more details on the derivation of this value.

Table B-8d. Values used for daily intake calculations – dermal contact with sediment by adult tribal members during netfishing (CT scenario)

Scenario Timeframe: Current/Future

Medium: Sediment

Exposure medium: Sediment

Exposure Route	Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference	Intake Equation/ Model Name
Dermal	EPC	Exposure point concentration in sediment	mg/kg	Table B-15	Section B.2.4.3	Chronic Daily Intake (CDI) (mg/kg-day) = EPC x ABS x SA x AF x FI x EF x ED x CF x 1/BW-a x 1/AT
	ABS	Dermal absorption factor	unitless	Table B-13	Section B.2.4.2	
	SA	Skin surface area exposed	cm^2	3,600 [a]	EPA 2001a	
	AF	Adherence factor by event	mg/cm^2-event	0.02 [b]	EPA 2001b	
	FI	Fractional intake derived from source	unitless	1 [c]	–	
	EF	Exposure frequency	event/ year	63 [d]	Subappendix B.3	
	ED	Exposure duration	years	29 [e]	–	
	CF	Conversion factor	kg/mg	0.000001	–	
	BW-a	Body weight-adult	kg	79	Suquamish Tribe 2000	
	AT-C	Averaging time – cancer	days	25,550	EPA 1989	
	AT-N	Averaging time – noncancer	days	10,585	EPA 1989	

Source: Standard Table 4 in EPA (1998a)

Note: CT = central tendency

[a] Recommended surface area for commercial/industrial worker. Assumes that head, hands, and forearms are exposed. Selected value represents sum of 50^{th} percentile surface areas for men for these body parts; taken from Table 6-2 in EPA (1997). Given the higher body weight of individuals surveyed in Suquamish Tribe (2000) compared to the general US population, the surface area values selected here for commercial/industrial workers may underestimate the surface area of tribal fishermen body parts. However, no conversion can be derived at the present time.

[b] Default value for CT industrial workers from Exhibit 3-5 in RAGS Part E (EPA 2001b)

[c] No available data suggest using any value other than a default of 1

[d] Value recommended by EPA based on conversation with Muckleshoot Tribe Assistant Harvest Manager. Selected value is duration of coho fishing season (see Subappendix B.3). Most individuals fish for coho.

[e] Value recommended by EPA based on conversation with Muckleshoot Tribe Assistant Harvest Manager. Selected value is best professional judgment assuming that fishing starts at age 16 and ends at age 45.

Table B-8e. Values used for daily intake calculations – ingestion of seafood by adults in tribal/subsistence non-commercial (RME) scenario

Scenario Timeframe: Current/Future

Medium: Sediment

Exposure medium: Fish and shellfish tissue

Exposure Route	Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference	Intake Equation/ Model Name
Ingestion	EPC-p	Exposure point concentration in pelagic fish	mg/kg	Table B-14a	Section B.2.4.3	Chronic Daily Intake (CDI) (mg/kg-day) = [(EPC-p x IR-p) + (EPC-b x IR-b) + (EPC-c x IR-c) + (EPC-m x IR-m)] x FI x EF x ED-a x CF x 1/BW-a x 1/AT
	EPC-b	Exposure point concentration in benthic fish	mg/kg	Table B-14b	Section B.2.4.3	
	EPC-c	Exposure point concentration in crabs	mg/kg	Table B-14c	Section B.2.4.3	
	EPC-m	Exposure point concentration in mussels	mg/kg	Table B-14d	Section B.2.4.3	
	IR-p	Ingestion rate – pelagic fish	g/day	16	Section B.3.4.1	
	IR-b	Ingestion rate – benthic fish	g/day	15	Section B.3.4.1	
	IR-c	Ingestion rate – crabs	g/day	45	Section B.3.4.1	
	IR-m	Ingestion rate – mussels	g/day	7.8	Section B.3.4.1	
	FI	Fractional intake derived from source	unitless	1 [a]	–	
	EF	Exposure frequency	days/yr	365 [b]	EPA 1991a	
	ED-a	Exposure duration – adult	years	55	Subappendix B.4	
	CF	Conversion factor	kg/g	0.001	–	
	BW-a	Body weight-adult	kg	79	Suquamish Tribe 2000	
	AT-C	Averaging time – cancer	days	25,550	EPA 1989	
	AT-N	Averaging time – noncancer	days	20,075	EPA 1989	

Source: Standard Table 4 in EPA (1998a)

Note: RME = reasonable maximum exposure

[a] No available data suggest using any value other than a default of 1

[b] Default exposure frequency of 350 days/year modified to 365 to account for the fact that seafood consumption rate estimates are based on 365 days/year

Table B-8f. Values used for daily intake calculations – ingestion of seafood by children in tribal/subsistence non-commercial (RME) scenario

Scenario Timeframe: Current/Future

Medium: Sediment

Exposure medium: Fish and shellfish tissue

Exposure Route	Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference	Intake Equation/ Model Name
Ingestion	EPC-p	Exposure point concentration in pelagic fish	mg/kg	Table B-14a	Section B.2.4.3	Chronic Daily Intake (CDI) (mg/kg-day) = [(EPC-p x IR-p) + (EPC-b x IR-b) + (EPC-c x IR-c) + (EPC-m x IR-m)] x FI x EF x ED-a x CF x 1/BW-a x 1/AT
	EPC-b	Exposure point concentration in benthic fish	mg/kg	Table B-14b	Section B.2.4.3	
	EPC-c	Exposure point concentration in crabs	mg/kg	Table B-14c	Section B.2.4.3	
	EPC-m	Exposure point concentration in mussels	mg/kg	Table B-14d	Section B.2.4.3	
	IR-p	Ingestion rate – pelagic fish	g/day	3.9	Section B.3.4.1	
	IR-b	Ingestion rate – benthic fish	g/day	0.97	Section B.3.4.1	
	IR-c	Ingestion rate – crabs	g/day	40	Section B.3.4.1	
	IR-m	Ingestion rate – mussels	g/day	0.16	Section B.3.4.1	
	FI	Fractional intake derived from source	unitless	1 [a]	–	
	EF	Exposure frequency	days/yr	365 [b]	EPA 1991a	
	ED-c	Exposure duration – child	years	6	EPA 1991a	
	CF	Conversion factor	kg/g	0.001	–	
	BW-c	Body weight-child	kg	17	Suquamish Tribe 2000	
	AT-C	Averaging time – cancer	days	25,550	EPA 1989	
	AT-N	Averaging time – noncancer	days	2,190	EPA 1989	

Source: Standard Table 4 in EPA (1998a)

Note: RME = reasonable maximum exposure

[a] No available data suggest using any value other than a default of 1

[b] Default exposure frequency of 350 days/year modified to 365 to account for the fact that seafood consumption rate estimates are based on 365 days/year

Table B-8g. Values used for daily intake calculations – ingestion of seafood by Asian and Pacific Islander adults in subsistence non-commercial (RME) scenario

Scenario Timeframe: Current/Future

Medium: Sediment

Exposure medium: Fish and shellfish tissue

Exposure Route	Parameter Code	Parameter Definition	Units	Value	Rationale/ Reference	Intake Equation/ Model Name
Ingestion	EPC-p	Exposure point concentration in pelagic fish	mg/kg	Table B-14a	Section B.2.4.3	Chronic Daily Intake (CDI) (mg/kg-day) = [(EPC-p x IR-p) + (EPC-b x IR-b) + (EPC-c x IR-c) + (EPC-m x IR-m)] x FI x EF x ED-a x CF x 1/BW-a x 1/AT
	EPC-b	Exposure point concentration in benthic fish	mg/kg	Table B-14b	Section B.2.4.3	
	EPC-s	Exposure point concentration in shellfish	mg/kg	Table B-14e	Section B.2.4.3	
	IR-p	Ingestion rate – pelagic fish	g/day	2.7	Section B.3.4.1	
	IR-b	Ingestion rate – benthic fish	g/day	1.4	Section B.3.4.1	
	IR-s	Ingestion rate – shellfish	g/day	3.8 [a]	Section B.3.4.1	
	FI	Fractional intake derived from source	unitless	1 [b]	–	
	EF	Exposure frequency	days/yr	365 [c]	EPA 1991a	
	ED-a	Exposure duration – adult	years	30	EPA 1989	
	CF	Conversion factor	kg/g	0.001	–	
	BW-a	Body weight-adult	kg	63 [d]	EPA 1999c	
	AT-C	Averaging time – cancer	days	25,550	EPA 1989	
	AT-N	Averaging time – noncancer	days	10,950	EPA 1989	

Source: Standard Table 4 in EPA (1998a)

Note: RME = reasonable maximum exposure

[a] Ingestion rate used for shellfish is the 50th percentile consumption rate for all shellfish. This rate is assumed to represent a higher percentile consumer of just crabs and mussel, but still accounts for potential resource switching in the LDW where the presence of shellfish species typically consumed by this population has yet to be determined.

[b] No available data suggest using any value other than a default of 1

[c] Default exposure frequency of 350 days/year modified to 365 to account for the fact that seafood consumption rate estimates are based on 365 days/year

[d] Average body weight for all surveyed individuals from EPA (1999c)

Table B-8h. Values used for daily intake calculations – incidental ingestion of sediment by children during beach play (RME scenario)

Scenario Timeframe: Current/Future

Medium: Sediment

Exposure medium: Sediment

EXPOSURE ROUTE	PARAMETER CODE	PARAMETER DEFINITION	UNITS	VALUE	RATIONALE/ REFERENCE	INTAKE EQUATION/ MODEL NAME
Ingestion	EPC	Exposure point concentration in sediment	mg/kg	Table B-16	Section B.2.4.3	Chronic Daily Intake (CDI) (mg/kg-day) = (EPC x IR-s x FI x EF x CF x 1/AT) x **G** (ED_i x 1/BW_i-c)
	IR-s	Incidental sediment ingestion rate	g/day	0.200	EPA 1991a	
	FI	Fractional intake derived from source	unitless	1 [a]	–	
	EF	Exposure frequency	days/yr	41 [b]	–	
	ED_i	Exposure duration – by age class	years	[c]	EPA 1991a	
	CF	Conversion factor	kg/g	0.001	–	
	BW_i-c	Body weight-child	kg	[d]	EPA 1997	
	AT-C	Averaging time – cancer	days	25,550	EPA 1989	
	AT-N	Averaging time – noncancer	days	2,190	EPA 1989	

Source: Standard Table 4 in EPA (1998a)

Note: RME = reasonable maximum exposure

[a] No available data suggest using any value other than a default of 1

[b] Assumes 3 days/wk during school vacation of 84 days (mid-June to first week of September) and 1 day/wk for 5 weeks from mid-September to end of October

[c] Doses for 6 different age classes are calculated separately: 2 to 3, 3 to 4, 4 to 5, 5 to 6, 6 to 7, and 7 to 8. Total exposure duration is 6 years, but duration for each year class is 1 year.

[d] Body weights for each age class are means for boys and girls combined from Table 7-3 in EPA (1997)

Age class	BW_i (kg)	Age class	BW_i (kg)
2 to 3	13.3	5 to 6	19.7
3 to 4	15.3	6 to 7	22.6
4 to 5	17.4	7 to 8	24.9

Table B-8i. Values used for daily intake calculations – dermal contact with sediment by children during beach play (RME scenario)

Scenario Timeframe: Current/Future

Medium: Sediment

Exposure medium: Sediment

EXPOSURE ROUTE	PARAMETER CODE	PARAMETER DEFINITION	UNITS	RME VALUE	RME RATIONALE/ REFERENCE	INTAKE EQUATION/ MODEL NAME
Dermal	EPC	Exposure point concentration in sediment	mg/kg	Table B-16	Section B.2.4.3	Chronic Daily Intake (CDI) (mg/kg-day) = (EPC x ABS x AF x FI x EF x CF x 1/AT) x **G** (SA_i x ED_i x 1/BW_i-c)
	ABS	Dermal absorption factor	unitless	Table B-13	Section B.2.4.2	
	SA_i	Skin surface area exposed – by age class	cm^2	[a]	EPA 1997	
	AF	Adherence factor by event	mg/cm^2-event	0.2	EPA 1999b	
	FI	Fractional intake derived from source	unitless	1 [b]	–	
	EF	Exposure frequency	events /year	41 [c]	–	
	ED_i	Exposure duration – by age class	years	[d]	EPA 1991a	
	CF	Conversion factor	kg/mg	0.000001	–	
	BW_i-c	Body weight, child – by age class	kg	[e]	EPA 1997	
	AT-C	Averaging time – cancer	days	25,550	EPA 1989	
	AT-N	Averaging time – noncancer	days	2,190	EPA 1989	

Source: Standard Table 4 in EPA (1998a) Note: RME = reasonable maximum exposure

[a] Assumes that 35% of the total body surface area is exposed, roughly corresponding to an individual wearing a short-sleeve shirt and short pants, but no shoes (EPA 1992a). Body surface area data taken from Tables 6-6 and 6-7 in EPA (1997). Values below are means of the 50th percentile surface areas (total surface area x 0.35) between male and female children.

Age class	SA_i (cm^2)	Age class	SA_i (cm^2)	Age class	SA_i (cm^2)
2 to 3	2,069	4 to 5	2,445	6 to 7	2,900
3 to 4	2,224	5 to 6	2,674	7 to 8	3,133

[b] No available data suggest using any value other than a default of 1

[c] Assumes 3 days/wk during school vacation of 84 days (mid-June to first week of September) and 1 day/wk for 5 weeks from mid-September to end of October

[d] Doses for 6 different age classes are calculated separately: 2 to 3, 3 to 4, 4 to 5, 5 to 6, 6 to 7, and 7 to 8. Total exposure duration is 6 years, but duration for each year class is 1 year.

[e] Body weights for each age class are means for boys and girls combined from Table 7-3 in EPA (1997)

Age class	BW_i (kg)	Age class	BW_i (kg)
2 to 3	13.3	5 to 6	19.7
3 to 4	15.3	6 to 7	22.6
4 to 5	17.4	7 to 8	24.9

B.3.4.1 Seafood consumption rates (IR)

The primary documented use of the LDW as a fishery is the commercial salmon fishery conducted by the Muckleshoot Tribe. However, as indicated in Section B.2.1.2, because of the migratory nature of salmon, chemicals found in salmon tissue are not likely to be representative of exposure of salmon to chemicals present in the LDW and therefore salmon consumption is not considered here. The LDW is also within a much larger resource area utilized by the Suquamish Tribe. Additionally, some information suggests that other relatively high fish-consuming populations may use the LDW for at least part of their fish collection. Available data suggest that use of the LDW for collection of seafood other than salmon is limited. For the purposes of this HHRA, health protective assumptions have been made regarding potential current and future seafood consumption.

Four seafood consumption surveys have been conducted within the last five years that included the LDW in a much larger survey area (Toy et al. 1996, King County 1999b, EPA 1999c, Suquamish Tribe 2000). Information from these surveys was used to select consumption rates for both adults and children who might consume fish from the LDW now or in the future. Consumption rates were estimated in all of the four surveys and each survey identified populations thought to be representative of those who might consume fish from the LDW. These populations may consume more fish and shellfish than the average American recreational angler (EPA 1997a). Thus, although data are not specific to the LDW, they provide a health protective means to estimate seafood consumption rates that are consistent with the intent of the RME scenario as defined by EPA (1989).

Table B-9 compares the characteristics of each survey cited above. Only the survey conducted by Suquamish Tribe (2000) contained information on all five desirable categories described in Table B-9. Very few of the respondents in Suquamish Tribe (2000) indicated that they fish in the WDFW catch area that includes the Duwamish River. The Asian and Pacific Islander (API) populations studied in EPA (1999c) may consume fish and shellfish they collect from the Duwamish River, but the survey did not include geographic distinctions that would make it possible to determine the fishing frequency in the LDW compared to other areas in King County over which the survey was based. However, information collected by WDFW enforcement personnel (Frame 2002) indicate that individuals of Asian and Pacific Islander ethnicity are more commonly encountered engaging in non-commercial fishing in the LDW than any other ethnic group. Although there is uncertainty regarding the degree of seafood consumption by any group within the LDW, this HHRA has provided estimates for both tribal and Asian and Pacific Islander populations as these groups consume more seafood than the general public.

Table B-9. Seafood consumption survey characteristics

CHARACTERISTIC	TOY ET AL. (1996)	EPA (1999c)	KING COUNTY (1999b)	SUQUAMISH TRIBE (2000)
Target population	Tulalip and Squaxin Island Tribes	Asian and Pacific Islanders	recreational anglers using Elliott Bay and LDW	Suquamish Tribe
Survey method	interview	interview	interview	interview
Number of respondents	259	202	1,183	123
Included LDW study area	no	yes	yes	yes
Includes consumption estimates for specific seafood groups	yes	yes	no	yes
Includes consumption estimates for specific species	no	yes	no	yes
Included information on source of fish and shellfish consumed	yes	yes	yes	yes
Included children	yes	no	yes	yes

Consumption data from both the Suquamish Tribe (2000) and the Asian and Pacific Islander study (EPA 1999c) were selected for use in the exposure assessment. Although the Suquamish consumption rates are higher than API rates, cooking and consumption practices such as fish and shellfish preparation may be very different between the two groups. For example, the Asian and Pacific Islanders apparently consume crab hepatopancreas and whole fish more frequently than do Suquamish tribal members. Quantitative analyses of risks associated with seafood consumption are presented for both tribal (non-commercial) and API scenarios in the risk characterization section (Section B.5).

The Suquamish Tribe (2000) study included consumption data for 55 species in 7 categories. Although one or more individuals consumed each of the 55 species within the Usual and Accustomed fishing area of the Suquamish Tribe, not all these species and species groups can be found within the LDW. The most highly consumed species group was salmon. Salmon are found in the LDW, but consumption of salmon does not represent an exposure pathway for this HHRA because the adult fish do not feed as they return to the LDW; thus, there is no linkage between potentially contaminated sediments in the LDW and consumption of these fish.

The shellfish group used by Suquamish Tribe (2000) includes approximately 20 species or species groups, all of which are obligate saltwater dwellers. Some species may be found in the LDW because of the saltwater wedge associated with the tidal cycle, but the limited available data suggest that they are rare or nonexistent. Additional research on the ability of the LDW to sustain harvestable populations of shellfish will be conducted during the Phase 2 RI. Both crabs and mussels are potentially present and are considered in the HHRA.

The relevance of each species group defined by Suquamish Tribe (2000) to the LDW and this HHRA is shown in Table B-10.

Table B-10. Rationale for including seafood groups used in Suquamish seafood consumption study (Suquamish Tribe 2000) in the Phase 1 HHRA

SEAFOOD GROUP	MEMBERS	INCLUDE IN PHASE 1 HHRA?	REASON
A – Fish	Salmon	No	Adult salmon common in LDW, but these fish do not feed when migrating upstream; not relevant for assessing chemicals in LDW sediment because of lack of linkage between chemicals in LDW sediments and those found in adult salmon tissues
B – Fish	Smelt, herring	Yes	Longfin smelt common in the LDW
C – Fish	Cod, perch, etc.	Yes	Perch common in the LDW
D – Fish	Halibut, sole, rockfish, snappers	Yes	English sole and other benthic species common in the LDW
E – Shellfish	Bivalves, snails, shrimp, crab	Yes (crab and mussels only)	Some marine shellfish species (crab and mussels) are present in parts of the LDW
F – Other fish	Cabezon, trout, tuna, groupers, sardines, mackerel, shark	No	Harvestable numbers not present in LDW
G – Other shellfish	Abalone, squid, lobster, octopus, limpets	No	Harvestable numbers not present in LDW

The API study (EPA 1999c) also estimated consumption rate data for individual fish and shellfish species, but only the seafood group rates were published in the document. The applicability of the seafood groups used in the API study to this HHRA is described in Table B-11.

Table B-11. Rationale for including seafood groups used in API seafood consumption study (EPA 1999c) in the Phase 1 HHRA

SEAFOOD GROUP	MEMBERS	INCLUDE IN PHASE 1 HHRA?	REASON
Anadromous fish	Salmon, steelhead	No	Adult salmon common in LDW, but these fish do not feed when migrating upstream; not relevant for assessing chemicals in LDW sediment because of lack of linkage between chemicals in LDW sediments and those found in adult salmon tissues
Pelagic fish	Tuna, cod, rockfish etc	Yes	Surfperch common in the LDW
Freshwater fish	Catfish, bass, carp etc.	No	Obligate freshwater fish are rare in the LDW
Bottom fish	Halibut, sole, etc	Yes	English sole and other benthic species common in the LDW
Seaweed/ kelp	Kelp	No	Kelp species are rare in the LDW
Shellfish	Bivalves, snails, shrimp, crab	Yes	Some marine shellfish species (crab and mussels) are present in parts of the LDW; the shellfish group is used as a surrogate for these species in this HHRA

Windward (2000) conducted a reconnaissance survey to document the presence or absence of clams in the intertidal zone of several areas[16] within the LDW. Abundance was highest at Kellogg Island, but one or more clams were found at each sampling site. Five different species were identified: Eastern soft-shell clam (*Mya arenaria*), butter clam (*Saxidomis giganteus*), sand clam (*Macoma secta*), bent-nose clam (*Macoma nasuta*), and the inconspicuous macoma (*Macoma inconspicua*). Individuals from the latter three species were small (maximum shell width of 54 mm). Because of the preliminary nature of this reconnaissance survey, no voucher collection of specimens was acquired so that taxonomic identifications could be verified. The Muckleshoot Tribe has raised concerns regarding some of the identifications presented in Windward (2000). Any additional sampling conducted in Phase 2 will include a voucher collection so taxonomy can be verified.

WDFW has tables that relate allowable harvest to clam densities, but data are insufficient to calculate clam densities in the LDW. Although intertidal habitat enhancement projects within the LDW may occur in the future, it is unlikely these improvements will greatly affect the salinity regime within the LDW. With the exception of the non-native species *Mya arenaria*, which is tolerant of relatively low salinities (Snelgrove et al. 1999), it appears that salinity is an important controlling factor for clam abundance. Additional research on the clam abundance and habitat characteristics will be conducted during the Phase 2 RI.

Since the likelihood that harvestable populations of clams exist or will exist in the LDW is uncertain, and site-specific tissue chemistry data are not available for clams, consumption rates associated with clams are not included in this HHRA. Alternate exposure scenarios that include consumption of clams are presented in the uncertainty analysis (Section B.6). Consumption associated with clams will be revisited in the Phase 2 HHRA following collection of additional site-specific data.

Multiple consumption rates were given in Suquamish Tribe (2000) and EPA (1999c) for each seafood group in Tables B-10 and B-11, respectively. These surveys also reported the source for each seafood group. Survey respondents were asked to choose among the following sources for each seafood group: groceries, harvest within Puget Sound, harvest outside Puget Sound, restaurants, and other / unknown. The fraction of fish and shellfish harvested from Puget Sound was used to calculate seafood consumption rates for this HHRA (Table B-12). Following the approach used by Environmental Solutions Group (1999), consumption estimates were made separately for pelagic fish, benthic fish, crab, and mussels. This "market basket" approach is useful in that EPCs for specific species groups can be tied to consumption rates specific to that group. Consumption rates selected for the adult API RME scenario were recommended by EPA (2002c) in their comments on the draft HHRA.

[16] Terminal 105, Kellogg Island, Slip 2, Slip 4, and Duwamish Yacht Club.

The consumption rates in Table B-12 were used to calculate the chronic daily intake for both adults and children using the equations presented in Tables B-8e, B-8f, and B-8g. Although the equations presented in the exposure assessment are intended to represent a single hypothetical individual for each scenario, the use of 90th or 95th percentile consumption rate estimates for multiple species or species groups may represent a higher percentile for any particular individual since individual preferences between groups are not equivalent. Any overestimation of consumption rates attributed to summing 90th or 95th percentiles may be offset by the fact that these rates included non-consumers. EPA's (2001d) analysis indicates that combining consumption rates for individual species groups could overestimate the total consumption rate for an individual by approximately 9%. This health protective approach is appropriate for the Phase 1 HHRA, but the raw data from the consumption survey may be utilized in the Phase 2 HHRA to yield a more statistically appropriate upper-end consumption rate estimate.

Table B-12. Seafood consumption rates (g/day) used in the Phase 1 HHRA

Seafood Group	Adult – RME Scenario (Tribal Non-commercial)	Child - RME Scenario (Tribal Non-commercial)	Adult – RME (API)
Pelagic fish	16 (1)	3.9 (5)	2.7 (9)
Benthic fish	15 (2)	0.97 (6)	1.4 (10)
Shellfish (all species)	n/a	n/a	3.8 (11)
Shellfish (crab only)	45 (3)	40 (7)	n/a
Shellfish (mussels only)	7.8 (4)	0.16 (8)	n/a

Data from sources 1 to 8 from Suquamish Tribe (2000), groups referred to in footnotes below are described in Table B-10; sources 9 to 11 from EPA (1999c), groups referred to in footnotes below are described in Table B-11. Application of these data is consistent with recommendations made by EPA in their comment letters dated January 23, 2002 and September 16, 2002.

Sources (in parentheses above):

(1) = 95th percentile consumption rate for Groups B and C (Table C-1 – consumers and non-consumers) x 79 kg body weight x 0.26 (95% UCL of mean fraction of Groups B and C derived from harvest within Puget Sound – Table T-18)

(2) = 95th percentile consumption rate for Group D (Table C-1 – consumers and non-consumers) x 79 kg body weight x 0.30 (95% UCL of mean fraction of Group D derived from harvest within Puget Sound – Table T-18)

(3) = 95th percentile consumption rate for Dungeness and red rock crab, and crab consumed at gatherings (Table C-1 – consumers and non-consumers) x 79 kg body weight x 0.86 (95% UCL of mean fraction of Group E derived from harvest within Puget Sound – Table T-18)

(4) = 95th percentile consumption rate for mussels (Table C-1 – consumers and non-consumers) x 79 kg body weight x 0.86 (95% UCL of mean fraction of Group E derived from harvest within Puget Sound – Table T-18)

(5) = 95th percentile consumption rate for Groups B and C (Table C-6 – consumers and non-consumers) x 17 kg body weight x 0.26 (95% UCL of mean fraction of Groups B and C derived from harvest within Puget Sound – Table T-18).

(6) = 95th percentile consumption rate for Group D (Table C-6 – consumers and non-consumers) x 17 kg body weight x 0.30 (95% UCL of mean fraction of Group D derived from harvest within Puget Sound – Table T-18)

(7) = 95th percentile consumption rate for Dungeness and red rock crabs (Table C-6 – consumers and non-consumers) x 17 kg body weight x 0.86 (95% UCL of mean fraction of Group E derived from harvest within Puget Sound – Table T-18)

(8) = 95th percentile consumption rate for mussels (Table C-6 – consumers and non-consumers) x 17 kg body weight x 0.86 (95% UCL of mean fraction of Group E derived from harvest within Puget Sound – Table T-18)

(9) = 90th percentile consumption rate for pelagic fish group across all ethnic groups in API study (Appendix M-3 – consumers and non-consumers) x 63 kg body weight x 0.052 (95% UCL of mean fraction of pelagic fish group derived from harvest within King County)

(10) = 90th percentile consumption rate for bottom fish group across all ethnic groups in API study (Appendix M-3 – consumers and non-consumers) x 63 kg body weight x 0.084 (95% UCL of mean fraction of bottom fish group derived from harvest within King County)

(11) = 50th percentile consumption rate for all shellfish species for all ethnic groups in API study (Appendix M-3 – consumers and non-consumers) x 63 kg body weight x 0.12 (95% UCL of mean fraction of shellfish group derived from harvest within King County)

B.3.4.2 Dermal absorption factor (ABS)

The dermal absorption factor (ABS) refers to the fraction of the chemical in sediment applied to the skin surface that is absorbed into the bloodstream. Many studies have focused on this topic, but there is considerable uncertainty regarding chemical-specific values (EPA 1992a). EPA Region 10 Resource Conservation and Recovery Act (RCRA) guidance (EPA 1998b) includes dermal absorption factors for various chemicals, including all the COPCs identified in Section B.3.3.1 (Table B-13). EPA (2001b) has developed supplemental draft guidance for dermal risk assessment that provides similar ABS values to those provided in Table B-13, but this document includes ABS values for only two metals, arsenic and lead. The draft guidance document states that speciation of inorganic substances is crucial to dermal absorption and data are insufficient to derive default values for other inorganic substances. Nonetheless, default values of 0.01 are used for inorganic substances in this HHRA until such time as the EPA guidance is finalized.

The toxicological benchmarks presented in Section B.3 are based on orally administered doses, which are not necessarily equivalent to dermally absorbed doses. Although EPA (2001b) provides a summary of gastrointestinal absorption data for many chemicals, data are not available for all chemicals evaluated. In the case of organic chemicals evaluated in this HHRA, absorption via the oral route is greater than 50%, indicating that no conversion of the oral toxicity value is needed (EPA 2001b). Thus, for this Phase 1 HHRA, a gastrointestinal absorption factor of 1 was used for organic chemicals; oral toxicological benchmarks were applied without modification. For some inorganic chemicals, adjustment factors shown in Table B-13 were applied to the oral reference dose.

Table B-13. Dermal absorption factors (ABS) for the dermal exposure pathway

PARAMETER	ABS (UNITLESS)[a]	ORAL ABSORPTION ADJUSTMENT[b]
1,2,3-Trichloropropane	0.1	none
2,3,7,8-TCDD TEQ	0.03	none
Aluminum	0.01	none
Antimony	0.01	RfD * 0.15
Arsenic	0.03	none
Barium	0.01	RfD * 0.07

PARAMETER	ABS (UNITLESS)[a]	ORAL ABSORPTION ADJUSTMENT[b]
Benzidine	0.1	none
Cadmium	0.001	RfD * 0.025
cPAHs	0.13	none
Chromium	0.01	RfD * 0.025
Copper	0.01	none
DDTs (total)	0.03	none
Dieldrin	0.1	none
Heptachlor epoxide	0.1	none
Hexachlorobenzene	0.1	none
Iron	0.01	none
Lead	0.01	none
Manganese	0.01	RfD * 0.04
Mercury	0.01	RfD * 0.07
Nickel	0.01	RfD * 0.04
N-Nitrosodimethylamine	0.1	none
PCBs (total-calc'd)	0.14	none
Silver	0.01	RfD * 0.04
Thallium	0.01	none
Vanadium	0.01	RfD * 0.026
Zinc	0.01	none

[a] Based on Region 10 RCRA guidance (EPA 1998b). RAGS E guidance (EPA 2001b) indicates the only inorganic chemicals to be evaluated in the dermal pathway are arsenic and cadmium and that the dermal absorption (ABS) should be 0.03 for arsenic and 0.001 for cadmium

[b] Based on RAGS E (EPA 2001b) guidance

B.3.4.3 Exposure point concentrations

B.3.4.3.1 Tissue

The exposure point concentrations (EPCs) in fish and shellfish were calculated according to EPA (1989) guidelines. The mean chemical concentration in fish and shellfish is the most appropriate estimate for the exposure point of interest in this HHRA. EPA (1989) recommends that the mean be represented using a one-sided 95% upper confidence limit (UCL) on the mean. In cases where the variability in the data is extremely high, the maximum value may be exceeded by the 95% UCL on the mean. In these cases, EPA (1989) recommends that the maximum value be used.

Given the small number of data points available for calculating EPCs for tissue, it is difficult to determine the underlying distribution of the data so that the appropriate formulas can be used to compute the 95% UCL. Based on work by Singh et al (1997) and Schulz and Griffen (1999), recent EPA (2002a) guidance recommends the use of non-parametric methods for the calculation of 95% UCLs on the mean when the data distribution cannot be reliably determined. This may occur when sample sizes are small (i.e., less than 30), data sets are highly skewed, data sets contain outliers, or

when data sets are comprised of a mixture of two or more subpopulations. For the Phase 1 risk assessment, where fewer than six samples were available the maximum value was used as the EPC; for samples with a minimum of six observations a non-parametric bootstrapping method was used to estimate the 95% UCLs. The bootstrapping method applied here is referred to as the bootstrap-*t* method (described briefly below and explained in greater detail in Manly 1997 and EPA 2002a). The bootstrap-*t* estimates of the UCL on the mean are more health protective than UCLs constructed using other nonparametric procedures (Singh et al 1997, EPA 2002a, and Manly 1997). Simulations performed by Manly (1997) showed that the bootstrap-*t* confidence intervals on the mean had the best coverage among other bootstrap procedures.

Bootstrapping is a statistical resampling procedure that uses the sample data as the population to construct confidence limits around the true underlying population parameters. Bootstrapping assumes that the sample data are representative of the underlying population, so random sampling is a pre-requisite for appropriate application of this method. Where random sampling was not utilized (e.g., collection of the mussel data) potential biases resulting from the bootstrap procedure may result. For example, because the mussel data were collected from areas of known or suspected contamination the application of the bootstrap method to these sample results is likely to overestimate concentrations for the LDW as a whole.

Bootstrapping procedures entail resampling, with replacement,[17] from the observed sample population of size n. Each time the sample population is resampled, a summary statistic (e.g., mean or standard deviation) of the bootstrapped sample is computed and stored. After repeating this procedure many times, a summary of the bootstrapped statistics is used to construct the confidence limit. For the bootstrap-*t* method, the bootstrapped statistic (T) is a pivotal statistic, which means that the distribution of T is the same for all values of the mean. The pivotal statistic T is defined as:

$$T = \frac{\bar{x} - \mu}{SE(\bar{x})} \qquad (Equation\ 3a)$$

where μ is the true population mean and the values $\bar{x}$ and $SE(\bar{x})$ are sample estimates of the mean and the standard error of the mean, respectively. The 5th percentile of the T distribution, $T_{0.05}$, satisfies the following probability statement

$$\Pr[T_{0.05} < \frac{\bar{x} - \mu}{SE(\bar{x})}] = 0.95 \qquad (Equation\ 3b)$$

Rearranging this equation yields 95% confidence in the following inequality:

[17] Replacement means that once sampled, it remains in the data set, eligible to be sampled again; a given sample may be selected more than once during a given bootstrapping replicate

$$\mu < \bar{x} - T_{0.05} SE(\bar{x}) \qquad \textit{(Equation 3c)}$$

The right side of equation 3c represents the 95% upper confidence limit on the population mean. Bootstrapping is used to estimate the $T_{0.05}$ value while the other parameters are estimated from the original sample.

The specific steps used to compute the 95% UCL using the bootstrap-*t* method are described below.

1. Bootstrap (i.e., sample with replacement from the original sample population of size n) 1000 samples of size n and compute the T statistic for each bootstrapped sample. $T_{B,i}$ is the bootstrapped-*t* value computed from the i^{th} bootstrap sample, defined by the equation 3d:

$$T_{B,i} = \frac{\bar{x}_{B,i} - \bar{x}}{SE(\bar{x}_{B,i})} \qquad \textit{(Equation 3d)}$$

   where $\bar{x}_{B,i}$ and $SE(\bar{x}_{B,i})$ are the mean and the standard error of the mean (the standard deviation divided by the square root of n) computed for the i^{th} bootstrapped sample, and $\bar{x}$ is the original sample mean. This step yields 1000 values of the bootstrapped-*t* statistic which comprise the "bootstrap-*t* distribution".

2. Find $T_{0.05,}$ the 5th percentile of the bootstrap-*t* distribution. This value satisfies the equation $\Pr[T_B < T_{0.05}] = 0.05$, where T_B are the values in the bootstrap-*t* distribution. For 1000 bootstrap samples, $T_{0.05}$ is the 50th ordered value in the bootstrap-*t* distribution.

3. Applying equation 3c using the value $T_{0.05}$ found in Step 2 gives the bootstrap-*t* estimate of the 95% UCL on the population mean, i.e.,

$$\text{95\% UCL} = \bar{x} - T_{0.05} SE(\bar{x}) \qquad \textit{(Equation 4)}$$

   where $\bar{x}$ and $SE(\bar{x})$ are the mean and the standard error of the mean (the standard deviation divided by the square root of n) computed from the original sample. Note that the $T_{0.05}$ value in Equation 4 is negative, thus yielding an upper bound on the mean.

Mussel samples were not collected using strict random sampling; the data used in this risk assessment suggest an unbalanced stratified sampling design with strata defined by location and season. The bootstrapping procedure used to estimate 95% UCLs was applied to the pooled set of mussel data without regard for differences in chemical concentration among the different strata. This simplified approach treats the data as one homogeneous population rather than a mixture of several subpopulations defined by location and season. However, since the original study (King County 1999b) targeted sampling locations adjacent to potential sources of some chemicals (i.e., CSOs), the UCLs presented here represent a health protective estimate of exposure from the population of mussels found within the entire Duwamish site for a number

of the COPCs. Ignoring possible strata differences will result in an inflated variance estimate for the data. However, ignoring strata differences is also consistent with an exposure scenario whereby a consumer may harvest mussels from any location and at any time. Evaluation of the data by location and season (i.e., including the strata differences) would be consistent with a consumer harvesting mussels exclusively from one location and during one season. Additional analysis of the impacts to risk estimates from the statistical treatment of the mussel data is provided in the uncertainty assessment (Section B.6.1.2).

For tissue samples with undetected values, one-half the detection limit was substituted to calculate the 95% UCL on the mean. The EPC was set equal to one-half the maximum detection limit if the chemical was never detected for a given species. Where the estimated 95% UCL on the mean for the composite samples exceeded the maximum detected concentration, the maximum detected concentration was selected for the EPC. Samples with many undetected values reported with the same detection limit can result in bootstrap samples with zero or close to zero variance (since sampling occurs with replacement) yielding an undefined T statistic. Therefore, when detection frequencies were less than 10%, a 95% UCL on the mean was not computed and the EPC was set at the maximum detected concentration.

The EPCs and other summary statistics are provided in Tables B-14a through B-14d for each of the four species, and in Table B-14e for a weighted average EPC for shellfish based on the pool of individual crab and mussel EPCs. The latter EPC was used to estimate dose for the adult API RME seafood consumption scenario. As described in Section B.3.4.1 and Table B-12, the API seafood consumption scenario included a consumption rate for all shellfish species, rather than individual consumption rates for crab and mussels. Accordingly, the EPC for all shellfish for this scenario includes both species. The weighted average EPC was calculated by first compiling fractional consumption rates for crabs and mussels for each individual surveyed in EPA (1999c) .[18] From these fractions, a median ratio of 0.87 crab to 0.13 mussel was calculated. The weighted average EPC for shellfish was then calculated using the following equation: (0.87 x crab EPC) + (0.13 x mussel EPC).

EPCs based on detected concentrations are presented separately from EPCs based on detection limits for chemicals that were always undetected. Risk estimates for COPCs that were never detected are presented in the uncertainty assessment (Section B.6.3).

A summary of LDW tissue chemistry data is provided in Subappendix B.2 of the HHRA and Appendix D of the RI. The RI Appendix D tables summarize the data by species, whereas the summary in Subappendix B.2 summarizes all tissue chemistry used in the HHRA in a single table. Appendix D of the RI also describes a CD that accompanies the RI report that contains all the raw data used in the RI and RAs.

[18] Each fraction was calculated according to the following equation: single species consumption / (crab consumption +mussel consumption)

Table B-14a. Exposure point concentrations and summary statistics (mg/kg ww) for English sole

COPC	Number of Samples [a]	Number of Detections	Maximum Detection Limit	Maximum detected Value	Mean	95% UCL on Mean	EPC
Detected COPCs							
Arsenic	9	9	n/a	15.1	10.9	12.7	1.27 [b]
BEHP	6	1	0.016	0.040	0.011	0.045	0.040
Chromium	3	2	0.049	0.062	0.047	n/a	0.062
Copper	9	9	n/a	0.37	0.24	0.30	0.30
DDTs (total)	9	7	0.0020	0.011	0.0047	0.0073	0.0073
Mercury	15	15	n/a	0.083	0.052	0.062	0.062
PCBs (total)	15	15	n/a	0.53	0.23	0.29	0.29
Tributyltin (as ion)	9	3	0.0020	0.0056	0.0021	0.0040	0.0040
Zinc	3	3	n/a	4.57	4.10	n/a	4.57
Undetected COPCs							
1,2-Diphenylhydrazine	6	0	0.053	n/a	n/a	n/a	0.027
3,3'-Dichlorobenzidine	3	0	0.027	n/a	n/a	n/a	0.014
Aldrin	9	0	0.00050	n/a	n/a	n/a	0.00025
alpha-BHC	9	0	0.00050	n/a	n/a	n/a	0.00025
Benzidine	3	0	0.64	n/a	n/a	n/a	0.32
beta-BHC	9	0	0.00050	n/a	n/a	n/a	0.00025
Bis(2-chloroethyl)ether	6	0	0.016	n/a	n/a	n/a	0.0080
Bis(2-chloroisopropyl)ether	6	0	0.053	n/a	n/a	n/a	0.027
Cadmium	3	0	0.0079	n/a	n/a	n/a	0.0040
cPAHs	6	0	0.044	n/a	n/a	n/a	0.022
Dieldrin	9	0	0.0010	n/a	n/a	n/a	0.00050
gamma-BHC	9	0	0.00050	n/a	n/a	n/a	0.00025
Heptachlor	9	0	0.00050	n/a	n/a	n/a	0.00025
Heptachlor epoxide	9	0	0.00050	n/a	n/a	n/a	0.00025
Hexachlorobenzene	6	0	0.018	n/a	n/a	n/a	0.0090
Hexachlorobutadiene	6	0	0.027	n/a	n/a	n/a	0.014
Lead	9	0	0.030	n/a	n/a	n/a	0.015
N-Nitrosodimethylamine	6	0	0.11	n/a	n/a	n/a	0.055
N-Nitroso-di-n-propylamine	6	0	0.027	n/a	n/a	n/a	0.014
Pentachlorophenol	6	0	0.036	n/a	n/a	n/a	0.018
Toxaphene	9	0	0.010	n/a	n/a	n/a	0.0050

n/a = not applicable

No data available for chlordane (a tissue COPC)

[a] All samples are composites of multiple individuals (see Table B-2 for additional details)

[b] EPC for arsenic calculated using inorganic/total arsenic ratio of 0.1 (see Section B.2.3) multiplied by the maximum or 95% UCL (whichever is lower)

Table B-14b. Exposure point concentrations and summary statistics (mg/kg ww) for perch

COPC	Number of Samples [a]	Number of Detections	Maximum Detection Limit	Maximum Detected Value	Mean	95% UCL on Mean	EPC
Detected COPCs							
Mercury	3	2	0.020	0.060	0.030	n/a	0.060
PCBs (total)	3	3	n/a	0.228	0.151	n/a	0.228
Tributyltin (as ion)	3	3	n/a	0.0080	0.0057	n/a	0.0080

n/a = not applicable

COPCs from Table B-7 that are not listed in this table have not been analyzed in perch filets

[a] Two of the three samples are composites of multiple individuals (see Table B-2 for additional details)

Table B-14c. Exposure point concentrations and summary statistics (mg/kg ww) for crab

COPC	Number of Samples [a]	Number of Detections	Maximum Detection Limit	Maximum Detected Value	Mean	95% UCL on mean	EPC
Detected COPCs							
Arsenic	2	2	n/a	12.5	9.95	n/a	1.25 [b]
Cadmium	2	2	n/a	0.022	0.017	n/a	0.022
Chromium	2	2	n/a	0.160	0.145	n/a	0.160
Copper	2	2	n/a	15.8	14.6	n/a	15.8
Lead	2	2	n/a	0.244	0.243	n/a	0.244
Mercury	5	5	n/a	0.111	0.0861	n/a	0.111
PCBs (total)	5	5	n/a	0.177	0.128	n/a	0.177
Tributyltin (as ion)	5	2	0.0020	0.0819	0.0264	n/a	0.0819
Zinc	2	2	n/a	39.1	36.8	n/a	39.1
Undetected COPCs							
1,2-Diphenylhydrazine	2	0	0.053	n/a	n/a	n/a	0.027
3,3'-Dichlorobenzidine	2	0	0.027	n/a	n/a	n/a	0.014
Benzidine	2	0	0.64	n/a	n/a	n/a	0.32
Bis(2-chloroethyl)ether	2	0	0.016	n/a	n/a	n/a	0.0080
BEHP	2	0	0.016	n/a	n/a	n/a	0.0080
Bis(2-chloroisopropyl)ether	2	0	0.053	n/a	n/a	n/a	0.027
cPAHs	2	0	0.044	n/a	n/a	n/a	0.022
Hexachlorobenzene	2	0	0.016	n/a	n/a	n/a	0.0080
Hexachlorobutadiene	2	0	0.027	n/a	n/a	n/a	0.014
N-Nitrosodimethylamine	2	0	0.11	n/a	n/a	n/a	0.055

COPC	Number of Samples [a]	Number of Detections	Maximum Detection Limit	Maximum Detected Value	Mean	95% UCL on mean	EPC
N-Nitroso-di-n-propylamine	2	0	0.027	n/a	n/a	n/a	0.014
Pentachlorophenol	2	0	0.027	n/a	n/a	n/a	0.014

n/a = not applicable

COPCs from Table B-7 (organochlorine pesticides) that are not listed in this table have not been analyzed in crab tissue

[a] All but one sample analyzed for mercury, PCBs, and TBT was a composite of multiple individuals (see Table B-2 for additional details)

[b] EPC for arsenic calculated using inorganic/total arsenic ratio of 0.1 (see Section B.2.3) multiplied by the maximum or 95% UCL (whichever is lower)

Table B-14d. Exposure point concentrations and summary statistics (mg/kg ww) for mussels

COPC	Number of Samples [a]	Number of Detections	Maximum Detection Limit	Maximum Detected Value	Mean	95% UCL on Mean	EPC
Detected COPCs							
Arsenic	22	22	n/a	1.07	0.809	0.867	0.0867 [b]
BEHP	22	2	0.016	0.187	0.0170	n/a	0.187
Cadmium	22	22	n/a	0.840	0.485	0.550	0.550
cPAHs	22	11	0.0437	0.0482	0.0338	0.0386	0.0386
Chromium	22	21	0.050	0.346	0.159	0.186	0.186
Copper	22	22	n/a	1.71	1.19	1.29	1.29
Lead	22	22	n/a	0.723	0.412	0.479	0.479
Mercury	21	21	n/a	0.0228	0.0129	0.0144	0.0144
PCBs (total)	22	18	0.013	0.060	0.0336	0.0397	0.0397
Tributyltin (as ion)	22	22	n/a	0.0367	0.0230	0.0253	0.0253
Zinc	22	22	n/a	44.1	30.0	32.3	32.3
Undetected COPCs							
1,2-Diphenylhydrazine	22	0	0.053	n/a	n/a	n/a	0.027
3,3'-Dichlorobenzidine	22	0	0.027	n/a	n/a	n/a	0.014
Aldrin	11	0	0.0013	n/a	n/a	n/a	0.00065
alpha-BHC	11	0	0.0013	n/a	n/a	n/a	0.00065
Benzidine	22	0	0.64	n/a	n/a	n/a	0.32
beta-BHC	11	0	0.0013	n/a	n/a	n/a	0.00065
Bis(2-chloroethyl)ether	22	0	0.016	n/a	n/a	n/a	0.0080
Bis(2-chloroisopropyl)ether	22	0	0.053	n/a	n/a	n/a	0.027
Chlordane	11	0	0.0067	n/a	n/a	n/a	0.0033
DDTs (total)	11	0	0.0013	n/a	n/a	n/a	0.00065
Dieldrin	11	0	0.0013	n/a	n/a	n/a	0.00065
gamma-BHC	11	0	0.0013	n/a	n/a	n/a	0.00065

COPC	Number of Samples [a]	Number of Detections	Maximum Detection Limit	Maximum Detected Value	Mean	95% UCL on Mean	EPC
Heptachlor	11	0	0.0013	n/a	n/a	n/a	0.00065
Heptachlor epoxide	11	0	0.0013	n/a	n/a	n/a	0.00065
Hexachlorobenzene	22	0	0.016	n/a	n/a	n/a	0.0080
Hexachlorobutadiene	22	0	0.027	n/a	n/a	n/a	0.014
N-Nitrosodimethylamine	22	0	0.11	n/a	n/a	n/a	0.055
N-Nitroso-di-n-propylamine	22	0	0.027	n/a	n/a	n/a	0.014
Pentachlorophenol	22	0	0.027	n/a	n/a	n/a	0.014
Toxaphene	11	0	0.013	n/a	n/a	n/a	0.0065

n/a = not applicable

[a] All samples are composites of multiple individuals (see Table B-2 for additional details)

[b] EPC for arsenic calculated using inorganic/total arsenic ratio of 0.1 (see Section B.2.3) multiplied by the maximum or 95% UCL (whichever is lower)

Table B-14e. Exposure point concentrations for shellfish group (mg/kg ww) based on crab and mussel data

COPC	Crab EPC (from Table B-13c)	Mussel EPC (From Table B-13d)	Shellfish EPC [a]
Detected COPCs			
Arsenic	1.25	0.0867	1.10
BEHP	0.0080	0.187	0.031
Cadmium	0.022	0.550	0.091
cPAHs	0.022	0.039	0.024
Chromium	0.160	0.186	0.163
Copper	15.8	1.29	13.9
Lead	0.244	0.479	0.275
Mercury	0.111	0.0144	0.098
PCBs (total)	0.177	0.0397	0.159
Tributyltin (as ion)	0.0819	0.025	0.075
Zinc	39.1	32.3	38.2
Undetected COPCs			
1,2-Diphenylhydrazine	0.027	0.027	0.027
3,3'-Dichlorobenzidine	0.014	0.014	0.014
Aldrin	n/a	0.00065	0.00065
Benzidine	0.32	0.32	0.32
Bis(2-chloroethyl)ether	0.0080	0.0080	0.0080
Bis(2-chloroisopropyl)ether	0.027	0.027	0.027
Chlordane	n/a	0.0033	0.0033
DDTs (total)	n/a	0.00065	0.00065

COPC	CRAB EPC (FROM TABLE B-13C)	MUSSEL EPC (FROM TABLE B-13D)	SHELLFISH EPC [a]
Dieldrin	n/a	0.00065	0.00065
gamma-BHC	n/a	0.00065	0.00065
Heptachlor	n/a	0.00065	0.00065
Heptachlor epoxide	n/a	0.00065	0.00065
Hexachlorobenzene	0.0080	0.0080	0.0080
Hexachlorobutadiene	0.014	0.014	0.014
N-Nitrosodimethylamine	0.055	0.055	0.055
N-Nitroso-di-n-propylamine	0.014	0.014	0.014
Pentachlorophenol	0.014	0.014	0.014
Toxaphene	n/a	0.0065	0.0065

n/a = not applicable

[a] Weighted average for shellfish group calculated using the following equation: (0.87 x crab EPC + 0.13 x mussel EPC). See text for additional details.

B.3.4.3.2 Sediment

As described above, EPA (1989) recommends that a one-sided 95% UCL on the mean be used in deterministic risk assessments. Calculating such a parameter using data that have not been randomly collected is likely to result in an overestimate of the EPC because many of the samples have been collected at locations where contamination is known or suspected to exist (Burmaster and Thompson 1997). To account for such spatial variability in sampling intensity, many scientists use Thiessen polygons for spatial analysis. The Thiessen polygon associates each point in a plane with the closest sampling location for which a measurement is available (Burmaster and Thompson 1997). In effect, this algorithm assumes that the concentration at any point where measurements have not been made is the same as the concentration in the sample closest to that point. The spatial average concentration is a weighted average of all measurements, with each weight equal to the area of its associated polygon as a fraction of the total area for which samples have been collected. The formula for calculating spatially weighted averages is shown in Equation 5 (Scott et al. 2000).

$$\bar{x}_w = \sum w_i x_i$$

(*Equation 5***)**

where:

$\bar{x}_w$ = spatially weighted arithmetic mean
w_i = fractional area weight associated with the i^{th} sample
x_i = concentration of the i^{th} sample

Polygons were defined by the distances to the nearest sampling locations. The northern and southern extents of the surface sediment sampling events used in calculating the spatially weighted averages were defined by the southern edge of Harbor Island and the southern boundary of the GIS shape files created by Weston for

the EPA Site Inspection, respectively. Similar boundaries were created along the upland margins of the intertidal sampling locations.

To remain consistent with EPA (1989) guidance on calculating EPCs, a 95% UCL on the spatially weighted mean was calculated for each COPC. There are a variety of methods for computing this parameter using Thiessen polygons. The differences between the methods are based on the assumed distribution of the underlying data. The method described by EPA (1992b) assumes that the data are normally distributed. For data that are lognormally distributed, EPA (2002a) recommends the Land (1971, 1975) method. Finally, there are various non-parametric methods that rely on bootstrapping (as described above)[19] that do not require any assumptions about the underlying distribution.

For the Phase 1 HHRA, 95% UCLs on the spatially weighted means for each COPC identified in Tables B-4 and B-5 were calculated using Equations 5, 6, and 7, which are based on the assumption that the data were normally distributed (Scott et al. 2000). This approach is used for Phase 1, but additional methods for calculating this parameter may be explored during the Phase 2 HHRA.

$$s_w = \sqrt{\sum w_i (x_i - \bar{x}_w)^2} \qquad \textbf{(}\textit{Equation 6}\textbf{)}$$

where:

s_w = area-weighted standard deviation

$$95\%UCL = \bar{x}_w + t_{0.95,n-1}\frac{s_w}{\sqrt{n}} \qquad \textbf{(}\textit{Equation 7}\textbf{)}$$

where:

95% UCL = 95% upper confidence limit on the area-weighted arithmetic mean

$t_{0.95,n-1}$ = value of a Student's *t* distribution with n-1 degrees of freedom associated with a probability of 0.95

n = number of samples

In cases where multiple analyses were conducted at a single location, because of laboratory or field duplicates, or because multiple samples were collected at that location, either during one event or multiple events, concentrations were averaged according to the following procedure. A hierarchical approach was used where concentrations from laboratory replicates were averaged, followed by concentrations from field duplicates, and finally by concentrations from multiple samples at a single location. Averaging rules were dependent on whether the concentration was a "detect" or "non-detect." If all concentrations were detects for a given parameter, the values were simply averaged arithmetically. If all concentrations were undetected for

[19] Bootstrapping is a technique by which the original dataset is randomly sampled to create pseudoreplicate datasets. The parameter of interest can be calculated for each pseudoreplicate dataset, thereby providing information about the variability of that parameter.

a given parameter, the minimum detection limit was reported as the "average", since this minimum is the primary constraint on the "true" concentration. If the concentrations are a mixture of detects and non-detects, one-half the detection limit for the non-detects was averaged with the detected concentrations and the resulting mean value was treated as a detect.

For the netfishing scenario, Thiessen polygons were created around each location in both the intertidal and subtidal strata (see Map B-5 for an example using total PCBs). Intertidal and subtidal sediment data were used in the analysis to reflect the fact that fishing nets may come in contact with sediment in both strata.

For the beach play scenario, only locations within the intertidal strata were used, for the following reasons. Although children may be exposed to sediments below -2 ft MLLW (i.e., the boundary between the intertidal and subtidal strata), direct contact with subtidal sediments at and near this boundary would occur only a few hours per year during extremely low tides. In addition, exposure to submerged sediments through wading is a fundamentally different exposure route than beach play above the tide line in that sediment is unlikely to adhere to skin that is underwater.

Given the patchy distribution of intertidal sediment sampling, Thiessen polygons were created in three separate areas (Map B-3). Sampling density in each of the three regions is relatively high. Data from outside these three areas were not used to calculate EPCs because the sampling density was too low to make the calculation of area-weighted concentrations meaningful. Separate EPCs for this scenario were calculated for each region. The excluded intertidal samples generally had concentrations similar to, or lower than, the concentrations from the three included intertidal regions. For example, the arithmetic mean total PCB concentration for all intertidal samples that were not included in any of the three intertidal regions was 600 µg/kg dry weight, compared to the spatially weighted average concentrations of 150, 1400, and 620 µg/kg dry weight for the three intertidal regions (Table B-16).

EPCs for the two scenarios are given in Tables B-15 (netfishing) and B-16 (beach play). EPCs are shown separately for COPCs that were detected and COPCs that were never detected. The EPCs for chemicals that were never detected have a very high uncertainty. Accordingly, risk estimates for these chemicals are presented in the uncertainty assessment rather than in the risk characterization.

As described in Section 3.3.5.1, total PCB data for most of the sampling events were derived by summing detected Aroclors. The NOAA Site Characterization used a non-standard method for calculating total PCBs (NOAA 1997). Although the total PCB data generated using these two different methods may not be strictly comparable, EPCs were calculated using the total PCB values generated by both methods.

A summary of LDW sediment chemistry data is provided in Subappendix B.2 of the HHRA and Appendix D of the RI. Appendix D of the RI also describes a CD that accompanies the RI report that contains all the raw data used in the RI and RAs.

Table B-15. Exposure point concentrations and summary statistics (mg/kg dw) in the netfishing exposure scenario

COPC	Detection Frequency	Arithmetic Mean Conc.	95% UCL of Normal Data	Maximum Conc.	Spatially Weighted Mean Conc.	95% UCL of Spatially Weighted Mean Conc.	Exposure Point Conc.
Detected COPCs							
2,3,7,8-TCDD TEQ	29/29	0.000020	0.000035	0.00022	0.000019	0.000033	0.000033
Aluminum	450/450	19,000	20,000	110,000	18,000	19,000	19,000
Antimony	97/389	5.7	6.3	110	5.1	5.4	5.4
Arsenic	525/575	13	14	99	12	13	13
Barium	430/430	130	160	7,400	120	160	160
Cadmium	430/567	1.2	1.7	120	0.44	0.52	0.52
cPAHs	531/557	0.55	0.66	31	0.42	0.49	0.49
Chromium	571/571	41	47	1,100	29	30	30
Copper	575/575	120	170	12,000	58	65	65
Dieldrin	5/100	0.0051	0.0098	0.28	0.0041	0.0079	0.0079
Iron	448/448	28,000	29,000	160,000	27,000	27,000	27,000
Lead	575/575	130	200	23,000	48	57	57
Manganese	445/445	350	370	3,300	310	320	320
PCBs (total-calc'd)	905/957	1.1	1.5	220	0.36	0.49	0.49
Undetected COPCs							
1,2,3-Trichloropropane	0/44	0.015	0.035	0.53	0.0084	0.022	0.022
Benzidine	0/8	0.56	0.63	0.75	n/a	n/a	0.63
N-Nitrosodimethylamine	0/87	0.091	0.096	0.13	0.098	0.10	0.10

Table B-16. Exposure point concentrations and summary statistics (mg/kg dw) in the beach play exposure scenario

COPC	Detection Frequency	Arithmetic Mean Conc.	95% UCL of Normal Data	Maximum Conc.	Spatially Weighted Mean Conc.	95% UCL of Spatially Weighted Mean Conc.	Exposure Point Conc.
Kellogg Island area							
Detected COPCs							
2,3,7,8-TCDD TEQ [a]	8/8	0.000034	0.000086	0.00022	n/a	n/a	0.000086
Aluminum	4/4	15,000	18,000	19,000	14,000	17,000	17,000
Antimony	1/4	5.3	5.8	6.0	5.5	6.1	6.1
Arsenic	15/15	10	12	18	11	12	12
Barium	4/4	62	85	89	58	75	75
Cadmium	13/15	0.46	0.56	0.80	0.40	0.48	0.48
cPAHs	15/15	0.14	0.27	1.1	0.38	0.59	0.59
Chromium	15/15	33	36	43	31	34	34

COPC	Detection Frequency	Arithmetic Mean Conc.	95% UCL of Normal Data	Maximum Conc.	Spatially Weighted Mean Conc.	95% UCL of Spatially Weighted Mean Conc.	Exposure Point Conc.
Copper	15/15	59	69	120	56	62	62
Heptachlor epoxide	1/2	0.00075	n/a	0.0010	n/a	n/a	0.0010
Iron	4/4	27,000	42,000	46,000	24,000	35,000	35,000
Lead	15/15	59	76	180	52	60	60
Manganese	4/4	200	250	260	190	230	230
Mercury	15/15	0.16	0.19	0.35	0.13	0.15	0.15
Nickel	15/15	25	28	37	22	25	25
PCBs (total-calc'd)	30/30	0.14	0.20	0.77	0.15	0.21	0.21
Silver	9/15	0.46	0.59	0.90	0.44	0.53	0.53
Thallium	4/4	0.14	0.18	0.18	0.13	0.16	0.16
Vanadium	4/4	52	62	65	50	58	58
Zinc	15/15	120	130	170	120	130	130
Nondetected COPCs							
1,2,3-Trichloropropane	0/2	0.0054	n/a	0.0092	n/a	n/a	0.0092
2-Nitroaniline	0/4	0.050	0.050	0.050	0.050	0.050	0.050
Benzidine	no data						
Bis(2-chloroethyl)ether	0/4	0.020	0.020	0.020	0.020	0.020	0.020
DDTs	no data						
Dieldrin	0/2	0.0010	n/a	0.0010	n/a	n/a	0.0010
Hexachlorobenzene	0/15	0.0071	0.091	0.010	0.0072	0.0092	0.0092
N-Nitrosodimethylamine	no data						
N-Nitroso-di-n-propylamine	0/4	0.020	0.020	0.020	0.020	0.020	0.020
SE intertidal area							
Detected COPCs							
2,3,7,8-TCDD TEQ [a]	8/8	0.000034	0.000086	0.00022	n/a	n/a	0.000086
Aluminum	109/109	18,000	20,000	110,000	17,000	18,000	18,000
Antimony	20/47	12	18	110	5.8	7.6	7.6
Arsenic	111/148	11	13	79	11	12	12
Barium	103/103	140	210	3,500	63	79	79
Cadmium	98/148	3.4	5.3	120	0.91	1.5	1.5
cPAHs	130/141	0.58	0.74	8.6	0.64	0.83	0.83
Chromium	148/148	69	88	1,100	37	44	44
Copper	148/148	280	470	12,000	62	110	110
DDTs (total-calc'd)	4/13	0.24	0.63	2.9	0.85	1.5	1.5
Dieldrin	1/11	0.027	0.073	0.28	0.083	0.15	0.15
Hexachlorobenzene	7/141	0.026	0.030	0.070	0.021	0.025	0.021
Iron	109/109	30,000	33,000	160,000	25,000	26,000	26,000
Lead	150/150	360	620	23,000	77	130	130

COPC	Detection Frequency	Arithmetic Mean Conc.	95% UCL of Normal Data	Maximum Conc.	Spatially Weighted Mean Conc.	95% UCL of Spatially Weighted Mean Conc.	Exposure Point Conc.
Manganese	111/111	450	530	3,300	320	350	350
Mercury	113/148	0.21	0.27	4.6	0.15	0.17	0.17
Nickel	145/145	50	63	910	26	31	31
PCBs (total-calc'd)	229/237	3.1	4.7	220	1.4	2.2	2.2
Silver	78/150	3.4	6.5	270	0.80	1.4	1.4
Thallium	38/110	6.2	7.2	30	2.5	3.1	3.1
Vanadium	97/97	58	61	150	54	57	57
Zinc	150/150	380	520	9,700	140	190	190
Nondetected COPCs							
1,2,3-Trichloropropane	0/5	0.0014	0.0016	0.0017	0.0014	0.0016	0.0016
2-Nitroaniline	0/131	0.016	0.18	1.0	0.12	0.14	0.14
Benzidine	0/3	0.58	0.83	0.75	n/a	n/a	0.75
Bis(2-chloroethyl)ether	0/133	0.061	0.067	0.14	0.047	0.053	0.053
Heptachlor epoxide	0/11	0.0054	0.014	0.050	0.015	0.027	0.027
N-Nitrosodimethylamine	0/12	0.058	0.075	0.090	0.089	0.092	0.092
N-Nitroso-di-n-propylamine	0/135	0.067	0.076	0.55	0.053	0.063	0.063
SW intertidal area							
Detected COPCs							
2,3,7,8-TCDD TEQ [a]	8/8	0.000034	0.000086	0.00022	n/a	n/a	0.000086
Aluminum	23/23	15,000	16,000	23,000	14,000	15,000	15,000
Antimony	3/23	5.2	5.3	7.0	5.2	5.4	5.4
Arsenic	30/30	9.9	11	16	9.0	9.7	9.7
Barium	23/23	48	53	73	50	55	55
Cadmium	21/30	0.18	0.20	0.33	0.15	0.17	0.17
cPAHs	30/30	0.21	0.29	1.5	0.27	0.36	0.36
Chromium	30/30	22	23	30	22	23	23
Copper	30/30	32	34	44	29	31	31
Hexachlorobenzene	3/30	0.030	0.069	0.69	0.018	0.042	0.042
Iron	23/23	23,000	25,000	31,000	23,000	24,000	24,000
Lead	30/30	29	47	330	57	90	90
Manganese	23/23	350	400	780	330	380	380
Mercury	26/30	0.09	0.11	0.23	0.089	0.10	0.10
Nickel	30/30	17	18	25	16	17	17
PCBs (total-calc'd)	60/65	0.60	1.0	12	0.62	1.1	1.1
Silver	23/30	0.15	0.17	0.29	0.13	0.14	0.14
Thallium	23/23	0.055	0.061	0.10	0.055	0.061	0.061
Vanadium	23/23	52	54	69	49	52	52
Zinc	30/30	77	82	130	74	80	80

COPC	Detection Frequency	Arithmetic Mean Conc.	95% UCL of Normal Data	Maximum Conc.	Spatially Weighted Mean Conc.	95% UCL of Spatially Weighted Mean Conc.	Exposure Point Conc.
Nondetected COPCs							
1,2,3-Trichloropropane	0/2	0.0013	n/a	0.0013	n/a	n/a	0.0013
2-Nitroaniline	0/30	0.050	0.050	0.050	0.050	0.050	0.050
Benzidine	no data						
Bis(2-chloroethyl)ether	0/30	0.020	0.020	0.020	0.020	0.020	0.020
DDTs (total-calc'd)	0/3	0.0014	0.0030	0.0025	n/a	n/a	0.0025
Dieldrin	0/3	0.00092	0.0012	0.0010	n/a	n/a	0.0010
Heptachlor epoxide	0/3	0.00058	0.00083	0.00075	n/a	n/a	0.00075
N-Nitrosodimethylamine	no data						
N-Nitroso-di-n-propylamine	0/30	0.020	0.020	0.020	0.020	0.020	0.020

Statistics calculated assuming one-half detection limit for non-detects

Spatially weighted concentrations were not calculated for COPCs with less than 10 polygons over the entire LDW or with less than 4 polygons in a particular intertidal region

[a] Intertidal data for this COPC are from all intertidal regions of the LDW because the maximum concentration does not fall within any of the three regions shown in Map B-4.

B.3.4.4 Lead modeling

Risk estimates from lead exposure are not made using the equations presented in Section B.3.4. Instead, risks are estimated using the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK; EPA 1994) and the Adult Lead Model (ALM; EPA 1996c). The parameterization of each model is discussed in separate sections below.

B.3.4.4.1 Children (IEUBK)

The IEUBK model (version 1.0 for Windows) predicts blood-lead concentrations for children exposed to lead in their environment. The model requires input such as relevant absorption parameters, and intake and exposure rates. The model then calculates and recalculates a complex set of equations to estimate the potential concentration of lead in the blood for a hypothetical population of children (ages 6 months to 7 years). The input parameters for this model are described below.

Default input parameters exist in the model for lead intake via air, drinking water, soil, and diet. The model also allows for alternate dietary data to be input if data are available to calculate the alternate dietary source lead concentration and the percentage of its total food class that is composed of the alternate dietary source. The alternate dietary data are added to the other source data to derive a combined intake from all sources. For this HHRA, all default parameters were maintained except for soil concentration (default = 200 mg/kg) and alternate dietary source data for fish (default concentration = 0 mg/kg, default percentage of all meat consumed = 0%.).

Three sediment lead EPCs were calculated for intertidal sediments in the beach play scenario, corresponding to the Kellogg Island, southwest, and southeast intertidal regions. Each sediment lead EPC was used together with the exposure frequency for sediments. In addition, in each of the intertidal evaluations, exposure to soils was also assumed to occur on the days when sediment exposures did not occur, i.e., 324 days per year. In this way a time-weighted average was calculated at each intertidal location (Table B-17). Alternate dietary data (fish and shellfish) were input to the food parameter section of the model as described in Table B-17. The IEUBK model applies average or central tendency estimates for all terms (EPA 1994). For seafood consumption rates, the median rate identified in the Suquamish Tribe data set of 0.90 g/day was applied, and the percentage of the alternate food source (fish) of its food group (all meat) was set at 0.92% (Table B-17). Two alternate food source concentrations were calculated using lead concentrations in sole, crab, and mussels, and a high and low estimated lead concentration for perch, for which no lead data were available. The high estimated lead concentration for perch was set equal to the mussel EPC, which was 0.479 mg/kg. The low estimated lead concentration for perch was set equal to the sole EPC, which was 0.015 mg/kg. Two model scenarios were run: one with the highest soil concentration (Southeast intertidal) and the higher estimated alternate food source concentration, and the other with the lowest soil concentration (from Southwest intertidal) and the lower estimated alternate food source concentration. The results of the IEUBK model runs are presented in Section B.5.4.

Table B-17. Input parameters for IEUBK lead model

PARAMETER	VALUE	UNITS
Sediment/soil concentration [a]	184 (Kellogg Island)	mg/kg dw
	192 (Southeast Intertidal)	mg/kg dw
	184 (Southwest Intertidal)	mg/kg dw
Alternate food source concentration [b]	0.19 [c]	mg/kg ww
	0.27 [d]	mg/kg ww
Alternate food source fraction [e]	0.92	%

[a] Derived from sediment EPCs:

[$(Pb_{sed} * EF_{sed}) + (Pb_{soil} * EF_{soil}) / (EF_{sed} + EF_{soil})$], where Pb_{sed} = EPC (mg/kg); EF_{sed} = beach play exposure frequency, 41 days/yr; Pb_{soil} = average default soil Pb concentration, 200 mg/kg; EF_{soil} = soil exposure frequency, calculated by subtraction from default exposure frequency ($EF_{sed} + EF_{soil} = 365$), value is 324 days/yr

[b] Derived from tissue EPCs and median child seafood consumption rates from Table C-6 in Suquamish Tribe (2000), as calculated using 17 kg body weight and the 95% UCL on the mean fraction of consumption, by species group, from Puget Sound:

[0.479 mg/kg (mussel EPC) x 0 g/day (mussel ingestion rate for children) + 0.244 mg/kg (crab EPC) x 0.69 g/day (crab ingestion rate for children) + 0.015 (sole EPC) x 0.051 g/day (sole ingestion rate for children) + X mg/kg (estimated perch lead concentration) x 0.16 g/day (perch ingestion rate)] / 0.90 (sum of ingestion rates)

[c] Low estimated perch lead concentration = 0.015 mg/kg

[d] High estimated perch lead concentration = 0.479 mg/kg

[e] 0.90 g/day (fish consumed per day) / 98.05 (meat consumed per day)

B.3.4.4.2 Adults (ALM)

This ALM is based on protecting the developing fetus of a pregnant woman, the most sensitive subpopulation for adult lead exposure. The model incorporates exposure to soil that is more representative of older children and adults than young children. Accordingly, EPA has used this model to estimate soil lead cleanup levels for sites at which the likely exposed population would be older children or adults. Although the model was developed to assess soil exposures, it has been applied here, in agreement with EPA Region 10, to evaluate exposure to lead in both sediments and in fish and shellfish. Adjustments were made to the model to account for fish intake. Specifically, Kissinger (2002) provided a revised algorithm that incorporates an exposure term for seafood consumption. This approach provides a means to evaluate cumulative exposure to lead in the LDW, but is somewhat uncertain due to the lack of complete knowledge on intake from fish and resulting blood lead concentrations.

The adult lead model applied for the LDW estimates an average blood lead level based on additional exposure (above a baseline level) to lead in sediments, seafood, and air. The contribution of lead from air was considered negligible for this site because blood lead levels are much less sensitive to passive re-entrainment of lead from soil in air. The equation is thus:

$$PbB_{central} = \frac{PbB_0 + BKSF \times FI \times ((PB_s \times IR_s \times AF_s \times EF_s) + (PB_f \times IR_f \times AF_f \times EF_f))}{AT} \quad Equation\ 8$$

where $PbB_{central}$ is the geometric mean blood lead level ($\mu g / dL$) in exposed adults. The definition and parameterization of the other variables in Equation 8 is provided in Table B-18.

Table B-18. Input parameters for ALM

PARAMETER	DESCRIPTION	VALUE	UNITS
PbB_0	baseline blood lead level	1.7 [a]	µg/dL
BKSF	biokinetic slope factor	0.4 (EPA default)	µg/dL per µg/day
FI	fractional intake	1	unitless
IR_s	sediment ingestion rate	50 (EPA default) [b]	mg/day
IR_f	seafood ingestion rate	6.0 [c]	g/day
Pb_s	lead concentration in sediment – high estimate	130 [d]	mg/kg dw
Pb_s	lead concentration in sediment – low estimate	60 [d]	mg/kg dw
Pb_f	lead concentration in seafood – high estimate	0.27 [e]	mg/kg ww
Pb_f	lead concentration in seafood – low estimate	0.17 [f]	mg/kg ww
EF_s	exposure frequency for sediment exposure	119 [g]	days/yr
EF_f	exposure frequency for seafood consumption	365	days/yr
AF_s	gastrointestinal absorption fraction for lead in sediment	0.12 (EPA default for soil)	unitless
AF_f	gastrointestinal absorption fraction for lead in tissue	0.12	unitless
AT	averaging time	365	days

[a] Because communities bordering the LDW include a sizable Mexican-American population, the average baseline blood lead level of Mexican-American women in the US (EPA 2002b) was used

[b] Although EPA has not developed default exposure assumptions for sediments, a health protective assumption was applied that sediment consumption would be equivalent to 100 percent of the assumed soil and dust intake on each day an individual visited the LDW.

[c] Median fish and crab consumption rates from Table C-1 in Suquamish Tribe (2000), adjusted for percentage harvested from Puget Sound (see Table B-12); sum of crab (4.1 g/day), sole (0.69 g/day), and perch (1.2 g/day) consumption rates

[d] The high estimate is the highest of the three lead EPCs (which is for southeast area) from Table B-16, while the low estimate is the lowest of the three lead EPCs (which is for Kellogg Island) from Table B-16

[e] Derived from tissue EPCs and median adult consumption rates from Table C-1 in Suquamish Tribe (2000):

[0.479 mg/kg (mussel EPC) x 0 g/day (mussel ingestion rate for adults) + 0.244 mg/kg (crab EPC) x 4.1 g/day (crab ingestion rate for adults) + 0.015 (sole EPC) x 0.69 g/day (sole ingestion rate for adults) + 0.479 mg/kg (perch lead assumed to be equal to mussel EPC) x 1.2 g/day (perch ingestion rate for adults)] / 6.0 (sum of ingestion rates)

[f] [0.479 mg/kg (mussel EPC) x 0 g/day (mussel ingestion rate for adults) + 0.244 mg/kg (crab EPC) x 4.1 g/day (crab ingestion rate for adults) + 0.015 (sole EPC) x 0.69 g/day (sole ingestion rate for adults) + 0.015 mg/kg (perch lead assumed to be equal to sole EPC) x 1.2 g/day (perch ingestion rate for adults)] / 6.0 (sum of ingestion rates)

[g] Assumed to be equal to tribal netfishing exposure frequency (see Table B-8a), which likely overestimates exposure of Mexican-American population

The model output includes a central tendency (geometric mean) blood lead level, which is based on central tendency estimates provided in Table B-18, and 95th percentile blood lead levels, which are calculated according to:

$$PbB_{95} = PbB_{central} \times GSD^{1.645} \qquad \textit{Equation 9}$$

where:

PbB_{95} = 95th percentile blood lead level (µg / dL)

GSD = geometric standard deviation of the blood lead distribution

1.645 = 95th percentile value for the Student's t distribution.

The GSD is an estimation of variation in blood lead around the geometric mean. It is used to estimate upper percentile blood lead levels for an individual and predict the probability of an individual exceeding a given blood lead level (target risk goal). A GSD of 2.29 identified by EPA (2002b) for Mexican-American women in the US was applied to this model.

Fetal blood lead levels are predicted based on the EPA assumption that fetal blood lead levels at birth are 90 percent of the maternal blood lead level. A 10 µg / dL blood lead level for a fetus is associated with a 11.1 µg / dL blood lead level for the mother according to EPA (1996c).

B.3.4.5 Chronic daily intake (CDI) results

The tables in this section present the results of CDI calculations performed according to Equations 1 and 2 and the exposure parameters given in Tables B-8a through B-8j. The CDI results are used in the risk characterization and uncertainty assessments (Sections B.5 and B.6). Risk estimates for COPCs that were never detected are

presented in the uncertainty assessment. The CDIs are expressed in scientific notation in the form of XE-Y, where X is an integer between 1 and 9, E represents an exponent (base 10), and Y is the value (negative) of the exponent. For example, 1E-5 is equivalent to 0.00001.

Table B-19 Netfishing, RME scenario

Table B-20 Netfishing, CT scenario

Table B-21a Beach play, Kellogg Island intertidal

Table B-21b Beach play, southeast intertidal

Table B-21c Beach play, southwest intertidal

Table B-22 Seafood consumption tribal and API RME scenarios

Table B-19. Chronic daily intake results (mg/kg-day) for the netfishing (RME) exposure scenario

COPC	CANCER		NON-CANCER	
	INGESTION	DERMAL	INGESTION	DERMAL
Detected COPCs				
2,3,7,8-TCDD	4E-12	2E-12	7E-12	3E-12
Aluminum	2E-3	4E-4	4E-3	6E-4
Antimony	7E-7	1E-7	1E-6	2E-7
Arsenic	2E-6	7E-7	3E-6	1E-6
Barium	2E-5	3E-6	3E-5	5E-6
Cadmium	7E-8	1E-8	1E-7	2E-8
cPAHs	6E-8	1E-7	1E-7	2E-7
Chromium	4E-6	6E-7	6E-6	9E-7
Copper	8E-6	1E-6	1E-5	2E-6
Dieldrin	1E-9	1E-9	2E-9	2E-9
Iron	4E-3	5E-4	6E-3	8E-4
Manganese	4E-5	6E-6	7E-5	1E-5
PCBs	6E-8	1E-7	1E-7	2E-7
Nondetected COPCs				
1,2,3-Trichloropropane	3E-9	4E-9	5E-9	7E-9
Benzidine	8E-8	1E-7	1E-7	2E-7
N-Nitrosodimethylamine	1E-8	2E-8	2E-8	3E-8

Table B-20. Chronic Daily Intake results (mg/kg-day) for the netfishing (CT) exposure scenario

COPC	CANCER		NON-CANCER	
	INGESTION	DERMAL	INGESTION	DERMAL
Detected COPCs				
2,3,7,8-TCDD	1E-12	6E-13	4E-12	2E-12
Aluminum	9E-4	1E-4	2E-3	3E-4
Antimony	2E-7	4E-8	6E-7	8E-8
Arsenic	6E-7	3E-7	1E-6	6E-7
Barium	7E-6	1E-6	2E-5	3E-6
Cadmium	2E-8	3E-9	6E-8	8E-9
cPAHs	2E-8	4E-8	5E-8	1E-7
Chromium	1E-6	2E-7	3E-6	5E-7
Copper	3E-6	4E-7	7E-6	1E-6
Dieldrin	4E-10	5E-10	9E-10	1E-9
Iron	1E-3	2E-4	3E-3	4E-4
Manganese	1E-5	2E-6	3E-5	5E-6
PCBs	2E-8	4E-8	5E-8	1E-7
Nondetected COPCs				
1,2,3-Trichloropropane	1E-9	1E-10	2E-9	3E-10
Benzidine	3E-8	4E-8	7E-8	1E-7
N-Nitrosodimethylamine	5E-9	7E-9	1E-8	2E-8

Table B-21a. Chronic daily intake results (mg/kg-day) for the beach play exposure scenario for the Kellogg Island intertidal area

COPC	CANCER		NON-CANCER	
	INGESTION	DERMAL	INGESTION	DERMAL
Detected COPCs				
2,3,7,8-TCDD	9E-12	2E-12	1E-10	2E-11
Aluminum	2E-3	1E-4	2E-2	2E-3
Antimony	7E-7	5E-8	8E-6	6E-7
Arsenic	1E-6	3E-7	1E-5	3E-6
Barium	8E-6	6E-7	9E-5	7E-6
Cadmium	5E-8	4E-9	6E-7	4E-8
cPAHs	6E-8	6E-8	7E-7	7E-7
Chromium	4E-6	3E-7	4E-5	3E-6
Copper	7E-6	5E-7	8E-5	6E-6
Heptachlor epoxide	1E-10	8E-11	1E-9	9E-10
Iron	4E-3	3E-4	4E-2	3E-3

COPC	Cancer		Non-Cancer	
	Ingestion	Dermal	Ingestion	Dermal
Manganese	2E-5	2E-6	3E-4	2E-5
Mercury	2E-8	1E-9	2E-7	1E-8
Nickel	3E-6	2E-7	3E-5	2E-6
PCBs	2E-8	2E-8	3E-7	3E-7
Silver	6E-8	4E-9	7E-7	5E-8
Thallium	2E-8	1E-9	2E-7	1E-8
Vanadium	6E-6	4E-7	7E-5	5E-6
Zinc	1E-5	1E-6	2E-4	1E-5
Nondetected COPCs				
1,2,3-Trichloropropane	1E-9	7E-10	1E-8	8E-9
2-Nitroaniline	5E-9	4E-9	6E-8	5E-8
Bis(2-chloroethyl)ether	2E-9	2E-9	2E-8	2E-8
Dieldrin	1E-10	8E-11	1E-9	9E-10
Hexachlorobenzene	1E-9	7E-10	1E-8	8E-9
N-Nitroso-di-n-propylamine	2E-9	2E-9	2E-8	2E-8

Table B-21b. Chronic daily intake results (mg/kg-day) for the beach play exposure scenario for the southeast intertidal area

COPC	Cancer		Non-Cancer	
	Ingestion	Dermal	Ingestion	Dermal
Detected COPCs				
2,3,7,8-TCDD	9E-12	2E-12	1E-10	2E-11
Aluminum	2E-3	1E-4	2E-2	2E-3
Antimony	8E-7	6E-8	9E-6	7E-7
Arsenic	1E-6	3E-7	1E-5	3E-6
Barium	8E-6	6E-7	1E-4	7E-6
Cadmium	2E-7	1E-8	2E-6	1E-7
cPAHs	9E-8	8E-8	1E-6	1E-6
Chromium	5E-6	3E-7	5E-5	4E-6
Copper	1E-5	9E-7	1E-4	1E-5
DDTs	2E-7	3E-8	2E-6	4E-7
Dieldrin	2E-8	1E-8	2E-7	2E-7
Hexachlorobenzene	2E-9	2E-9	3E-8	2E-8
Iron	3E-3	2E-4	3E-2	2E-3
Manganese	4E-5	3E-6	4E-4	3E-5
Mercury	2E-8	1E-9	2E-7	2E-8
Nickel	3E-6	2E-7	4E-5	3E-6

COPC	Cancer		Non-Cancer	
	Ingestion	Dermal	Ingestion	Dermal
PCBs	2E-7	2E-7	3E-6	3E-6
Silver	1E-7	1E-8	2E-6	1E-7
Thallium	3E-7	2E-8	4E-6	3E-7
Vanadium	6E-6	4E-7	7E-5	5E-6
Zinc	2E-5	1E-6	2E-4	2E-5
Nondetected COPCs				
1,2,3-Trichloropropane	2E-10	1E-10	2E-9	1E-9
2-Nitroaniline	1E-8	1E-8	2E-7	1E-7
Benzidine	8E-8	6E-8	9E-7	7E-7
Bis(2-chloroethyl)ether	6E-9	4E-9	7E-8	5E-8
Heptachlor epoxide	3E-9	2E-9	3E-8	2E-8
N-Nitrosodimethylamine	1E-8	7E-9	1E-7	8E-8
N-Nitroso-di-n-propylamine	7E-9	5E-9	8E-8	6E-8

Table B-21c. Chronic daily intake results (mg/kg-day) for the beach play exposure scenario for the southwest intertidal area

COPC	Cancer		Non-Cancer	
	Ingestion	Dermal	Ingestion	Dermal
Detected COPCs				
2,3,7,8-TCDD	9E-12	2E-12	1E-10	2E-11
Aluminum	2E-3	1E-4	2E-2	1E-3
Antimony	6E-7	4E-8	7E-6	5E-7
Arsenic	1E-6	2E-7	1E-5	3E-6
Barium	3E-6	4E-7	7E-5	5E-6
Cadmium	2E-8	1E-9	2E-7	2E-8
cPAHs	4E-8	4E-8	4E-7	4E-7
Chromium	2E-6	2E-7	3E-5	2E-6
Copper	3E-6	2E-7	4E-5	3E-6
Hexachlorobenzene	4E-9	3E-9	5E-8	4E-8
Iron	3E-3	2E-4	3E-2	2E-3
Manganese	4E-5	3E-6	5E-4	3E-5
Mercury	1E-8	8E-10	1E-7	9E-9
Nickel	2E-6	1E-7	2E-5	2E-6
PCBs	1E-7	1E-7	1E-6	1E-6
Silver	1E-8	1E-9	2E-7	1E-8
Thallium	7E-9	5E-10	8E-8	6E-9
Vanadium	6E-6	4E-7	6E-5	5E-6

COPC	Cancer		Non-Cancer	
	Ingestion	Dermal	Ingestion	Dermal
Zinc	9E-6	6E-7	1E-4	7E-6
Nondetected COPCs				
1,2,3-Trichloropropane	1E-10	1E-10	2E-9	1E-9
2-Nitroaniline	5E-9	4E-9	6E-8	5E-8
Bis(2-chloroethyl)ether	2E-9	2E-9	2E-8	2E-8
DDTs	3E-10	6E-11	3E-9	7E-10
Dieldrin	1E-10	8E-11	1E-9	9E-10
Heptachlor epoxide	8E-11	6E-11	9E-10	7E-10
N-Nitroso-di-n-propylamine	2E-9	2E-9	2E-8	2E-8

Table B-22. Chronic daily intake results (mg/kg-day) for the RME tribal and API seafood consumption scenarios

COPC	RME Tribal –Adult		RME Tribal – Child		RME API – Adult	
	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer
Detected COPCs						
Arsenic	8E-4	1E-3	3E-4	3E-3	4E-5	9E-5
BEHP	2E-5	3E-5	2E-6	2E-5	1E-6	3E-6
Cadmium	5E-5	7E-5	5E-6	6E-5	2E-6	6E-6
cPAHs	2E-5	2E-5	5E-6	5E-5	8E-7	2E-6
Chromium	1E-4	1E-4	3E-5	4E-4	5E-6	1E-5
Copper	7E-3	9E-3	3E-3	4E-2	4E-4	8E-4
DDTs	1E-6	1E-6	4E-8	4E-7	9E-8	2E-7
Mercury	7E-5	9E-5	2E-5	3E-4	4E-6	1E-5
PCBs	2E-4	2E-4	4E-5	5E-4	1E-5	3E-5
TBT	4E-5	5E-5	2E-5	2E-4	2E-6	5E-6
Zinc	2E-2	3E-2	8E-3	9E-2	1E-3	2E-3
Nondetected COPCs						
1,2-Diphenylhydrazine	2E-5	2E-5	5E-6	6E-5	9E-7	2E-6
3,3'-Dichlorobenzidine	9E-6	1E-5	3E-6	3E-5	5E-7	1E-6
Aldrin	9E-8	1E-7	2E-9	2E-8	2E-8	4E-8
alpha-BHC	9E-8	1E-7	2E-9	2E-8	2E-8	4E-8
Benzidine	2E-4	3E-4	7E-5	8E-4	1E-5	3E-5
beta-BHC	9E-8	1E-7	2E-9	2E-8	2E-8	4E-8
bis(2-Chloroethyl)ether	5E-6	7E-6	2E-6	2E-5	3E-7	7E-7
bis(2-Chloroisopropyl)ether	2E-5	2E-5	5E-6	6E-5	9E-7	2E-6
Chlordane	3E-7	3E-7	3E-9	3E-8	9E-8	2E-7

COPC	RME Tribal –Adult		RME Tribal – Child		RME API – Adult	
	Cancer	Non-Cancer	Cancer	Non-Cancer	Cancer	Non-Cancer
Dieldrin	1E-7	2E-7	3E-9	3E-8	2E-8	5E-8
gamma-BHC	9E-8	1E-7	2E-9	2E-8	2E-8	4E-8
Heptachlor	9E-8	1E-7	2E-9	2E-8	2E-8	4E-8
Heptachlor epoxide	9E-8	1E-7	2E-9	2E-8	2E-8	4E-8
Hexachlorobenzene	6E-6	7E-6	2E-6	2E-5	3E-7	7E-7
Hexachlorobutadiene	9E-6	1E-5	3E-6	3E-5	5E-7	1E-6
N-Nitrosodimethylamine	4E-5	5E-5	1E-5	1E-4	2E-6	5E-6
N-Nitroso-di-n-propylamine	9E-6	1E-5	3E-6	3E-5	5E-7	1E-6
Pentachlorophenol	1E-5	1E-5	3E-6	3E-5	5E-7	1E-6
Toxaphene	1E-6	2E-6	3E-8	3E-7	2E-7	5E-7

B.4 Toxicity Assessment

The toxicity assessment is an evaluation of each chemical's potential to cause health effects based on available toxicological information. Toxicity information was obtained primarily from EPA's Integrated Risk Information System (IRIS; www.epa.gov/iris), EPA's 1997 Health Effects Assessment Summary Tables (HEAST), toxicological profiles presented in EPA (2000), EPA's Office of Ground Water and Drinking Water (OGWDW; www.epa.gov/OGWDW/hfacts.html), the Agency for Toxic Substance and Disease Registry (ATSDR) ToxFAQs (www.atsdr.cdc.gov/toxfaq.html), and the Hazardous Substance Data Bank (HSDB; toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB). IRIS, HSDB, ToxFAQs, and HEAST are cited only by acronym in the sections below. Other citations are presented in standard form.

Chemicals may be quantitatively evaluated on the basis of their non-carcinogenic and/or carcinogenic potential. The toxicity values used for evaluating exposure to chemicals with non-carcinogenic and carcinogenic effects are called the reference dose (RfD) and slope factor (SF), respectively.

The RfD is an estimate, with uncertainty spanning perhaps an order of magnitude or greater, of the daily exposure to the human population, including sensitive sub-populations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. The SF represents a plausible upper-bound estimate of the probability of response per unit intake of a chemical over a lifetime. The SF is based on a dose-response curve using available carcinogenic data for a given chemical. Mathematical models are used to extrapolate from high experimental doses to the low doses expected for human contact in the environment.

The pharmacokinetics, acute toxicity, chronic toxicity, and potential carcinogenicity for each COPC are discussed in Subappendix B.4. The discussion of toxic effects includes many different exposure routes, some of which are not relevant to environmental exposure within the LDW, such as occupational exposure.

The toxicity values used in this HHRA are summarized in Tables B-23 (non-cancer) and B-24 (cancer). The toxicological endpoints used to establish the RfDs are given in Table B-25. This information is used in the risk characterization to establish a hazard index and lifetime excess cancer risk. In developing toxicity values for noncancer effects, EPA reviews available data to identify the most sensitive endpoint and population, i.e., the effects that occur at the lowest concentration. These available data include effects on children and other sensitive subpopulations. Chemicals may have additional adverse effects that occur at higher exposure levels. Thus, many chemicals may have adverse effects that are not included in Table B-25 because they occur at higher doses than the effects upon which the RfDs were based. Additional discussion of uncertainties associated with the RfDs used for risk characterization is provided in Section B.6.2.1.

Table B-23. Non-cancer toxicity data (oral) for chemicals of potential concern

Chemical of Potential Concern	Chronic/ Subchronic	Oral RfD (mg/kg-day)	Critical Effect	Combined Uncertainty/ Modifying Factors	Source of RfD	Date of RfD
1,2,3-Trichloropropane	Subchronic	0.006	Alterations in clinical chemistry and reduction in red cell mass	1,000	IRIS	4.1.2002
2-Nitroaniline	n/a	0.000057	n/a	n/a	HEAST	8.15.2001
Aldrin	Chronic	0.00003	Liver toxicity	1,000	IRIS	4.1.2002
Aluminum	n/a	1	n/a	n/a	NCEA	8.15.2001
Antimony	Chronic	0.0004	Longevity, blood glucose, and cholesterol	1,000	IRIS	4.1.2002
Arsenic	Chronic	0.0003	Hyperpigmentation, keratosis and possible vascular complications	3	IRIS	4.1.2002
Barium	Subchronic	0.07	Increased kidney weight	3	IRIS	4.1.2002
Benzidine	Chronic	0.003	Brain cell vacuolization, liver cell alterations	1,000	IRIS	4.1.2002
bis(2-Chloroisopropyl)ether	Chronic	0.04	Decrease in hemoglobin and possible erythrocyte destruction	1,000	IRIS	4.1.2002
BEHP	Subchronic	0.02	Increased liver weight	1,000	IRIS	4.1.2002
Cadmium	Chronic	0.001	Proteinuria	10	IRIS	4.1.2002
Chlordane	Chronic	0.0005	Hepatic necrosis	300	IRIS	4.1.2002
Chromium (hexavalent)	Chronic	0.003	None reported	900	IRIS	4.1.2002
Copper	n/a	0.04	n/a	n/a	HEAST	8.15.2001
DDT (total)	Subchronic	0.0005	Liver lesions	100	IRIS	4.1.2002
Dieldrin	Chronic	0.00005	Liver lesions	100	IRIS	4.1.2002
gamma-BHC (lindane)	Subchronic	0.0003	Liver and kidney toxicity	1,000	IRIS	4.1.2002
Heptachlor	Chronic	0.0005	Increased liver weight	300	IRIS	4.1.2002
Heptachlor epoxide	Chronic	0.000013	Increased liver-to-body weight ratio	1,000	IRIS	4.1.2002
Hexachlorobenzene	Chronic	0.0008	Liver effects	100	IRIS	4.1.2002

Chemical of Potential Concern	Chronic/ Subchronic	Oral RfD (mg/kg-day)	Critical Effect	Combined Uncertainty/ Modifying Factors	Source of RfD	Date of RfD
Iron	n/a	0.3	n/a	n/a	NCEA	8.15.2001
Lead	n/a	n/a	Developmental neurobehavioral effects	n/a	n/a	n/a
Manganese	Chronic	0.14	CNS effects	1	IRIS	4.1.2002
Mercury (as methylmercury)	Chronic	0.0001	Developmental neuropsychological impairment	10	IRIS	4.1.2002
Nickel	Chronic	0.02	Decreased body and organ weights	300	IRIS	4.1.2002
PCBs (based on Aroclor 1254)	Chronic	0.00002	Ocular exudate, inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody response	300	IRIS	4.1.2002
Pentachlorophenol	Chronic	0.03	Liver and kidney pathology	100	IRIS	4.1.2002
Silver	Chronic	0.005	Argyria	3	IRIS	4.1.2002
Thallium chloride	Chronic	0.00008	Altered blood chemistry	3,000	IRIS	4.1.2002
Thallium (by conversion from thallium chloride)	-	0.00007	-	-	-	-
Tributyltin oxide (TBTO)	Chronic	0.0003	Decreased immunologic function	100	IRIS	4.1.2002
Tributyltin (by conversion from TBTO)	-	0.00015	-	-	-	-
Vanadium pentoxide	Chronic	0.009	Decreased hair cystine	100	IRIS	4.1.2002
Vanadium (by conversion from vanadium pentoxide)	-	0.004	-	-	-	-
Zinc	Subchronic	0.3	Altered blood chemistry	3	IRIS	4.1.2002

Sources: IRIS = Integrated Risk Information System, HEAST = Health Effects Assessment Summary Tables, NCEA = EPA's National Center for Exposure Assessment (provisional RfDs)

The IRIS date is the date the database was searched; the HEAST and NCEA date is the date that the EPA Region III RBC table (the source of the HEAST and NCEA values) was updated

Table B-24. Cancer toxicity data (oral/dermal) for chemicals of potential concern

Chemical of Potential Concern	Oral Cancer Slope Factor (kg-day/mg)	Cancer Guideline Description	Source	Date
1,2-Diphenylhydrazine	0.8	B2	IRIS	4.1.2002
2,3,7,8-TCDD	150,000	B2	HEAST	8.15.2001
3,3'-Dichlorobenzidine	0.45	B2	IRIS	4.1.2002
Aldrin	17	B2	IRIS	4.1.2002
alpha-BHC	6.3	B2	IRIS	4.1.2002
Arsenic	1.5	A	IRIS	4.1.2002
Benzidine	230	A	IRIS	4.1.2002
beta-BHC	1.8	B2	IRIS	4.1.2002
bis(2-chloroethyl)ether	1.1	B2	IRIS	4.1.2002
BEHP	0.014	B2	IRIS	4.1.2002
cPAHs (based on benzo[a]pyrene)	7.3	B2	IRIS	4.1.2002
Chlordane	0.35	B2	IRIS	4.1.2002
Dieldrin	16	B2	IRIS	4.1.2002
gamma-BHC (lindane)	1.3	B2	HEAST	8.15.2001
Heptachlor	4.5	B2	IRIS	4.1.2002
Hexachlorobenzene	1.6	B2	IRIS	4.1.2002
Hexachlorobutadiene	0.078	C	IRIS	4.1.2002
N-Nitrosodimethylamine	51	B2	IRIS	4.1.2002
N-Nitroso-di-N-propylamine	7	B2	IRIS	4.1.2002
PCBs	2	B2	IRIS	4.1.2002
Pentachlorophenol	0.12	B2	IRIS	4.1.2002
Toxaphene	1.1	B2	IRIS	4.1.2002

Sources: IRIS = Integrated Risk Information System, HEAST = Health Effects Assessment Summary Tables

Cancer Guideline Description: A = known human carcinogen, B2 = probable human carcinogen, sufficient evidence in animals and inadequate or no evidence in humans, C = possible human carcinogen, limited evidence from animal studies and inadequate or no data in humans.

Table B-25. Toxicological endpoints for COPCs with non-carcinogenic effects

Chemical of Potential Concern	Kidney	Liver	Develop. Effects	Cardio-vascular	Endocrine	Hemat-ologic	Immune Function	Nervous System	Skin
1,2,3-Trichloropropane						X			
2-Nitroaniline						X			
Aldrin		X							
Aluminum									
Antimony					X	X			
Arsenic				X					X
Barium	X								
Benzidine		X							
bis(2-Chloroisopropyl)ether						X			
BEHP		X							
Cadmium	X								
Chlordane		X							
Chromium (hexavalent)									
Copper									
DDT (total)		X							
Dieldrin		X							
gamma-BHC (lindane)	X	X							
Heptachlor		X							
Heptachlor epoxide		X							
Hexachlorobenzene		X							
Iron									
Lead			X					X	
Manganese								X	
Mercury (as methylmercury)			X					X	
Nickel									
PCBs (based on Aroclor 1254)			X				X		
Pentachlorophenol	X	X							

Chemical of Potential Concern	Kidney	Liver	Develop. Effects	Cardio-vascular	Endocrine	Hemat-ologic	Immune Function	Nervous System	Skin
Silver									X
Thallium (by conversion from thallium chloride)						X			
Tributyltin (by conversion from TBTO)							X		
Vanadium (by conversion from vanadium pentoxide)									
Zinc						X			

B.5 Risk Characterization

B.5.1 RISK CHARACTERIZATION EQUATIONS

Carcinogenic risks and noncarcinogenic health effects are evaluated separately due to fundamental differences in their critical toxicity values. Equations for each type of effect are presented in separate sections below.

B.5.1.1 Carcinogenic risks

For chemicals with carcinogenic effects, the risk of cancer is proportional to dose with the assumption that there is no threshold. In other words, there is never a zero probability of cancer risk when exposed to these chemicals at any concentration. Carcinogenic risk probabilities are calculated by multiplying the estimated exposure level by the cancer slope factor (SF) for each chemical.

$$Risk = CDI \times SF \qquad \textbf{(}\textit{Equation 5}\textbf{)}$$

Where:

Risk	=	estimated chemical-specific individual excess lifetime cancer risk (unitless)
CDI	=	chemical-specific chronic daily intake (mg / kg-day)(Eq. 1)
SF	=	route- and chemical-specific carcinogenic slope factor (kg-day / mg)

Cancer risk is expressed as a lifetime excess cancer risk. This concept assumes that the risk of cancer from a given chemical is in "excess" of the background risk of developing cancer (i.e., approximately 1 in 3 chances during a lifetime according to the American Cancer Society).

In assessing carcinogenic risks posed by a site, EPA's National Contingency Plan (NCP; 40 CFR 300) establishes an excess cancer risk of 1×10^{-6} (1 chance in 1 million) as a "point of departure" for establishing remediation goals. Excess cumulative cancer risks lower than 1×10^{-6} are not addressed by the NCP. Where the cumulative cancer risk to an individual based on the RME for current and future land use is less than 1×10^{-4} (1 chance in 10,000), and the noncarcinogenic hazard index (see Section B.5.1.2) is less than one, action generally is not warranted unless there are adverse environmental impacts (EPA 1991b). Excess cumulative cancer risks between 1×10^{-6} and 1×10^{-4} may or may not be considered acceptable, depending on site-specific factors such as the potential for exposure, technical limitations of remediation, and data uncertainties.

Cancer risks are presented in the format of XE-Y, where X is an integer between 1 and 9, E represents an exponent (base 10), and Y is the value (negative) of the exponent. For example, 1E-5 is equivalent to 0.00001, 1×10^{-5} or 1 in 100,000. Cancer risks are

presented with only one significant figure to acknowledge the uncertainty in the underlying cancer slope factors.

B.5.1.2 Noncarcinogenic health effects

Chemicals with noncarcinogenic health effects are generally not toxic below a certain threshold; a critical chemical dose must be exceeded before health effects are observed. The potential for noncarcinogenic health effects is represented by the ratio of a chemical's exposure level and the route-specific RfD, and is expressed as a hazard quotient (HQ).

$$HQ = CDI/RfD \qquad \textbf{(}\textit{Equation 6}\textbf{)}$$

Where:

HQ = estimated chemical-specific hazard quotient (unitless)

CDI = chemical-specific chronic daily intake (mg/kg-day)

RfD = route- and chemical-specific reference dose (mg/kg-day)

The HQ is accepted by EPA as a way to quantify the potential for noncarcinogenic health effects (EPA 1989). HQs are not risk probabilities; the probability an adverse effect will occur does not usually increase linearly with the calculated value. An HQ greater than one may indicate a potential adverse health effect from a chemical exposure, although the same HQ may not equate to the same potential for adverse health effects for all chemicals. HQs may be interpreted by considering the shape and slope of the dose-response curve in the area of observation, the magnitude of uncertainty and modifying factors to the RfD, and the confidence assigned to the RfD by EPA.

HQs for individual COPCs with similar toxicological endpoints may be summed to yield a hazard index (EPA 1989). The hazard index (HI) is an expression of the additivity of noncarcinogenic health effects. An HI is calculated by summing HQs for chemicals with similar toxicological endpoints, as described in Table B-23. HIs were calculated for the following endpoints: liver, kidney, hematologic, immune function, nervous system, developmental effects, and skin. HIs were not calculated for two other endpoints shown in Table B-23 (cardiovascular and endocrine) because only a single COPC is listed for each endpoint.

B.5.2 RISK CHARACTERIZATION FORMAT

Cancer risks and HQs are presented according to the format recommended in EPA (1998a). Since the primary purpose of the Phase 1 HHRA is to identify candidate sites for early remedial action, risks are characterized in this section only for detected chemicals. A number of COPCs that have not been detected were identified for each exposure pathway because detection limits exceeded RBCs. Risks to these COPCs are

characterized in the uncertainty assessment (Section B.6). Risks attributable to these undetected chemicals have very high uncertainty and would have little meaning for identifying candidate sites for early action.

Risks and HQs for six exposure scenarios are presented in this section:

- netfishing – RME, Tables B-26a and B-27a
- netfishing – CT, Tables B-26b and B-27b
- seafood consumption – adult tribal RME, Tables B-28a and B-29a
- seafood consumption – child tribal RME, Tables B-28b and B-29b
- seafood consumption – adult API RME, Tables B-28c and B-29c
- beach play – RME, Tables B-30a and B-30b

Cancer risks are summed for all chemicals within each exposure scenario. Exposure scenarios where the same receptor is exposed via multiple pathways simultaneously were addressed by summing RME estimates for all pathways. This approach was applied to the children's beach play dermal and ingestion scenarios. For exposure scenarios where the same receptor is exposed via multiple pathways but not simultaneously, the RME exposure estimates for the highest exposure pathway were combined with CT estimates for the other pathways. This was intended to prevent an overestimate of risks resulting from summing multiple RMEs. Adult tribal exposures via netfishing and non-commercial seafood consumption were evaluated by adding risks for the RME tribal seafood consumption pathway with risks from the CT netfishing dermal and ingestion pathways. Risks associated with surface water recreation, although not explicitly estimated in this HHRA, were also considered as part of the cumulative risk evaluation. Risk estimates calculated by King County (1999b) for highly exposed adult and child swimmers were added to the sum described above for the seafood and netfishing pathways.

HQs are summed within each exposure pathway and scenario to form an HI for chemicals with similar toxicological endpoints, as defined in Table B-25. COPCs with cancer risks greater than 1E-6 or HQs greater than 1 are considered chemicals of concern (COCs).

B.5.3 RISK CHARACTERIZATION RESULTS

The results are presented below for each exposure scenario group: Section B.5.3.1 (netfishing), Section B.5.3.2 (seafood consumption), and Section B.5.3.3 (beach play).

B.5.3.1 Netfishing

For the netfishing RME scenario, the total cancer risk for both ingestion and dermal exposure routes was 7E-6 (Table B-26a). The risk from ingestion was double the risk from the dermal route. Approximately two-thirds of the total cancer risk from both dermal and ingestion exposure was attributable to arsenic. At least a portion of the

risk attributed to arsenic is likely to be from sources outside the LDW and from naturally occurring background. Background issues associated with arsenic are discussed further in the uncertainty assessment (Section B.6.1.1). The total cancer risk estimate for the combined exposure routes for the CT scenario was 2E-6 (Table B-26b). The relative risk between the two exposure routes and between arsenic and the other COPCs is similar to the netfishing RME scenario.

HQs from both exposure routes were all less than 1 (Tables B-27a and B-27b). The total hazard index across all exposure routes for all chemicals was 0.07 for the netfishing RME scenario and 0.04 for the netfishing CT scenario. Endpoint-specific HIs were not calculated since the total HI across all endpoints was less than 1.

Arsenic was the only COC identified for the netfishing exposure scenarios.

Table B-26a. Calculation of cancer risks for netfishing exposure scenario (RME)

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure medium: Sediment
Receptor Population: Commercial fishermen
Receptor Age: Adult

Exposure Route	COPC	EPC (mg/kg) [a]	Cancer CDI (mg/kg-day)	Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Cancer Risk
Ingestion	2,3,7,8-TCDD (TEQ)	0.000033	4E-12	150,000	6E-7
	Arsenic	13	2E-6	1.5	3E-6
	cPAHs (TEQ)	0.49	6E-8	7.3	5E-7
	Dieldrin	0.0079	1E-9	16	2E-8
	PCBs (total)	0.49	6E-8	2	1E-7
	Total				4E-6
Dermal	2,3,7,8-TCDD (TEQ)	0.000033	2E-12	150,000	3E-7
	Arsenic	13	7E-7	1.5	1E-6
	cPAHs (TEQ)	0.49	1E-7	7.3	9E-7
	Dieldrin	0.0079	1E-9	16	2E-9
	PCBs (total)	0.49	1E-7	2	3E-7
	Total				3E-6
		Total risk for ingestion and dermal exposure routes			**7E-6**

[a] All EPCs are medium-specific, rather than route-specific

Table B-26b. Calculation of cancer risks for netfishing exposure scenario (CT)

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure medium: Sediment
Receptor Population: Commercial fishermen
Receptor Age: Adult

Exposure Route	COPC	EPC (mg/kg) [a]	Cancer CDI (mg/kg-day)	Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Cancer Risk
Ingestion	2,3,7,8-TCDD (TEQ)	0.000033	1E-12	150,000	2E-7
	Arsenic	13	6E-7	1.5	9E-7
	cPAHs (TEQ)	0.49	2E-8	7.3	2E-7
	Dieldrin	0.0079	4E-10	16	6E-9
	PCBs (total)	0.49	2E-8	2	4E-8
	Total				1E-6
Dermal	2,3,7,8-TCDD (TEQ)	0.000033	6E-13	150,000	1E-7
	Arsenic	13	3E-7	1.5	4E-7
	cPAHs (TEQ)	0.49	4E-8	7.3	3E-7
	Dieldrin	0.0079	5E-11	16	8E-9
	PCBs (total)	0.49	4E-8	2	9E-8
	Total				9E-7
		Total risk for ingestion and dermal exposure routes			**2E-6**

[a] All EPCs are medium-specific, rather than route-specific

Table B-27a. Calculation of non-cancer hazards for netfishing exposure scenario (RME)

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure medium: Sediment
Receptor Population: Commercial fishermen
Receptor Age: Adult

Exposure Route	COPC	EPC (mg/kg) [a]	Noncancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
Ingestion	Aluminum	19,000	4E-3	1	0.004
	Antimony	5.4	1E-6	0.0004	0.003
	Arsenic	13	3E-6	0.0003	0.009
	Barium	160	3E-5	0.07	0.005
	Cadmium	0.52	1E-7	0.001	0.0001
	Chromium	30	6E-6	0.003	0.002
	Copper	65	1E-5	0.04	0.0003
	Dieldrin	0.0079	2E-9	0.00005	0.00003
	Iron	27,000	6E-3	0.6	0.009
	Manganese	320	7E-5	0.14	0.0005
	PCBs (total)	0.49	1E-7	0.00002	0.005
	Total				0.04
Dermal	Aluminum	19,000	6E-4	1	0.0006
	Antimony	5.4	2E-7	0.0004	0.003
	Arsenic	13	1E-6	0.0003	0.004
	Barium	160	5E-6	0.07	0.001
	Cadmium	0.52	2E-8	0.001	0.0006
	Chromium	30	9E-7	0.003	0.01
	Copper	65	2E-6	0.04	0.00005
	Dieldrin	0.0079	2E-9	0.00005	0.00005
	Iron	27,000	8E-4	0.6	0.001
	Manganese	320	1E-5	0.14	0.002
	PCBs (total)	0.49	2E-7	0.00002	0.001
	Total				0.03
	Total hazard index for ingestion and dermal exposure routes				**0.07**

[a] All EPCs are medium-specific, rather than route-specific

Table B-27b. Calculation of non-cancer hazards for netfishing exposure scenario (CT)

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure medium: Sediment
Receptor Population: Commercial fishermen
Receptor Age: Adult

EXPOSURE ROUTE	COPC	EPC (mg/kg) [a]	NONCANCER CDI (mg/kg-day)	REFERENCE DOSE (mg/kg-day)	HAZARD QUOTIENT
Ingestion	Aluminum	19,000	2E-3	1	0.002
	Antimony	5.4	6E-7	0.0004	0.001
	Arsenic	13	1E-6	0.0003	0.005
	Barium	160	2E-5	0.07	0.0002
	Cadmium	0.52	6E-8	0.001	0.00006
	Chromium	30	3E-6	0.003	0.001
	Copper	65	7E-6	0.04	0.0002
	Dieldrin	0.0079	9E-10	0.00005	0.00002
	Iron	27,000	3E-3	0.6	0.005
	Manganese	320	3E-5	0.14	0.0003
	PCBs (total)	0.49	5E-8	0.00002	0.003
	Total				0.02
Dermal	Aluminum	19,000	3E-4	1	0.0003
	Antimony	5.4	8E-8	0.0004	0.001
	Arsenic	13	6E-7	0.0003	0.002
	Barium	160	3E-6	0.07	0.0005
	Cadmium	0.52	8E-9	0.001	0.0003
	Chromium	30	5E-7	0.003	0.006
	Copper	65	1E-6	0.04	0.00003
	Dieldrin	0.0079	1E-9	0.00005	0.000002
	Iron	27,000	4E-4	0.6	0.0007
	Manganese	320	5E-6	0.14	0.0009
	PCBs (total)	0.49	1E-7	0.00002	0.005
	Total				0.02
	Total hazard index for ingestion and dermal exposure routes				**0.04**

[a] All EPCs are medium-specific, rather than route-specific

B.5.3.2 Seafood consumption

Total cancer risk estimates for the three different seafood consumption scenarios were 2E-3 for adult tribal RME (Table B-28a), 5E-4 for child tribal RME (Table B-28b), and 9E-5 for adult API RME (Table B-28c) . Most of the total cancer risk was attributable to arsenic. At least a portion of the risk attributed to arsenic is likely to be from naturally

occurring background sources. Background issues associated with arsenic will be discussed further in the uncertainty assessment (Section B.6.1.1).

Estimated cancer risks for the adult API RME scenario (Table B-28c) were based on composite consumption rates for the 10 different ethnicities surveyed in EPA (1999c). The species group-specific consumption rates for each ethnic group vary, as does the percentage of self-caught fish from King County, and body weight. Excess cancer risk estimates were within a range of less than an order of magnitude across ethnic groups, i.e., from a low of 2E-5 for Filipinos to a high of 1E-4 for Japanese.

Total HIs for all chemicals were 15 for adult tribal RME (Table B-29a), 40 for child tribal RME (Table B-29b), and 1.8 for adult API RME (Table B-29c) . The majority of the total HI was attributable to PCBs, although arsenic, mercury, and TBT had HQs greater than 1 for at least one scenario. These 4 COPCs were identified as COCs for the seafood consumption scenarios.

Three endpoint-specific HIs were also calculated for the seafood consumption pathway. DDTs and BEHP both share a common toxicological endpoint (liver function), as do PCBs and TBT (immune function), and PCBs and mercury (developmental effects). The other chemicals with non-cancer effects do not share a common endpoint. The liver HI was much less than 1 for all three scenarios. The immune function HIs were 10, 25, and 1.3 for the adult tribal RME, child tribal RME, and adult API RME scenarios, respectively (Tables B-29a, B-29b, and B-29c). The developmental HIs were very similar to, but slightly higher, than the immunological HIs.[20]

Table B-28a. Calculation of cancer risks for seafood consumption scenario (RME tribal adult)

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor Population: Tribal/subsistence fish and shellfish consumers
Receptor Age: Adult

Exposure Route	COPC	EPC (mg/kg) [a]	Cancer CDI (mg/kg-day)	Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Cancer Risk
Ingestion	Arsenic	[b]	8E-4	1.5	1E-3
	BEHP	[b]	2E-5	0.014	3E-7
	cPAHs (TEQ)	[b]	2E-5	7.3	1E-4
	PCBs (total)	[b]	2E-4	2	3E-4

[20] The hazard indicies for immunological effects are based on the RfD of 0.00002 mg/kg-day for Aroclor 1254, which is the most protective of the EPA RfDs for PCBs. This reference is also protective of developmental effects, which occur at higher dose levels. For example, ATSDR (2000b) has derived an intermediate-duration maximum risk level (MRL) for PCBs of 0.00003 mg/kg-day based on the lowest adverse effect level of neurobehavioral effects in infant monkeys exposed to PCBs from birth to 20 weeks of age. Thus, application of the EPA RfD is protective for both immunological and developmental endpoints related to PCB exposure.

		Total risk across all exposure routes/pathways	**2E-3**

[a] All EPCs are medium-specific, rather than route-specific

[b] Risk estimates made from four EPCs (see Tables B-14a to B-14d)

Table B-28b. Calculation of cancer risks for seafood consumption scenario (RME tribal child)

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor Population: Tribal/subsistence fish and shellfish consumers
Receptor Age: Child

EXPOSURE ROUTE	COPC	EPC (mg/kg) [a]	CANCER CDI (mg/kg-day)	CANCER SLOPE FACTOR $(mg/kg\text{-}day)^{-1}$	CANCER RISK
Ingestion	Arsenic	[b]	3E-4	1.5	4E-4
	BEHP	[b]	2E-6	0.014	3E-8
	cPAHs (TEQ)	[b]	5E-6	7.3	3E-5
	PCBs (total)	[b]	4E-5	2	8E-5
		Total risk across all exposure routes/pathways			**5E-4**

[a] All EPCs are medium-specific, rather than route-specific

[b] Risk estimates made from four EPCs (see Tables B-14a to B-14d)

Table B-28c. Calculation of cancer risks for seafood consumption scenario (adult API RME)

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor Population: Asian and Pacific Islander fish and shellfish consumers
Receptor Age: Adult

EXPOSURE ROUTE	COPC	EPC (mg/kg) [a]	CANCER CDI (mg/kg-day)	CANCER SLOPE FACTOR $(mg/kg\text{-}day)^{-1}$	CANCER RISK
Ingestion	Arsenic	[b]	4E-5	1.5	6E-5
	BEHP	[b]	1E-6	0.014	2E-8
	cPAHs (TEQ)	[b]	8E-7	7.3	6E-6
	PCBs (total)	[b]	1E-5	2	2E-5
		Total risk across all exposure routes/pathways			**9E-5**

[a] All EPCs are medium-specific, rather than route-specific

[b] Risk estimates made from three EPCs (see B-14a, B-14b, B-14e)

Table B-29a. Calculation of non-cancer hazards for seafood consumption scenario (RME tribal adult)

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor Population: Tribal/subsistence fish and shellfish consumers
Receptor Age: Adult

Exposure Route	COPC	EPC (mg/kg) [a]	Non-cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
Ingestion	Arsenic	[b]	1E-3	0.0003	3.2
	BEHP	[b]	3E-5	0.02	0.002
	Cadmium	[b]	7E-5	0.001	0.07
	Chromium	[b]	1E-4	0.003	0.04
	Copper	[b]	9E-3	0.04	0.23
	DDTs (total)	[b]	1E-6	0.0005	0.003
	Mercury	[b]	9E-5	0.0001	0.89
	PCBs (total)	[b]	2E-4	0.00002	10
	Tributyltin	[b]	5E-5	0.00015	0.34
	Zinc	[b]	3E-2	0.3	0.09
	Hazard index for liver endpoint (BEHP and DDTs)				**0.005**
	Hazard index for immunological endpoint (PCBs and TBT)				**10**
	Hazard index for developmental endpoint (PCBs and mercury)				**11**
	Total hazard index across all exposure routes/pathways				**15**

[a] All EPCs are medium-specific, rather than route-specific

[b] Risk estimates made from four EPCs (see Tables B-14a to B-14d)

Table B-29b. Calculation of non-cancer hazards for seafood consumption scenario (RME tribal child)

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor Population: Tribal/subsistence fish and shellfish consumers
Receptor Age: Child

Exposure Route	COPC	EPC (mg/kg) [a]	Non-cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
Ingestion	Arsenic	[b]	3E-3	0.0003	10
	BEHP	[b]	2E-5	0.02	0.001
	Cadmium	[b]	6E-5	0.001	0.06
	Chromium	[b]	4E-4	0.003	0.13
	Copper	[b]	4E-2	0.04	0.93
	DDTs (total)	[b]	4E-7	0.0005	0.0009
	Mercury	[b]	3E-4	0.0001	2.8
	PCBs (total)	[b]	5E-4	0.00002	24
	Tributyltin	[b]	2E-4	0.00015	1.3

Exposure Route	COPC	EPC (mg/kg) [a]	Non-cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
	Zinc	[b]	9E-2	0.3	0.31
	Hazard index for liver endpoint (BEHP and DDTs)				**0.002**
	Hazard index for immunological endpoint (PCBs and TBT)				**25**
	Hazard index for developmental endpoint (PCBs and mercury)				**27**
	Total hazard index across all exposure routes/pathways				**40**

[a] All EPCs are medium-specific, rather than route-specific

[b] Risk estimates made from four EPCs (see Tables B-14a to B-14d)

Table B-29c. Calculation of non-cancer hazards for seafood consumption scenario (adult API RME)

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure medium: Fish and shellfish tissue
Receptor Population: Asian and Pacific Islander fish and shellfish consumers
Receptor Age: Adult

Exposure Route	COPC	EPC (mg/kg) [a]	Non-cancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
Ingestion	Arsenic	[b]	9E-5	0.0003	0.31
	BEHP	[b]	3E-6	0.02	0.0001
	Cadmium	[b]	6E-6	0.001	0.006
	Chromium	[b]	1E-5	0.003	0.004
	Copper	[b]	8E-4	0.04	0.02
	DDTs (total)	[b]	2E-7	0.0005	0.0004
	Mercury	[b]	1E-5	0.0001	0.10
	PCBs (total)	[b]	3E-5	0.00002	1.3
	Tributyltin	[b]	5E-6	0.00015	0.03
	Zinc	[b]	2E-3	0.3	0.008
	Hazard index for liver endpoint (BEHP and DDTs)				**0.0005**
	Hazard index for immunological endpoint (PCBs and TBT)				**1.3**
	Hazard index for developmental endpoint (PCBs and mercury)				**1.4**
	Total hazard index across all exposure routes/pathways				**1.8**

[a] All EPCs are medium-specific, rather than route-specific

[b] Risk estimates made from three EPCs (see Tables B-14a, B-14b, B-14e)

B.5.3.3 Beach play

Cancer risks and non-cancer hazards were estimated separately for three intertidal exposure areas: Kellogg Island, southeast LDW, and southwest LDW. Total cancer risks for all exposure routes and chemicals were 5E-6 for Kellogg Island, 6E-6 for southeast, and 5E-6 for southwest (Table B-30a). Approximately half the total cancer risk was attributable to arsenic. Most of the remainder was attributable to 2,3,7,8-TCDD TEQ (cancer risk of 1E-6). At least a portion of the risk attributed to arsenic is

likely to be from background sources outside the LDW. Background issues associated with arsenic will be discussed further in the uncertainty assessment (Section B.6.1.1). Identical cancer risk estimates were made for 2,3,7,8-TCDD TEQ in each intertidal area because data were insufficient in any one area to calculate an area-weighted concentration. This chemical, along with arsenic, was identified as a COC for the beach play scenario, but the uncertainty of this conclusion is high since it is based on a relatively small dataset (29 samples) compared to other COPCs.

Hazard quotients were less than 1 for all chemicals in all exposure routes (Table B-30b). Total HIs for all chemicals were 0.37 for Kellogg Island, 0.62 for southeast, and 0.44 for southwest. Endpoint-specific HIs were not calculated since the total HI across all endpoints was less than 1.

Table B-30a. Calculation of cancer risks for beach play exposure scenario (RME)

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure medium: Sediment
Receptor Population: Residents
Receptor Age: Child

Exposure Route	COPC	EPC (mg/kg) [a]	CANCER CDI (mg/kg-day)	CANCER SLOPE FACTOR $(mg/kg\text{-}day)^{-1}$	CANCER RISK
Kellogg Island intertidal exposure area					
Ingestion	2,3,7,8-TCDD (TEQ)	0.000086	9E-12	150,000	1E-6
	Arsenic	12	1E-6	1.5	2E-6
	cPAHs (TEQ)	0.59	6E-8	7.3	5E-7
	PCBs (total)	0.21	2E-8	2	4E-8
	Total				4E-6
Dermal	2,3,7,8-TCDD (TEQ)	0.000086	2E-12	150,000	3E-7
	Arsenic	12	3E-7	1.5	4E-7
	cPAHs (TEQ)	0.59	6E-8	7.3	4E-7
	PCBs (total)	0.21	2E-8	2	5E-8
	Total				1E-6
		Total risk for ingestion and dermal exposure routes			**5E-6**
Southeast intertidal exposure area					
Ingestion	2,3,7,8-TCDD (TEQ)	0.000086	9E-12	150,000	1E-6
	Arsenic	12	1E-6	1.5	2E-6
	cPAHs (TEQ)	0.83	9E-8	7.3	6E-7
	Dieldrin	0.15	2E-8	16	3E-7
	Hexachlorobenzene	0.021	2E-9	1.6	4E-9
	PCBs (total)	2.2	2E-7	2	5E-7
	Total				4E-6
Dermal	2,3,7,8-TCDD (TEQ)	0.000086	2E-12	150,000	3E-7
	Arsenic	12	3E-7	1.5	4E-7

Exposure Route	COPC	EPC (mg/kg) [a]	Cancer CDI (mg/kg-day)	Cancer Slope Factor $(mg/kg\text{-}day)^{-1}$	Cancer Risk
	cPAHs (TEQ)	0.83	8E-8	7.3	6E-7
	Dieldrin	0.15	1E-8	16	2E-7
	Hexachlorobenzene	0.021	2E-9	1.6	3E-9
	PCBs (total)	2.2	2E-7	2	5E-7
	Total				2E-6
		Total risk for ingestion and dermal exposure routes			**6E-6**
Southwest intertidal exposure area					
Ingestion	2,3,7,8-TCDD (TEQ)	0.000086	9E-12	150,000	1E-6
	Arsenic	9.7	1E-6	1.5	2E-6
	cPAHs (TEQ)	0.36	4E-8	7.3	3E-7
	Hexachlorobenzene	0.042	4E-9	1.6	7E-9
	PCBs (total)	1.1	1E-7	2	2E-7
	Total				4E-6
Dermal	2,3,7,8-TCDD (TEQ)	0.000086	2E-12	150,000	3E-7
	Arsenic	9.7	2E-7	1.5	3E-7
	cPAHs (TEQ)	0.36	4E-8	7.3	3E-7
	Hexachlorobenzene	0.042	3E-9	1.6	5E-9
	PCBs (total)	1.1	1E-7	2	2E-7
	Total				1E-6
		Total risk for ingestion and dermal exposure routes			**5E-6**

[a] All EPCs are medium-specific, rather than route-specific

Table B-30b. Calculation of non-cancer hazards for beach play exposure scenario (RME)

Scenario Timeframe: Current/Future
Medium: Sediment
Exposure medium: Sediment
Receptor Population: Resident
Receptor Age: Child

Exposure Route	COPC	EPC (mg/kg) [a]	Noncancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
Kellogg Island intertidal exposure area					
Ingestion	Aluminum	17,000	2E-2	1	0.02
	Antimony	6.1	8E-6	0.0004	0.02
	Arsenic	12	1E-5	0.0003	0.05
	Barium	75	9E-5	0.07	0.001
	Cadmium	0.48	6E-7	0.001	0.0006
	Chromium	34	4E-5	0.003	0.01
	Copper	62	8E-5	0.04	0.002

Exposure Route	COPC	EPC (mg/kg) [a]	Noncancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
	DDTs (total)	no data	no data	0.005	no data
	Heptachlor epoxide	0.001	1E-9	0.000013	0.0001
	Iron	35,000	4E-2	0.6	0.07
	Manganese	230	3E-4	0.14	0.002
	Mercury	0.15	2E-7	0.0001	0.002
	Nickel	25	3E-5	0.02	0.002
	PCBs (total)	0.21	3E-7	0.00002	0.01
	Silver	0.53	7E-7	0.005	0.0001
	Thallium	0.16	2E-7	0.00007	0.003
	Vanadium	59	7E-5	0.004	0.02
	Zinc	130	2E-4	0.3	0.0005
	Total				0.21
Dermal	Aluminum	17,000	2E-3	1	0.002
	Antimony	6.1	6E-7	0.0004	0.009
	Arsenic	12	3E-6	0.0003	0.01
	Barium	75	7E-6	0.07	0.001
	Cadmium	0.48	4E-8	0.001	0.002
	Chromium	34	3E-6	0.003	0.04
	Copper	62	6E-6	0.04	0.0001
	DDTs (total)	no data	no data	0.005	no data
	Heptachlor epoxide	0.001	9E-10	0.000013	0.00007
	Iron	35,000	3E-3	0.6	0.005
	Manganese	230	2E-5	0.14	0.004
	Mercury	0.15	1E-8	0.0001	0.002
	Nickel	25	2E-6	0.02	0.003
	PCBs (total)	0.21	3E-7	0.00002	0.01
	Silver	0.53	5E-8	0.005	0.0002
	Thallium	0.16	1E-8	0.00007	0.0002
	Vanadium	58	5E-6	0.004	0.05
	Zinc	130	1E-5	0.3	0.00004
	Total				0.16
	Total hazard index for ingestion and dermal exposure routes				**0.37**
Southeast intertidal exposure area					
Ingestion	Aluminum	18,000	2E-2	1	0.02
	Antimony	7.6	9E-6	0.0004	0.02
	Arsenic	12	1E-5	0.0003	0.05

Exposure Route	COPC	EPC (mg/kg) [a]	Noncancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
	Barium	79	1E-4	0.07	0.001
	Cadmium	1.5	2E-6	0.001	0.002
	Chromium	44	5E-5	0.003	0.02
	Copper	110	1E-4	0.04	0.003
	DDTs (total)	1.5	2E-6	0.005	0.004
	Dieldrin	0.15	2E-7	0.00005	0.004
	Hexachlorobenzene	0.021	3E-8	0.0008	0.00003
	Iron	26,000	3E-2	0.6	0.05
	Manganese	350	4E-4	0.14	0.003
	Mercury	0.17	2E-7	0.0001	0.002
	Nickel	31	4E-5	0.02	0.002
	PCBs (total)	2.2	3E-6	0.00002	0.1
	Silver	1.4	2E-6	0.005	0.0004
	Thallium	3.1	4E-6	0.00007	0.06
	Vanadium	57	7E-5	0.004	0.02
	Zinc	190	2E-4	0.3	0.0008
	Total				0.37
Dermal	Aluminum	18,000	2E-3	1	0.002
	Antimony	7.6	7E-7	0.0004	0.01
	Arsenic	12	3E-6	0.0003	0.01
	Barium	79	7E-6	0.07	0.001
	Cadmium	1.5	1E-7	0.001	0.005
	Chromium	44	4E-6	0.003	0.05
	Copper	110	1E-5	0.04	0.0003
	DDTs (total)	1.5	4E-7	0.005	0.0008
	Dieldrin	0.15	1E-7	0.00005	0.003
	Hexachlorobenzene	0.021	2E-8	0.0008	0.00002
	Iron	26,000	2E-3	0.6	0.004
	Manganese	350	3E-5	0.14	0.006
	Mercury	0.17	2E-8	0.0001	0.002
	Nickel	31	3E-6	0.02	0.004
	PCBs (total)	2.2	3E-6	0.00002	0.1
	Silver	1.4	1E-7	0.005	0.0006
	Thallium	3.1	3E-7	0.00007	0.004
	Vanadium	57	5E-6	0.004	0.05
	Zinc	190	2E-5	0.3	0.00006

Exposure Route	COPC	EPC (mg/kg) [a]	Noncancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
	Total				0.25
	Total hazard index for ingestion and dermal exposure routes				**0.62**
Southwest intertidal exposure area					
Ingestion	Aluminum	15,000	2E-2	1	0.02
	Antimony	5.4	7E-6	0.0004	0.02
	Arsenic	9.7	1E-5	0.0003	0.04
	Barium	55	7E-5	0.07	0.001
	Cadmium	0.17	2E-7	0.001	0.0002
	Chromium	23	3E-5	0.003	0.01
	Copper	31	4E-5	0.04	0.001
	Hexachlorobenzene	0.042	5E-8	0.0008	0.00007
	Iron	24,000	3E-2	0.6	0.05
	Manganese	380	5E-4	0.14	0.004
	Mercury	0.1	1E-7	0.0001	0.001
	Nickel	17	2E-5	0.02	0.001
	PCBs (total)	1.1	1E-6	0.00002	0.07
	Silver	0.14	2E-7	0.005	0.00003
	Thallium	0.061	8E-8	0.00007	0.01
	Vanadium	52	6E-5	0.004	0.02
	Zinc	80	1E-4	0.3	0.0003
	Total				0.25
Dermal	Aluminum	15,000	1E-3	1	0.001
	Antimony	5.4	5E-7	0.0004	0.008
	Arsenic	9.7	3E-6	0.0003	0.009
	Barium	55	5E-6	0.07	0.001
	Cadmium	0.17	2E-8	0.001	0.0006
	Chromium	23	2E-6	0.003	0.03
	Copper	31	3E-6	0.04	0.00007
	Hexachlorobenzene	0.042	4E-8	0.0008	0.00005
	Iron	24,000	2E-3	0.6	0.004
	Manganese	380	3E-5	0.14	0.006
	Mercury	0.1	9E-9	0.0001	0.001
	Nickel	17	2E-6	0.02	0.002
	PCBs (total)	1.1	1E-6	0.00002	0.07
	Silver	0.14	1E-8	0.005	0.00006
	Thallium	0.061	6E-9	0.00007	0.00008

Exposure Route	COPC	EPC (mg/kg) [a]	Noncancer CDI (mg/kg-day)	Reference Dose (mg/kg-day)	Hazard Quotient
	Vanadium	52	5E-6	0.004	0.05
	Zinc	80	7E-6	0.3	0.00002
	Total				0.19
	Total hazard index for ingestion and dermal exposure routes				**0.44**

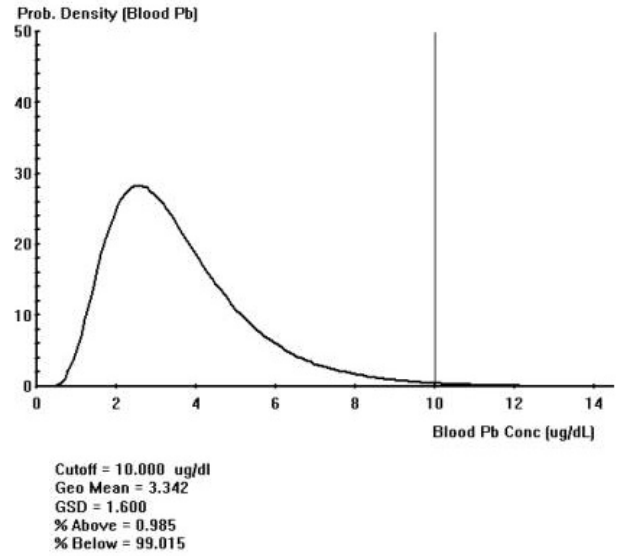
[a] All EPCs are medium-specific, rather than route-specific

B.5.4 LEAD

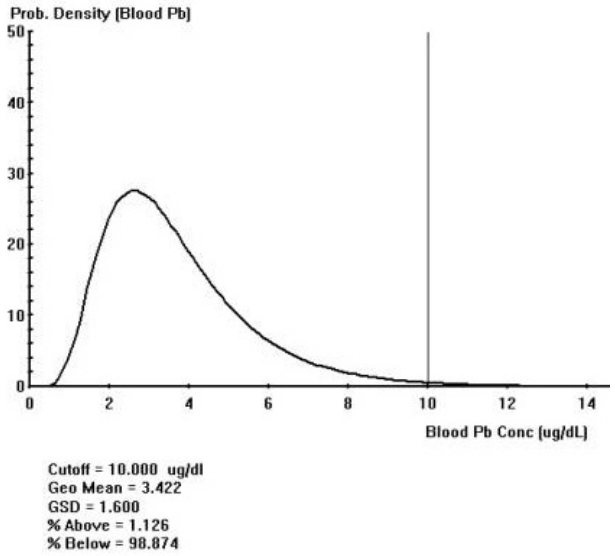
The results of lead modeling for both children (IEUBK) and adults (ALM) are presented in separate sections below.

B.5.4.1 Children (IEUBK)

The IEUBK lead model was run using default parameters except for the input data shown in Table B-17. Model output is in the form of a probability density curve that represents predicted blood lead concentrations to a hypothetical population of children. The US Centers for Disease Control and Prevention (CDC) has established 10 µg/dL as a level of concern above which appropriate medical followup is warranted. The probability density curves generated using 1) high estimates of sediment/soil (192 mg/kg dw) and fish (0.27 mg/kg) lead concentrations and 2) low estimates of soil (184 mg/kg dw) and fish (0.19 mg/kg) lead concentrations are shown in Figure B-3. Based on these input data, 1.1 and 0.99% of the modeled population would have blood lead levels exceeding the CDC level of concern. Because 5% is often used as a threshold of concern in risk assessments, lead will not be further investigated in Phase 2.



a) lower lead input data



b) higher lead input data

Figure B-3. Probability density curve for predicted blood lead concentrations using input data from beach play scenario and child seafood consumption scenario

B.5.4.2 Adults (ALM)

The adult lead model was run to estimate risks to the most sensitive population, which is a developing fetus. These risks were assessed by estimating the probability of exceeding a target blood lead level of 10 $\mu g / dL$ for the fetus. Results for both low and high estimates (based on high and low concentrations in sediment / soil and in fish and shellfish) are provided in Table B-31.

Table B-31. Results of adult lead model for both low and high exposure scenarios

OUTPUT	UNITS	LOW ESTIMATE [a]	HIGH ESTIMATE [b]
Exposure from sediment/soil only			
Predicted blood levels, central tendency	µg/dL	1.7	1.8
Predicted blood levels, 95th percentile	µg/dL	6.1	6.3
Probability of exceeding 10 µg/dL	%	1.3	1.4
Exposure from sediment/soil and seafood			
Predicted blood levels, central tendency	µg/dL	1.8	1.9
Predicted blood levels, 95th percentile	µg/dL	6.3	6.6
Probability of exceeding 10 µg/dL	%	1.4	1.6

[a] Based on lowest sediment EPC and sole lead data as a surrogate for perch

[b] Based on highest sediment EPC and mussel lead data as a surrogate for perch

Calculations performed for exposure to sediment and fish at the LDW in addition to background dietary and environmental exposures indicated that risks of elevated fetal blood lead levels are well within regulatory guidelines to protect public health. The differences between the low and high estimates, and the sediment only vs. sediment plus seafood consumption were very small (Table B-31).

B.5.5 RISK CHARACTERIZATION SUMMARY

The risk and non-cancer hazard estimates are summarized in Table B-32. For the purposes of brevity, chemical-specific risk and HQ estimates are provided only for chemicals identified as COCs (i.e., exceeding a cancer risk estimate of 1E-6 or an HQ of 1). The highest cancer risks and non-cancer hazard estimates were calculated for the seafood consumption pathways. The total cancer risk estimate ranged from 9E-5 for the adult API RME scenario to 2E-3 for the adult tribal RME scenario. Total cancer risks for the direct sediment exposure pathways (e.g., netfishing and beach play) were greater than 1E-6, but less than 1E-5.

The sum of cancer risk estimates for the adult tribal RME seafood consumption and adult CT netfishing scenarios are the same as the cancer risk estimates for the adult

tribal RME seafood consumption scenario alone, because the estimates from the seafood consumption scenario are so much higher than the estimates from the netfishing scenario (i.e., the netfishing scenario results do not change the results for the sum of netfishing and seafood consumption after rounding the sum to one significant figure, as recommended by EPA [1989]). Similarly, adding the risk estimates from King County (1999b) for highly exposed adult and child swimmers to the above sum does not change the total because of the much smaller magnitude of risks from swimming as compared to risks from seafood consumption; again, the increment from swimming is not seen after rounding the sum to one significant figure.

For the exposure scenarios evaluated in the Phase 1 HHRA, the following chemicals were identified as COCs based on their exceedance of a cancer risk estimate of 1E-6 or a non-cancer HQ of 1: PCBs, arsenic, cPAHs, TCDD TEQs, TBT, and mercury. The Phase 1 HHRA results were used in the early action site identification process (Windward 2002a), and were also useful for identifying data gaps that may be filled for the Phase 2 baseline HHRA (Windward 2002b).

Table B-32. Summary of risk and non-cancer hazard estimates

Medium	Exposure Medium	Exposure Scenario	Cancer Risk				Noncancer HQs			
			Chemical	Ingestion	Dermal	Exposure Route Total	Chemical	Ingestion	Dermal	Exposure Route Total
Sediment	Sediment	Netfishing, adult RME	Arsenic	3E-6	1E-6	4E-6				
			Total	4E-6	3E-6	7E-6	Total [a]	0.04	0.03	0.07
		Netfishing, adult CT	Total	1E-6	9E-7	2E-6	Total [a]	0.02	0.02	0.04
		Beach play, Kellogg Island	Arsenic	2E-6	4E-7	2E-6				
			TCDD	1E-6	3E-7	1E-6				
			Total	4E-6	1E-6	5E-6	Total [a]	0.21	0.16	0.37
		Beach play, southeast	Arsenic	2E-6	4E-7	2E-6				
			TCDD	1E-6	3E-7	1E-6				
			Total	4E-6	2E-6	6E-6	Total [a]	0.37	0.25	0.62
		Beach play, southwest	Arsenic	2E-6	3E-7	2E-6				
			TCDD	1E-6	3E-7	1E-6				
			Total	4E-6	1E-6	5E-6	Total [a]	0.25	0.19	0.44
		Swimming, highly exposed adults [b]	Arsenic	4E-7	7E-7	1E-6 [c]				
			Total	5E-7	8E-7	2E-6 [c]	Total [a]	0.001	0.002	0.004 [c]
		Swimming, highly exposed children [b]	Arsenic	4E-7	4E-6	4E-6 [c]				
			Total	4E-7	5E-6	6E-6 [c]	Total [a]	0.012	0.13	0.15 [c]
Sediment	Fish/ shellfish tissue	Consumption, adult tribal RME	Arsenic	1E-3		1E-3	Arsenic	3.2		3.2
			cPAHs	1E-4		1E-4				
			PCBs	3E-4		3E-4	PCBs	10		10
			Total	2E-3		2E-3	Total [a]	15		15
		Consumption, child tribal RME	Arsenic	4E-4		4E-4	Arsenic	10		10
			cPAHs	3E-5		3E-5	PCBs	24		24
			PCBs	8E-5		8E-5	TBT	1.3		1.3
							Mercury	2.8		2.8
			Total	5E-4		5E-4	Total [a]	40		40
		Consumption, adult API RME	Arsenic	6E-5		6E-5				
			cPAHs	6E-6		6E-6				
			PCBs	2E-5		2E-5				
							Mercury	1.3		1.3
			Total	9E-5		9E-5	Total	1.8		1.8

Note: Exposure route total chemical-specific cancer risk and HQ estimates less than 1E-6 and 1, respectively, are not shown in this table. See Section B.5.3 for all estimates.

cPAH = carcinogenic PAHs (TEQ)

[a] Total is for all chemicals, regardless of toxicological endpoint.

[b] Risk characterization results as reported by King County (1999b).

[c] Totals include estimates from ingestion and dermal contact with both water and sediment. Estimates for water are not shown individually because they are several orders of magnitude lower than estimates shown for sediment exposure.

B.6 Uncertainty Analysis

There is a degree of uncertainty in any quantitative risk assessment. The exposure and toxicity assumptions used for this risk assessment, which were based on EPA guidance, current scientific literature, and best scientific judgment, are inherently uncertain. Therefore, the resulting risk estimates carry a degree of uncertainty. This section discusses some of the key uncertainties in this risk assessment, and presents recalculated risk estimates based on alternate exposure assumptions.

Table B-33 lists some of the key uncertainties in the Phase 1 HHRA. Each uncertainty is characterized qualitatively as low, medium, or high (see footnote to table for explanation of descriptors). Table B-33 also characterizes each uncertainty by the impact on risk characterization of additional data collection or an alternate analysis, the feasibility of collecting additional data or conducting additional analyses, and whether risk estimates included in the risk characterization section are likely to be underestimates or overestimates. Some of these uncertainties may be addressed during the data gap analysis to be conducted after completion of the Phase 1 HHRA. Additional data may be collected during the Phase 2 RI to reduce some of the uncertainties identified below.

The uncertainties described below are grouped by the risk assessment stage at which they apply: exposure assessment, toxicity assessment, and risk characterization. Each uncertainty listed in Table B-33 is also described in greater detail in Sections B.6.1 (uncertainty in exposure assessment), B.6.2 (uncertainty in toxicity assessment), and B.6.3 (uncertainty in risk characterization).

B.6.1 Uncertainty in Exposure Assessment

For most risk assessments, including this one, assumptions made during the exposure assessment have the greatest impact on the risk estimates. Alternate values are possible for all the parameters described in Section B.3.4, each of which will have a linear effect on the resulting risk estimate. The values selected for exposure frequency and exposure duration have been the subject of considerable debate and analysis during preparation of this Phase 1 HHRA, and will not be discussed further in the uncertainty assessment. There are several other areas in the exposure assessment that warrant additional discussion, including background concentrations, exposure point concentrations, ingestion rates, fraction of dose obtained from site, representativeness of existing fish and shellfish data for all potentially exposed populations, and exposure area used for the beach play scenario. Each topic is discussed below in separate sections.

Table B-33. Summary of uncertainties identified in Phase 1 HHRA

Parameter	Level of Uncertainty	Effect of Uncertainty on Risk Estimate	Potential Means to Decrease Uncertainty	Potential Impact on Risk Estimates	Feasibility	Comment
Exposure Assessment						
Background chemical concentrations	Medium	Very slightly to greatly overestimated	Discount risk estimates for chemicals with concentrations not different from background	High	High	Risk estimates do not account for contribution from natural background or from sources outside the LDW, which are likely to be as great or greater for some chemicals such as arsenic and dioxins/furans
Detection limits for all EPCs in sediments	Low	Accuracy unknown for chemicals that were never detected	Collect more sediment data with lower detection limits	Low	High	One-half detection limit used in calculations
EPCs for fish and shellfish	High	Accuracy unknown, may over- or underestimate risks	Collect additional data	Unknown	High	Based on small number of samples
Identical tissue COPCs for each market basket component	Medium	Greatly overestimated for some chemicals	Conduct COPC screening separately for each market basket fraction	High	High	Some COPCs (e.g., PAHs) accumulate differently in fish compared to shellfish, so identifying identical COPCs for each market basket component may not be appropriate
EPCs for perch	High	Underestimated because most chemicals not analyzed in perch	Collect additional data	Medium	High	Only three chemicals analyzed in perch
EPCs for mussels	Low	Statistical methods make no difference to overall risk estimates	Collect additional mussel chemistry data	Low	High	Additional work on the presence of harvestable LDW mussel populations will be conducted. If harvestable populations are present, the need for additional mussel chemistry data will be evaluated.
EPCs for PCBs in sediment derived from different analytical methods	Medium	Excluding NOAA HPLC/PDA data slightly increases risk estimate	Exclude NOAA data	Low	High	Existing non-NOAA PCB data suggest risks from direct exposure to sediment-associated PCBs are insignificant

Parameter	Level of Uncertainty	Effect of Uncertainty on Risk Estimate	Potential Means to Decrease Uncertainty	Potential Impact on Risk Estimates	Feasibility	Comment
Seafood ingestion rates	High	Greatly overestimated for tribal populations for current conditions. The degree of overestimation for tribal populations under future conditions is uncertain, but likely lower than current conditions. API community members harvest fish from the LDW, but it is uncertain to what degree consumption rates from EPA's 1999 API study overestimate LDW-specific API consumption rates.	Collect additional data that reflects habitat suitability to support harvestable fish and shellfish populations	High	Low	Current site usage may not reflect future site usage
Clam consumption not included in market basket approach	Medium	If harvestable populations of clams are present, chemical concentrations in those clams are similar to concentrations in non-anadromous LDW fish, and the estimated clam consumption rate is similar to upper-end rates reported in the Suquamish (2000) study, then the current risk estimate is greatly underestimated	Collect additional data on clam abundance and chemistry	Medium	Medium	Existing data suggest suitable clam habitat is rare in LDW, but additional data collection on topic is necessary

Parameter	Level of Uncertainty	Effect of Uncertainty on Risk Estimate	Potential Means to Decrease Uncertainty	Potential Impact on Risk Estimates	Feasibility	Comment
Fraction of intake obtained from site	High	For most individuals, the fraction of fish and shellfish intake obtained from the site is likely to be moderately to greatly overestimated. There may be a small population that currently practices subsistence seafood harvest from the LDW. The representativeness for future use scenario is unknown. For the beach play scenario, the fraction of intake from the site is unknown.	Collect additional data that reflects site-specific usage and habitat suitability to support beach play and harvestable populations of fish and shellfish	High	Medium	Default assumption of 1 due to lack of site-specific data
Representativeness of existing tissue chemistry data for all potentially exposed populations	Medium to High	Underestimated for some consumers (e.g., those who consume crab hepatopancreas and perch)	Collect additional data for different tissue types and/or use alternate exposure assumptions for different populations	Unknown	Medium	Existing data indicate that filets are the primary parts of the fish consumed. However, API community members, particularly within the Hmong community, consume other fish parts, including heads, bones, eggs, and organs. Use of filet data in risk estimates will underestimate risks for people who consume other parts of the fish with higher concentrations of COPCs.
Exposure area used for beach play scenario	Medium	Accuracy unknown, high uncertainty	Collect additional data on site usage and habitat suitability to support beach play	Unknown	Medium	Relationship between areas where intertidal chemistry data exist and human use occurs is uncertain
Spatial coverage of sediment chemistry data	Low	Low	Research past industrial activities to determine if likely chemical sources have been adequately characterized	Unknown	Medium	Available information does not suggest there are large sources that have not been characterized, but some gaps in spatial coverage may exist
Toxicity Assessment						
Chemicals without toxicity benchmarks	Low	Underestimated to unknown degree	Develop additional toxicity benchmarks	Medium	Low	Risk estimates not made for these chemicals

Parameter	Level of Uncertainty	Effect of Uncertainty on Risk Estimate	Potential Means to Decrease Uncertainty	Potential Impact on Risk Estimates	Feasibility	Comment
Tissue chemistry data for dioxins/ furans and PCB congeners	Medium	Moderately underestimated	Collect additional data	High	Medium	No data are available for these chemicals, which are highly toxic and may be found in fish tissue
Cancer slope factor for PCBs	Medium	Moderately overestimated	Additional congener data unlikely to change approach for tissue exposure, but different slope factor may be applicable for sediment ingestion if more highly chlorinated congeners are uncommon	High	Medium	Most health protective slope factor probably not applicable for all PCB congeners
Arsenic speciation	Medium	Moderately overestimated	Collect additional data on arsenic species present in tissue	Medium	Medium	10% value for inorganic arsenic as required by EPA may overestimate exposure, but it accounts for the uncertainty in the toxicity of dimethyl arsenic acid
Chromium speciation	Medium	Moderately overestimated	Collect additional data on chromium species present in sediment and tissue	Low	Medium	RfD for hexavalent chromium used for total chromium; chromium not identified as COC
Risk Characterization						
Risk estimates for chemicals that were never detected	High	Greatly overestimated if these COPCs are not present, uncertain if these COPCs are present	Collect additional data with lower detection limits	Unknown	Low	Many of the chemicals that were never detected have no known LDW source, so lower detection limits may not be helpful. Additional research on past industrial practices will be conducted to determine if uncharacterized sources may exist.

Level of uncertainty key: **low** = large and relevant dataset
medium = small dataset or limited information
high = very limited data or no site-specific information

Potential impact key: **low** = additional data or analysis unlikely to result in a change in determination of COCs or pathway of concern (i.e., HQ greater than 1 or cumulative cancer risk greater than 1E-6)
medium = additional data or analysis could result in a change in determination of COCs or pathway of concern
high = additional data or analysis likely to result in a change in determination of COCs or pathway of concern

Feasibility key: **low** = high budget or difficult research study would be required to fill data gap
medium = data gap could be filled with a mid-level field sampling event or research study or a detailed assessment of literature
high = data gap could be filled with additional literature search or through limited field sampling

B.6.1.1 Background concentrations

Arsenic was one of the COCs identified in the risk characterization. Cancer risks for this chemical exceeded 1E-6 for sediment and exceeded 1E-4 for seafood consumption scenarios. The EPCs that led to these risk estimates, however, are very similar to background concentrations in Puget Sound (Table B-34) and from sources nationally. Much of the arsenic identified is naturally occurring in sediments and in tissues due to arsenic from the earth's crust. In addition, historic sources within the region may have contributed additional arsenic in some areas. For example, Ecology (2002) recently conducted a soil survey for arsenic and lead in south King County that suggested the former Asarco smelter located in Ruston, Washington is likely one of the sources responsible for elevated arsenic and lead concentrations throughout the LDW watershed. Although it is not possible to determine with certainty to what extent arsenic concentrations result from background sources or from the smelter, as shown in Figure B-4 (taken from Ecology 2002), maximum arsenic concentrations in the soil on both sides of the LDW are higher than any of the sediment arsenic EPCs shown in Table B-34. Additional analysis of the degree to which arsenic in the LDW can be distinguished from background or regional sources will have a high impact on the designation of arsenic as a COC in both sediment and tissue. Additional research on this topic will be conducted in the Phase 2 RI.

Other COPCs identified in sediment and tissue are also likely to have sources outside the LDW. For example, dioxins and furans are known to have many sources, including municipal waste incineration, that are outside the LDW. Dioxins and furans have been measured in only 29 LDW sediment samples, so a complete characterization of the nature and extent of contamination from this group of chemicals is not possible. A Puget Sound-wide investigation of background concentrations for dioxins and furans has not been conducted. If additional LDW data for dioxins and furans are collected during Phase 2, additional analysis will also be conducted to determine appropriate background concentrations for this ubiquitous group of chemicals.

Table B-34. Arsenic EPCs and background concentrations

EPC Location and Medium	Statistic	Conc. (mg/kg) [a]	Background Location and Medium	Statistic	Conc. (mg/kg)[a]	Reference
Sediment						
LDW intertidal and subtidal sediments	SWA	12	Puget Sound reference area sediments	Proposed reference area performance standard	22	PTI (1991)
LDW intertidal sediments (Kellogg Island)	SWA	11	Central Puget Sound, non-urban sediments	Mean	5.3	Ecology (2000)
LDW intertidal sediments (southeast)	SWA	11	Central Puget Sound, non-urban sediments	Maximum	10.4	Ecology (2000)
LDW intertidal sediments (southwest)	SWA	9.4	Puget Sound region soil	90th percentile	7.3	Ecology (1994)
			Puget Sound region soil	Maximum	17.2	Ecology (1994)
Fish						
LDW English sole	Mean	10.9	Puget Sound English sole from non-urban areas	Mean	7.7	West et al. (2001)
LDW English sole	Maximum	15.1	Puget Sound English sole from non-urban areas	Maximum	20	West et al. (2001)
Shellfish						
LDW crabs	Maximum	12.5	NMFS nationwide survey of shellfish: crab	Range of means	3-4 blue crab; 5-6 Dunge-ness crab	FDA (1993a)
LDW mussels	95% UCL on mean	0.877	NMFS nationwide survey of shellfish: molluscan bivalves	Range of means	2.8 to 3.8	FDA (1993a)

[a] Concentrations in dry weight units for soil and sediment; wet weight units for tissue

SWA = spatially-weighted average concentration

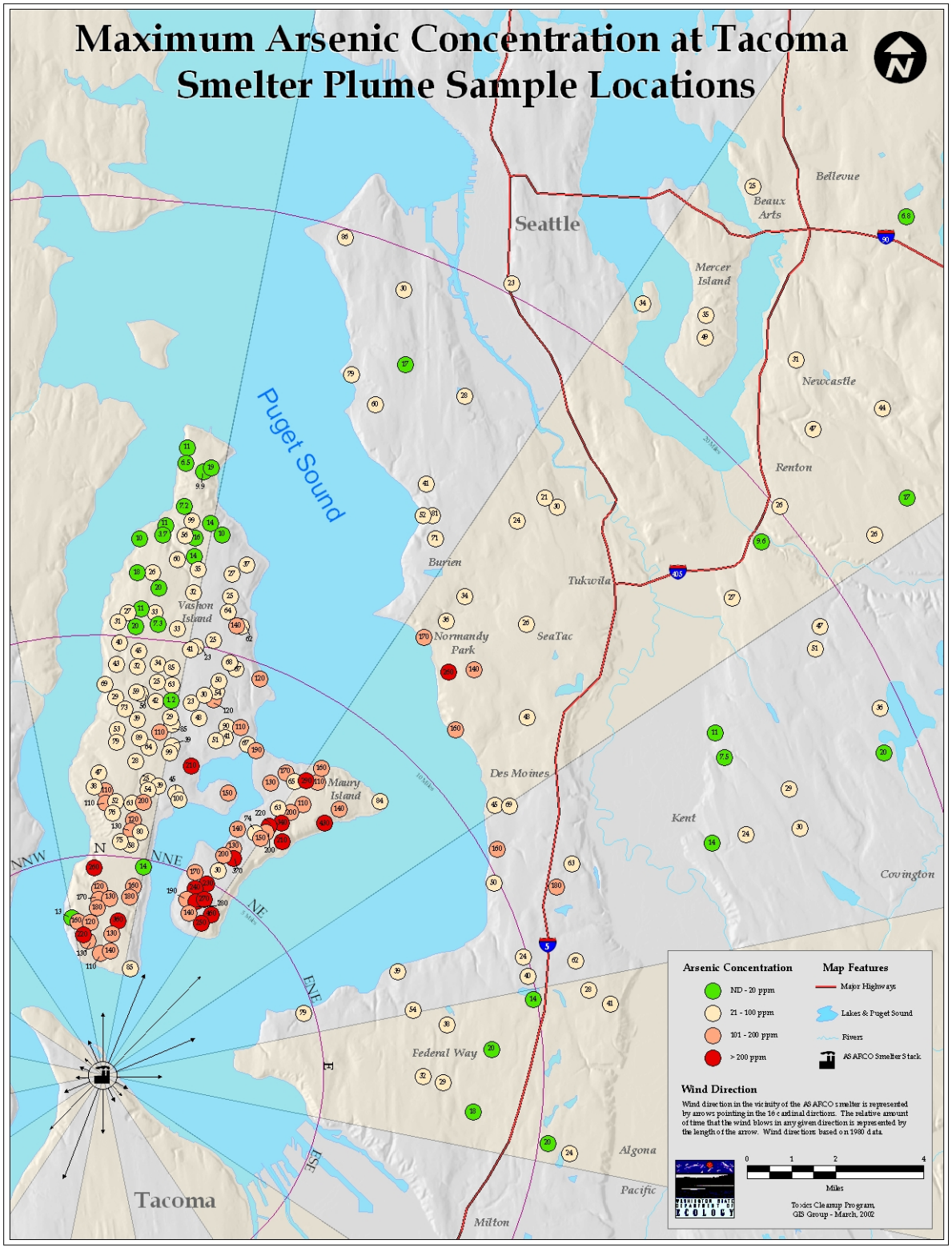


Figure B-4. Maximum arsenic concentrations at Tacoma smelter plume locations

B.6.1.2 Exposure point concentrations

EPCs were calculated for each COPC assuming one-half detection limit for non-detects. This assumption represents a compromise between assuming the chemical is not present at all (i.e., zero concentration for non-detect) and assuming the chemical is present at a concentration equal to the sample-specific detection limit. Application of this rule is not necessarily appropriate for all COPCs. For COPCs that were detected in the majority of the samples (e.g., PCBs), there is reason to believe that the "true" concentration for samples reported as non-detect is actually above zero. However, for chemicals which were rarely or never detected, the assumption of one-half the detection limit for non-detects may greatly overestimate the "true" concentration, especially for COPCs which have no known source within or in the vicinity of the LDW. Alternate assumptions are possible for COPCs that were never detected, and have no known source, including using a value of zero for these chemicals. Since these chemicals are not identified as COCs in the risk characterization (i.e., all risk estimates for these COPCs are presented below in Section B.6.3), this alternate assumption would have little impact on the overall risk conclusions in this HHRA. In Phase 2, chemicals that were rarely or never detected will be further evaluated to determine whether there is any reason to suspect they would be present (i.e., any possible sources). If no sources are found, these chemicals may not need to be further considered. Conversely, concentrations of chemicals with a confined point source might be elevated in a limited reach of the river and it might not be appropriate to average concentrations over the entire LDW.

EPCs were calculated for each exposure scenario using a different data set. The EPCs used in the seafood consumption scenario were based on the limited tissue chemistry data available. Sediment EPCs were based on a much larger data set. Due to the limited availability of existing data, single species served as surrogates for each seafood category used in the market basket approach.[21] The populations evaluated in this HHRA consume multiple species from each category. Therefore, estimated exposures using chemical concentrations of surrogate species may overestimate or underestimate chemical intake from each seafood category. Most of the tissue EPCs were based on the maximum detected concentration rather than the 95% UCL because of the high variability between samples. The implications of this high variability on the resulting risk characterization are difficult to determine because the impact of additional data points on the EPC cannot be predicted. However, additional data points should tend to increase knowledge of the mean, thus reducing the overall uncertainty of the estimate. Additional tissue chemistry data will be collected as part of the Phase 2 RI.

COPCs for the seafood consumption scenario were identified using the combined data for all four market basket fractions (i.e., English sole, perch, crab, and mussels). Some

[21] In the market basket approach, consumption rates and EPCs are derived independently for each diet component.

chemicals may accumulate differently in fish compared to shellfish. For example, cPAHs, which were detected in mussels, were not detected in any other LDW species. Mussels are filter feeders that derive their food through the water column and the other species are more likely to accumulate chemicals through sediment-associated prey. Consequently, it may not be appropriate to identify cPAHs as COPCs for species other than mussels. Exclusion of cPAHs as a COPC for English sole and crab (the chemicals were never detected in these two species and never measured in perch) would reduce the overall cancer risk estimate for cPAHs in the adult tribal RME seafood consumption scenario from 1E-4 to 2E-5.

For the seafood consumption scenarios, perch represent all pelagic fish, which is one of four components of the market basket approach. Available data for perch are very limited. Only 3 samples have been collected and these samples were analyzed only for PCBs, mercury, and TBT. None of the other tissue COPCs were analyzed in perch. Consequently, the overall risk estimates for the seafood consumption scenarios are underestimated because they are missing EPCs for one market basket component. Additional perch data will be collected for the Phase 2 RI. These additional data could result in identification of additional COCs for the seafood consumption scenarios.

The EPCs presented in Table B-14d for mussels are based on all 22 samples, with disregard to location and season collected. Table B-35 presents maximum concentrations for three carcinogenic COPCs based on location and season. The maximum concentrations for each location / season combination are generally within 50% of the EPCs that are based on the full data set, as used in the HHRA (Table B-14d). Alternate excess cancer risk estimates for the tribal RME seafood consumption scenario were made using the maxima shown in Table B-35 for three carcinogenic COPCs.[22] The excess cancer risk attributed to mussels varied from 3E-5 to 5E-5 depending on the maximum value used for arsenic, cPAHs, and PCBs. However, the total cancer risk estimates for these three COPCs using the maxima are identical to the risk estimate reported in Section B.5.3.2 (2E-3) regardless of which alternate EPC is used from Table B-35. This comparison suggests that the statistical treatment of mussel data does not have a major influence on the overall risk estimates for the seafood consumption pathways.

[22] The risks attributed to the fourth carcinogenic COPC, BEHP, are three orders of magnitude less than the three COPCs shown in Table B-34; hence, data for BEHP are not shown

Table B-35. Maximum concentrations (mg/kg ww) of three carcinogenic COPCs in LDW mussels from various locations and seasons and associated excess cancer risk for tribal RME seafood consumption scenario

COPC	Brandon Street		Duwamish/Diagonal		Kellogg Island	Slip 4	Terminal 107	EPC from Table B-14d
	dry	wet	dry	wet	wet	dry	dry	
Arsenic [a]	0.11	0.063	0.088	0.081	0.078	0.092	0.098	0.0867
cPAHs	0.022	0.046	0.022	0.048	0.045	0.022	0.022	0.039
PCBs (total-calc'd)	0.030	0.047	0.016	0.056	0.033	0.060	0.0065	0.0397
Mussel excess cancer risk	3E-5	4E-5	3E-5	5E-5	4E-5	3E-5	3E-5	4E-5
Total excess cancer risk	2E-3	2E-3	2E-3	2E-3	2E-3	2E-3	2E-3	2E-3

Arsenic concentrations were multiplied by 0.1 (the fraction of total arsenic assumed to be inorganic arsenic)

Dry and wet, as defined by King County (1999b), refer not necessarily to the time of year, but whether a large rain storm occurred prior to sample collection

Although over 120 chemicals have been analyzed in LDW tissue samples (see Table 3 in Subappendix B.2), some chemicals known to be toxic to humans have not been analyzed in fish or shellfish. For example, site-specific data sufficient for use in this HHRA have not been collected for PCB congeners or for dioxins and furans. Because PCBs are already an important COC for the seafood consumption scenario, additional PCB congener data would not impact the designation of this chemical as a COC. As indicated previously, if a determination is made to gather additional data for dioxins and furans in the LDW and in background locations, application of these data will likely increase risk estimates.

As indicated in Section B.2.3.5.1, the PCB sediment data used in this HHRA are from two different analytical methods: GC/ECD, which was used at approximately 600 locations, and HPLC/PDA, which was used by NOAA at approximately 300 locations. Some uncertainty exists on the suitability of the PCB data derived from the HPLC/PDA method. Alternate risk estimates were made using only PCB sediment data for the standard EPA (GC/ECD) method. Table B-36 presents summary statistics for total PCBs and the associated risk estimates for the RME netfishing scenario. Without the HPLC/PDA data, the arithmetic mean, spatially weighted mean, and 95% UCL on the spatially weighted mean are all approximately 30% higher than the corresponding statistics using the combined data set. Accordingly, the total cancer risk estimate (ingestion plus dermal) for total PCBs in the RME netfishing scenario is also slightly higher (5E-7 vs. 4E-7) using only the GC/ECD data. The suitability of continuing to use the HPLC/PDA data for the Phase 2 RI will be determined in consultation with EPA and Ecology.

Table B-36. Summary statistics (mg/kg dw) and associated cancer risk estimates for total PCBs in the RME netfishing scenario with and without the HPLC/PDA data for sediments

Statistic	Combined GC/ECD and HPLC/PDA Data	GC/ECD Data Only
Number of locations	956	651
Arithmetic mean	1.08	1.38
Spatially weighted mean	0.36	0.46
95% UCL on spatially weighted mean	0.49	0.66
Cancer risk estimate attributed to total PCBs in RME netfishing scenario	4E-7	5E-7

B.6.1.3 Ingestion rates

Site-specific estimates of ingestion rates were not available for any of the three exposure scenarios (i.e., netfishing, beach play, and seafood consumption). The incidental sediment ingestion rates used for the netfishing and beach play scenarios are commonly used in human health risk assessments, but it is not certain how applicable they are to the LDW. For example, the amount of sediment transferred to fishermen's hands when handling monofilament gill nets is not known.

The seafood consumption rates used in the adult and child RME scenarios were derived from the Suquamish (2000) and API (EPA 1999c) seafood consumption surveys. These surveys appear to fairly represent the populations that were interviewed, but the degree to which they might represent people who do or may in the future consume fish and shellfish from the LDW is not known.

The ingestion rates from the Suquamish (2000) study are derived from a much larger fishing area compared to the LDW. It is uncertain whether the ingestion rates from that study are sustainable in a system the size of the LDW over the time period evaluated in this HHRA (55 years). Additional research related to species habitat preferences and biological carrying capacity of the LDW will be conducted prior to completion of the baseline HHRA (Phase 2 RI). The additional research could have a high impact on the resulting risk conclusions, although collection of sufficient data to greatly reduce the uncertainty could be difficult (i.e., low feasibility).

The exposure assessment did not include a consumption rate for clams, which make up an important part of the fish and shellfish diet of the Suquamish tribe. The likelihood that the LDW can support harvestable clam populations will be determined prior to the completion of the Phase 2 HHRA. If existing habitat is sufficient to support a harvestable clam population, clam consumption may be included in the Phase 2 HHRA. These additional data could result in additional COPCs being identified, and consequently could result in an increased estimate of risk.

The cancer risk estimates provided in Section B.5.3.2 are based on 90th (API) or 95th (Suquamish) percentile consumption rates (Table B-12). The risk estimates derived from the Suquamish data did not include the consumption rate for all shellfish species

because of uncertainties about the sustainability of clam harvest within the LDW. Alternate risk estimates that do include the total shellfish group are provided in Table B-37. Two risk estimates are provided for each scenario – one using the EPCs for crabs[23] and one using the EPCs for mussels.[24] The EPCs are very different between the two species, particularly for arsenic and PCBs, reflecting potential differences in uptake/elimination kinetics, metabolism, life histories, and exposure routes. No site-specific tissue chemistry data are available for clams, which comprise the largest fraction of the total shellfish consumption rates from Suquamish (2000). Clam tissue chemistry data may be collected during the Phase 2 RI, pending additional analysis on harvest sustainability within the LDW. The total cancer risks for the alternate scenarios range from 1E-2 for the 95th percentile consumption rates using the crab EPCs to 3E-4 for the 50th percentile consumption rates using the mussel EPCs (Table B-37). As shown, inclusion of shellfish consumption would increase risk estimates.

Table B-37. Cancer risk estimates for alternate seafood consumption rates from the Suquamish (2000) survey

CONSUMPTION RATES USED	TOTAL CANCER RISK ESTIMATE FROM DETECTED COPCS	
95th percentile pelagic, benthic, crabs, mussels (as presented in Table B-29a)	2E-3	
	Based on crab EPCs	Based on mussel EPCs
95th percentile pelagic, benthic, total shellfish group	1E-2	3E-3
90th percentile pelagic, benthic, total shellfish group	8E-3	2E-3
75th percentile pelagic, benthic, total shellfish group	3E-3	6E-4
50th percentile pelagic, benthic, total shellfish group	1E-3	3E-4

B.6.1.4 Fraction of dose obtained from site

For the Phase 1 HHRA, the fractional intake of dose obtained from the site (FI) was set at one by default for all exposure pathways. This assumption is appropriate for the netfishing scenario, which occurs primarily within the LDW. For the beach play and seafood consumption scenarios, however, there is more uncertainty in selection of an appropriate FI value.

There are a number of factors to consider in selecting an FI for the seafood consumption scenario. The risk assessment utilized the fraction of fish caught from Puget Sound by Suquamish Tribal members, and assumed that all of the catch from Puget Sound could be obtained from the Lower Duwamish Waterway (i.e., FI = 1). Extrapolation from the Suquamish survey to characterize Muckleshoot Tribal consumption imparts considerable uncertainty. The type and quality of resources

[23] From Table B-14c, the relevant crab EPCs, in mg/kg ww, for cancer risk are 1.25 for arsenic, 0.0080 for BEHP, 0.022 for cPAHs, and 0.177 for PCBs

[24] From Table B-14d, the relevant mussel EPCs, in mg/kg ww, for cancer risk are 0.0864 for arsenic, 0.017 for BEHP, 0.0300 for cPAHs, and 0.028 for PCBs

available to each tribe are quite different. In general, Tribes draw most of their self-caught fish from their Usual and Accustomed (U&A) areas. The Muckleshoot Tribe has more freshwater resources in its U&A than does the Suquamish Tribe, and the balance of freshwater versus marine consumption is unknown. Consumption of self-caught freshwater species would reduce the FI. The mouth of the LDW is the only marine habitat accessible to the Muckleshoot Tribe. If the Muckleshoot Tribe preferred marine species, this would lead to use of a higher FI. The mouth of the LDW is the only source for marine shellfish, consequently an FI of one would be appropriate for shellfish consumption. Another confounding factor in selecting an FI value is consideration of future resource quality. Resource use and consequently FI could increase after removal of chemical contamination. However, the habitat quality may not permit sustainable high seafood consumption rates, leading to a lower FI. Habitat quality will be considered in the Phase 2 HHRA.

The exposure of some individuals represented by the RME scenarios may be adequately represented by an FI of one. A value of one was selected for the seafood consumption scenario because site-specific data are insufficient for deriving a specific quantitative estimate of FI that is applicable to all potentially exposed individuals. This value likely overestimates current exposures, but does allow for considerable future expansion of recreational and subsistence use of the LDW.

For the beach play scenario, exposure frequency determines how often children may encounter LDW sediments. On days when children are present at LDW intertidal areas, the fraction of total soil/sediment intake consisting of LDW sediment (e.g., the FI) is unclear. It seems reasonable that on days when children are present at LDW intertidal areas, the majority of their soil intake would consist of LDW sediments. Consequently, the HHRA uses a health protective FI value of one for the beach play scenario.

Because the use of a fractional intake of one may overestimate risks for many site users, and to offer different perspectives for risk management decisions to be made at the site, order-of-magnitude variations (i.e., 0.1 and 0.01) of the default FI value were evaluated for the RME beach play and seafood consumption scenarios (Table B-38). Even at a FI of 0.01, the combined cancer risk estimate for all chemicals in the adult and child tribal RME seafood consumption scenario still identifies seafood consumption as a pathway of concern (i.e., cancer risk greater than 1E-6).

Table B-38. Cancer risk estimates for beach play and seafood consumption scenarios using alternate assumptions for fractional intake of dose obtained from site

SCENARIO	FI = 1	FI = 0.1	FI = 0.01
Seafood consumption, adult tribal RME	2E-3	2E-4	2E-5
Seafood consumption, child tribal RME	5E-4	5E-5	5E-6
Seafood consumption, adult API RME	9E-5	9E-6	9E-7
Beach play, Kellogg Island	4E-6	4E-7	4E-8
Beach play, southeast	5E-6	5E-7	5E-8
Beach play southwest	4E-6	4E-7	4E-8

B.6.1.5 Representativeness of fish and shellfish COPC data for all potentially exposed populations

The tissue samples collected and analyzed in the studies summarized in this HHRA were uncooked portions of the total organism (i.e., filet for fish, muscle meat for crab). These portions represent the consumption habits of many, but not all, the potentially exposed populations. COPC concentrations in other tissues besides those evaluated in this HHRA may be different.

For example, most people cook fish or shellfish before eating them. Data from uncooked or raw samples were used in this HHRA because most chemistry data were collected for this type of sample. There is no standard cooking preparation that is used for environmental investigations. The King County Water Quality Assessment (King County 1999b) included analysis of two composite samples of crab that had been cooked and two composite samples of crab that had not been cooked. Mean concentrations of arsenic and PCBs, which are two COCs identified in the risk characterization section, were 9.95 mg/kg and 156 µg/kg, respectively, in the uncooked samples, and 4.84 mg/kg and 89.5 µg/kg, respectively, in the cooked samples. For these two chemicals, risk estimates associated with data from cooked samples would be lower than the risk estimates presented in Section B.5.3.2, which are based on data from uncooked samples.

Because there are no standard cooking practices, the assumption that risks would be uniformly reduced to the degree indicated by the King County data is inappropriate. For example, preparation of soups or stews from seafood would not likely reduce chemical concentrations to the same degree as broiling. Given the uncertainties in both chemical concentration reduction associated with different cooking practices, as well as the cooking practices employed by different groups, it is appropriate to use concentrations from uncooked tissue samples for risk assessment purposes.

Based on the API seafood consumption study (EPA 1999c), several API ethnic groups (e.g., Cambodian, Mien, Hmong, and Vietnamese) consume filets with skin more often than filets without skin. This HHRA used available data, which was primarily from filets without skin. Moreover, more than one-third of API survey respondents reported they "sometimes" eat head, bone, eggs, and/or organs. The API report (EPA

1999c) indicated a high consumption rate for crab, and also that more than half of the study respondents consume the internal organs, known as "crab butter." Some of these tissues (e.g., skin, eggs, organs) may contain more lipid than fish filet or crab meat samples. Given the tendency of PCBs and other organic chemicals to partition preferentially in lipid, the PCB EPCs used in this HHRA may underestimate exposure to API individuals.

A single composite crab hepatopancreas sample from the LDW measured by King County (1999b) during their Water Quality Assessment project had a total PCB concentration of 1.647 mg/kg wet weight, which is almost an order of magnitude higher than the crab EPC (0.177 mg/kg) given in Table B-14c. Arsenic was 6.98 mg/kg in this sample, which is less than the maximum arsenic concentration (12.5 mg/kg) detected in the whole-body crabs from the same study. Carcinogenic PAHs were not detected in this hepatopancreas sample. The API report (EPA 1999c) reported crab consumption rates and the percentage of time that individuals consumed the entire crab, including the hepatopancreas. From these data, a 95% UCL on the mean number of crab servings per day was calculated and shown in Table B-39. From King County (1999b) data, a hepatopancreas from a small crab weighed 36 g and the hepatopancreas from a large crab weighed 55 g. Using these weights, hepatopancreas consumption rates were derived for various API ethnic groups (Table B-39). The incremental risk associated with PCB exposure related to hepatopancreas consumption was derived through application of the hepatopancreas concentrations measured by King County and the hepatopancreas consumption rate derived from API data. Hepatopancreas consumption increased the total PCB cancer risk calculated for all API ethnic groups from 2E-5, to 3E-5 to 9E-5, depending on ethnic group. Data on consumption rates for specific ethnic groups were limited by small sample sizes in the API study and thus, these estimates may not be entirely representative of consumption rates for particular groups. Additional data may be collected for crab hepatopancreas during the Phase 2 RI.

Table B-39. Crab hepatopancreas consumption rates and associated excess cancer risks from PCBs by API ethnic group

Ethnic Group	95% UCL on Mean Crab servings/day [a]	Hepatopancreas consumption rate (g/day) [b]		Total Excess Cancer Risk From Exposure to PCBs in Fish and Shellfish [c]	
		Low	High	Low	High
Cambodian	0.026	0.95	1.4	4E-5	5E-5
Chinese	0.046	1.6	2.5	6E-5	8E-5
Filipino	0.012	0.44	0.68	3E-5	4E-5
Hmong	0.030	1.1	1.7	5E-5	6E-5
Japanese	0.021	0.76	1.2	4E-5	5E-5
Laotian	0.057	2.0	3.1	7E-5	9E-5
Mien	0.012	0.43	0.66	3E-5	4E-5
Samoan	0.024	0.85	1.3	4E-5	5E-5
Vietnamese	0.029	1.0	1.6	5E-5	6E-5

[a] Calculated from API data (EPA 1999c) by multiplying the number of servings of crab per year by the percentage of time that an individual consumes entire crab including hepatopancreas. 95% UCL calculated assuming the data are normally distributed using the formula: mean + (($t_{0.95}$ * standard deviation) / $n^{1/2}$)

[b] Low consumption rate calculated by multiplying 95% UCL on mean servings per day by 36 g/serving; high rate used 55 g/serving. Hepatopancreas weights from King County (1999b).

[c] Low and high cancer risk estimates correspond to low and high hepatopancreas consumption rates, respectively. Cancer risk estimate equals the total PCB cancer risk estimate for all species in the API RME seafood consumption scenario (2E-5, see Table B-28c) plus the ethnic group-specific PCB cancer risk attributable to the additional consumption of crab hepatopancreas

Chemical concentrations from whole-body fish samples were not used in the exposure assessment for the API RME scenario. Six whole-body fish samples, three for English sole and three for shiner perch, have been collected from the LDW. Concentrations of some chemicals, particularly PCBs, were higher in these whole-body samples compared to concentrations in the filet samples that were used in the exposure assessment. The mean PCB concentrations in the whole-body samples were 1.49 and 0.496 mg/kg ww, for English sole and shiner perch, respectively, compared to the mean PCB concentrations in filet samples of 0.285 and 0.228 mg/kg ww. PCB risks estimated using data from whole-body samples would be higher than the risk estimates presented in Section B.5. For example, PCB cancer risk estimates for the API RME scenario would increase from 2E-5 to 4E-5 if it was assumed that half the benthic and pelagic fish consumption was whole-body fish instead of filets.[25] Whole-body fish chemistry data will be incorporated into risk estimates for the Phase 2 HHRA.

The API seafood consumption rates given in Table B-12 are based on the percentage of the total fish and shellfish diet consisting of self-caught fish from King County. The degree to which these fractions (shown in the notes to Table B-12) represent the consumption practices of individuals who may consume LDW fish and shellfish is not known. These fractions represent the 95% UCL of the mean percentage of self-caught fish from King County. Many individuals reported zero for this variable during the interviews, while others reported very high percentages (EPA 1999c). Given the industrial nature of the LDW, some reviewers of the draft Phase 1 HHRA suggested that individuals with a lower income level might consume more self-caught fish and shellfish compared to individuals with higher income levels. Table B-40 presents data from EPA (1999c) that indicates that income level and percentage of self-caught fish and shellfish in the diet do not appear to be related. The individuals with the lowest income (i.e., below the federal poverty level) reported no greater percentage of self-caught fish and shellfish compared to individuals two-to-three times the federal poverty level.

[25] The revised risk estimate assumed modified EPCs half-way between the filet EPCs and the mean concentrations from the whole-body samples. For perch, the modified EPC was 0.362 mg/kg ww ([0.228 + 0.496]/2). For English sole, the modified EPC was 0.888 mg/kg ww ([0.285 + 1.49]/2)

Table B-40. Relationship of income level and percentage of self-caught fish and shellfish from API consumption survey

	Percentage of self-caught fish from King County in total diet			
	Under Federal Poverty Level (FPL)	1 – 2X FPL	2 – 3X FPL	> 3X FPL
Pelagic group	6.2	4.6	14	4.7
Benthic group	3.6	8.5	7.7	7.2
Shellfish group	20	17	20	9

B.6.1.6 Exposure area for beach play scenario

EPCs were calculated for the beach play scenario using three different intertidal areas. The intertidal portion of the LDW was divided into these areas because sampling density was patchy and certain areas were not represented well by existing data. For example, very few intertidal samples have been collected in the central portion of the LDW, so EPCs were not calculated for this region. The regions that were included in the EPC calculations were defined by data availability rather than the potential for human use. Thus the relevance of the beach play EPCs to the exposure scenario is uncertain. Additional research on human site use of intertidal areas will be conducted prior to completing the baseline HHRA (Phase 2 RI).

B.6.1.7 Spatial coverage of sediment chemistry data

As described in Section B.2.3, the existing historical sediment chemistry database is reasonably representative of both site-related contamination and human use patterns, with the exception of the spatial coverage in intertidal areas potentially used for beach play (see Section B.6.1.6). Although sampling coverage was generally thorough, the number of analyses conducted for each chemical differs (see Subappendix B.2). Many chemicals were analyzed in hundreds of samples, but some were analyzed much less frequently. For example, volatile organic compounds were analyzed in approximately 50 samples, chlorinated pesticides were analyzed in approximately 110 samples, and dioxins/furans were analyzed in 29 samples.

Volatile organic compounds and chlorinated pesticides were rarely detected in surface sediment samples, which suggests there are no localized sources of these chemicals in the areas sampled. Additional research on past industrial practices will be conducted during the Phase 2 RI to determine whether these chemicals are likely to be detected in areas that have not been sampled. This research may influence study design for the Phase 2 sediment sampling.

B.6.2 Uncertainty in Toxicity Assessment

B.6.2.1 Toxicity benchmarks

The toxicity benchmarks used in this HHRA are based on the most recent guidance provided by EPA. Approximately 20 chemicals (see Table 3 in Subappendix B.2) that have been measured in LDW tissue samples do not have toxicity benchmarks. The

toxicity associated with these chemicals is either unknown or not sufficiently characterized for EPA to derive toxicity values for use in risk assessment. Toxicity benchmarks could be developed for these chemicals by requesting a review from NCEA, as indicated in EPA guidance.

The toxicity benchmarks presented in Section B.4 are based on many different studies using both experimental animals and human populations. The RfDs published by EPA include consideration of available data for effects on children (based in some cases on developmental effects in animal studies), particularly the developing fetus. They are designed to be protective of sensitive sub-populations, including children and developing fetuses, but the inherent uncertainty may span an order of magnitude or greater. For example, the RfD for methylmercury and the evaluation of lead within the IEUBK model are based on developmental effects on children following exposure during gestation. EPA's RfD for methylmercury has been extensively peer-reviewed and is thought to be sufficiently health-protective for children (NRC 2000). The ATSDR (1999d) has calculated a maximum risk level (MRL) for methylmercury, using the same data used by EPA, that is three times higher than EPA's RfD, suggesting that the application of EPA's RfD provides an additional measure of health protectiveness.

Some chemicals may have developmental effects, but other endpoints were used by EPA to develop the RfDs. For example, several studies have documented developmental effects from exposure of pregnant women to PCBs through fish consumption (Fein et al. 1984; Jacobson and Jacobson 1996, 1997), but the RfD published in IRIS is based on an immunological endpoint because it was considered to be more health-protective than the developmental endpoint (i.e., to occur at a lower dose level). Similarly, arsenic may have some developmental effects at sufficient dose levels (ATSDR 2000a), but the critical study described in IRIS documenting dermal and cardiovascular effects was used to set the RfD because EPA considered these endpoints more health protective than the developmental endpoint.

B.6.2.2 PCBs

Although over 120 chemicals have been analyzed in LDW tissue samples (see Table 3 in Subappendix B.2), some chemicals known to be toxic to humans have not been analyzed in fish or shellfish. For example, site-specific tissue data sufficient for use in this HHRA have not been collected for PCB or dioxin/furan congeners. Since PCBs are already an important COC for the seafood consumption scenario, additional PCB congener data would not impact the designation of this chemical as a COC.

EPA (1996b) has recommended a tiered approach for establishing the most appropriate slope factor for assessing cancer risk from PCBs. The PCB cancer slope factor associated with high risk and persistence was used for the seafood consumption pathway. It was intended that this slope factor be applied to total PCBs rather than any specific Aroclor mixture (EPA 1996b). Additional PCB congener data may be collected during the Phase 2 RI. Application of different slope factors to PCBs, based on the congener distribution pattern, could have a high impact on the resulting risk

conclusions for the sediment ingestion pathway. Application of an alternate slope factor for the seafood consumption pathway is not likely to be realistic given the highly persistent nature of many of the PCB congeners that bioaccumulate in fish (Lake et al. 1995).

The PCB congeners that exhibit the highest mammalian toxicity resemble dioxin in structure and tend to be more readily bioaccumulated than less toxic congeners (EPA 1996b). Therefore, it is possible that an evaluation of total PCBs using a single PCB cancer slope factor may underestimate the risk from these dioxin-like congeners. Risk estimates for these PCB congeners, along with dioxin and furan congeners, can be made using the toxicity factor associated with 2,3,7,8-TCDD. This chemical, expressed as a TEQ, could be identified as a COC if additional data were collected. The high cost of PCB congener analysis will be a factor in the collection of additional data.

Cogliano (1998) reported the presence of the dioxin-like PCB congeners in Aroclors 1016, 1242, 1254, and 1260. Dioxin toxicity equivalence was much greater in Aroclor 1254 than in the other formulations. Approximately one-half the total PCBs concentrations used in this HHRA are attributable to Aroclor 1254. Therefore, cancer risks from dioxin-like PCB congeners may have been underestimated using the Aroclor-only approach.

B.6.2.3 Arsenic and chromium

The available arsenic data for fish and shellfish tissues is based on total arsenic. The cancer slope factor, however, is based on inorganic arsenic, not total arsenic. Most of the arsenic in fish and shellfish is present in the form of organic compounds rather than as inorganic arsenic (EPA 1997b). In accordance with pending EPA guidance on this topic, a draft correction factor of 0.1 (10%) was applied to all total arsenic data for fish and shellfish to approximate the proportion of inorganic arsenic in these samples. EPA[26] determined that the reasonable maximum value of 10% was appropriate given the wide range in percent inorganic arsenic among samples of a given species, the limited database on concentrations of inorganic arsenic in freshwater fish, the uncertainties in the toxicity and concentrations of dimethylarsinic acid (a probable human carcinogen) in fish, and the uncertainties in the analytical techniques used for speciating arsenic.

EPA's recommended guidance of 10% is heavily influenced by two studies of resident freshwater fish from the Columbia River Basin [Tetra Tech (1996) and EVS (2000)]. This guidance assumes that the fraction of inorganic arsenic in total arsenic is independent of the total arsenic concentration. Available data do not support this assumption. The total arsenic concentrations in the Columbia and Willamette River fish (carp, sucker, bass, sturgeon, salmon) were generally well below 1 mg/kg, in contrast to the total arsenic concentrations for Duwamish English sole, which are

[26] EPA's discussion of the default correction factor for inorganic arsenic was included in joint EPA/Ecology comments, dated June 26, 2001, on LDWG's interim LDW RI Phase 1 HHRA deliverable

approximately 10 mg/kg. Inorganic arsenic concentrations in the Columbia River Basin fish were all less than 0.05 mg/kg. Other recent literature surveys on this topic report maximum inorganic arsenic concentrations in marine fish of 0.16 mg/kg or less (Donohue and Abernathy 1999, Schoof et al. 1999b). The total arsenic concentrations in marine fish in the database from which the inorganic arsenic data were taken were as high as 65 mg/kg wet wt (Donohue and Abernathy 1999). Although there are no site-specific data on inorganic arsenic concentrations in Duwamish English sole, it appears that inorganic arsenic concentrations of 1 mg/kg or more (i.e., 10% of 10 mg/kg) are highly unlikely given the data compiled to date.

The results presented above suggest that marine fish, which tend to have higher body burdens of total arsenic compared to freshwater fish, have physiological mechanisms to regulate the amount of inorganic arsenic in their bodies. However, there are uncertainties associated with some of the literature supporting determination of the percent of inorganic arsenic in fish and shellfish including: 1) in some of the literature reviews, zero values were reported when inorganic arsenic was undetected, whereas detection limits should have been used (in such cases, the percent inorganic arsenic estimate would have been biased low); and 2) some summaries of tissue inorganic arsenic concentrations do not include analytical and sample handling considerations, which could add an unknown or low bias to percent inorganic arsenic estimates derived from studies with those limitations.

Additional data on the proportion of arsenic present in inorganic forms in fish and shellfish may be collected during the Phase 2 RI. Until these data are collected, the 10% correction factor is still appropriate given the uncertainties around the existing tissue arsenic database and the toxicity of dimethylarsinic acid. The impact of these additional data on risk conclusions is likely to be low, however, given the issues related to background concentrations discussed in Section B.6.1.1.

The available chromium data for both sediment and tissue are based on total chromium. The RfD used for chromium in this HHRA, is based on hexavalent chromium, however. This health protective assumption suggests that risks from chromium were likely overestimated. However, since this chemical was not identified as a COC (i.e., HQ was not greater than 1), the overall impact to the risk conclusions is low.

B.6.3 UNCERTAINTY IN RISK CHARACTERIZATION

As indicated in Section B.5.2, risks were characterized only for those chemicals that were detected in the medium specific to that exposure scenario (i.e., sediment or tissue). Several chemicals in each scenario were never detected, but the detection limits exceeded the applicable RBCs. To make health protective choices in the face of

uncertainty, these undetected chemicals were identified as COPCs (see Section B.3.3).[27] Hypothetical EPCs were calculated for these chemicals and are presented in Section B.3.4.3. The hypothetical EPCs correspond to half the highest detection limit for that chemical. Risks calculated using half detection limit values will overestimate risks if these COPCs are not present (or are present at concentrations lower than one-half the highest detection limit) and will underestimate risks if the COPCs are present at concentrations greater than one-half the detection limit. The degree of spatial coverage and assay detection limits are important factors to consider in determining whether the lack of detection truly indicates that a substance is not present. If these COPCs are present, then the effect of using half the detection limit in the risk analysis is uncertain, as the true concentration could be anywhere between zero and the detection limit. The Phase 2 RI will evaluate analytical methods that have detection limits lower than risk based concentrations.

Hypothetical risk estimates for the undetected COPCs are presented below. For the netfishing RME exposure scenario, benzidine and n-nitrosodimethylamine both had cancer risks greater than 1E-6 based on half detection limit EPCs (Table B-41). These chemicals would have been identified as COCs had these concentrations been associated with detections. The hypothetical cancer risks from benzidine alone were greater than the cancer risks from all other sediment COPCs combined. Hypothetical HQs from the undetected sediment COPCs were very low for this scenario (Table B-42). For the beach play scenario, the hypothetical cancer risk attributed to undetected COPCs was generally only a small fraction (1% or less) of the total risk from all COPCs.

Eleven of the sixteen carcinogenic tissue COPCs that were undetected had hypothetical cancer risks higher than 1E-6 for the adult tribal RME seafood consumption scenario (Table B-41). The highest hypothetical cancer risk estimates were for benzidine (5E-2) and n-nitrosodimethylamine (2E-3). The total hypothetical cancer risk estimates for these 16 COPCs were approximately 30x higher than the cancer risk estimates for detected tissue COPCs. None of the hypothetical non-cancer HQs for these ten COPCs with non-carcinogenic endpoints were greater than 0.1 (Table B-42).

As part of the data gaps analysis, additional research will be conducted on whether any of the undetected COPCs are likely to be present in LDW sediment or tissue samples. If such likelihood exists, additional sampling for these chemicals may be conducted as part of the Phase 2 RI. For example, benzidine was used as an intermediate in the production of azo dyes, sulfur dyes, fast color salts, naphthols, and other dyeing compounds. N-nitrosodimethylamine can be released from the manufacture of pesticides, rubber tires, alkylamines, and dyes, and also may form under natural conditions in air, water and soil as a result of chemical, photochemical,

[27] 19 of 31 tissue COPCs were undetected, 3 of 17 sediment COPCs for the netfishing scenario were undetected, 7 of 29 sediment COPCs for the beach play scenario were undetected

and biological processes. There are no known users or sources of either chemical present in the LDW. Other chemicals, such as pesticides, are widely present in the environment, but are not known to be related to LDW sources.

Table B-41. Hypothetical cancer risk estimates for undetected COPCs

COPC	Netfishing [a]		Beach play [a]			Seafood consumption				Adult tribal + Net CT
	RME	CT	Kellogg I.	Southeast	Southwest	Adult tribal	Child tribal	Adult API	Adult recreational	
1,2-Diphenylhydrazine	n/a	n/a	n/a	n/a	n/a	1E-5	4E-6	7E-7	2E-7	
3,3'-Dichlorobenzidine	n/a	n/a	n/a	n/a	n/a	4E-6	1E-6	2E-7	7E-8	
Aldrin	n/a	n/a	n/a	n/a	n/a	1E-6	3E-8	3E-7	8E-9	
alpha-BHC	n/a	n/a	n/a	n/a	n/a	6E-7	1E-8	1E-7	3E-9	
Benzidine	5E-5	2E-5	n/a	n/a	n/a	5E-2	2E-2	3E-3	9E-4	5E-2
beta-BHC	n/a	n/a	n/a	n/a	n/a	2E-7	3E-9	3E-8	8E-10	
bis(2-Chloroethyl)ether	n/a	n/a	4E-9	1E-8	4E-9	6E-6	2E-6	3E-7	1E-7	
Chlordane	n/a	n/a	n/a	n/a	n/a	9E-8	9E-10	3E-8	9E-8	
Dieldrin	n/a	n/a	3E-9	n/a	3E-9	2E-6	5E-8	3E-7	1E-8	
Heptachlor	n/a	n/a	n/a	n/a	n/a	4E-7	8E-9	9E-8	2E-9	
Hexachlorobenzene	n/a	n/a	3E-9	n/a	n/a	9E-6	3E-6	5E-7	2E-7	
Hexachlorobutadiene	n/a	n/a	n/a	n/a	n/a	7E-7	2E-7	4E-8	1E-8	
N-Nitrosodimethylamine	2E-6	6E-7	n/a	9E-7	n/a	2E-3	6E-4	1E-4	3E-5	2E-3
N-Nitroso-di-n-propylamine	n/a	n/a	3E-8	8E-8	3E-8	6E-5	2E-5	3E-6	1E-6	
Pentachlorophenol	n/a	n/a	n/a	n/a	n/a	1E-6	3E-7	6E-8	2E-8	
Toxaphene	n/a	n/a	n/a	n/a	n/a	1E-6	3E-8	2E-7	1E-8	
Total risks from non-detected COPCs	**5E-5**	**2E-5**	**4E-8**	**1E-6**	**3E-8**	**5E-2**	**2E-2**	**3E-3**	**9E-4**	**5E-2**
Total risks from non-detected and detected COPCs	**5E-5**	**2E-5**	**5E-6**	**4E-5**	**5E-6**	**5E-2**	**2E-2**	**3E-3**	**9E-4**	**5E-2**

[a] Risk estimates and hazard quotients for sediment exposure scenarios (netfishing and beach play) combine both ingestion and dermal exposure routes

n/a = not applicable

Table B-42. Hypothetical hazard quotient estimates for undetected COPCs

COPC	Netfishing [a]		Beach play [a]			Seafood consumption				Adult tribal + Net CT
	RME	CT	Kellogg I.	Southeast	Southwest	Adult tribal	Child tribal	Adult API	Adult recreational	
1,2,3-Trichloropropane	0.000002	0.0000005	0.000003	0.0000006	0.0000005	n/a	n/a	n/a	n/a	0.0000005
2-Nitroaniline	n/a	n/a	0.002	0.005	0.002	n/a	n/a	n/a	n/a	n/a
Aldrin	n/a	n/a	n/a	n/a	n/a	0.004	0.0007	0.001	0.00004	0.004
Benzidine	0.0001	0.00006	n/a	n/a	n/a	0.09	0.26	0.009	0.003	0.09
bis(2-Chloroisopropyl)ether	n/a	n/a	n/a	n/a	n/a	0.0006	0.002	0.00005	0.00002	0.0006
Chlordane	n/a	n/a	n/a	n/a	n/a	0.0007	0.00006	0.0004	0.001	0.0007
DDTs (total-calc'd)	n/a	n/a	n/a	n/a	0.00001					
Dieldrin	n/a	n/a	0.00004	n/a	0.00004	0.003	0.0007	0.001	0.00004	0.003
gamma-BHC	n/a	n/a	n/a	n/a	n/a	0.0004	0.00007	0.0002	0.000004	0.0004
Heptachlor epoxide	n/a	n/a	n/a	0.004	0.001	0.0002	0.00004	0.00009	0.000002	0.0002
Heptachlor	n/a	n/a	n/a	n/a	n/a	0.009	0.002	0.003	0.00008	0.009
Hexachlorobenzene	n/a	n/a	0.00002	n/a	n/a	0.009	0.02	0.0009	0.0003	0.009
Pentachlorophenol	n/a	n/a	n/a	n/a	n/a	0.0004	0.001	0.00004	0.00001	0.0004
Hazard index for non-detected COPCs	**0.0001**	**0.00006**	**0.002**	**0.01**	**0.002**	**0.12**	**0.29**	**0.02**	**0.005**	**0.12**
Hazard index for non-detected and detected COPCs	**0.07**	**0.04**	**0.36**	**0.71**	**0.41**	**15**	**40**	**1.8**	**0.68**	**15**

[a] Risk estimates and hazard quotients for sediment exposure scenarios (netfishing and beach play) combine both ingestion and dermal exposure routes

n/a = not applicable

B.7 Conclusions

The existence of historical sediment and tissue chemistry data for the LDW made it possible to make preliminary risk estimates for three exposure scenarios,[28] seafood consumption, beach play, and commercial netfishing, which were identified as representative of the highest exposure and risk related to site use. Risks were highest for the seafood consumption scenario; cancer risks ranged from 9E-5 for the adult API RME scenario to 2E-3 for the adult RME scenario. Cancer risks for the netfishing scenario (2E-6 for the CT scenario and 6E-6 for the RME scenario) and the beach play scenario (4E-6 for the Kellogg Island area to 5E-6 for the southeast intertidal area) were much lower. Based on the exposure scenarios evaluated in the Phase 1 HHRA, the following chemicals were identified as COCs[29] for one or more scenarios: PCBs, arsenic, cPAHs, TCDD TEQs, TBT, and mercury.

There are many uncertainties associated with the risk estimates for each exposure scenario. Due to the health protective nature of assumptions used in the Phase 1 HHRA, risks are potentially overestimated for many individuals. However, by using reasonable maximum exposure assumptions, EPA ensures protection of public health. Despite selection of exposure parameters consistent with RME, some aspects of the assessment may underestimate risks. The collection of additional data or performance of additional analyses could reduce many of the uncertainties. Depending on the direction and magnitude of the uncertainty, additional data could result in the identification of additional COCs or eliminate COCs identified in the current preliminary risk characterization. Data collection efforts for Phase 2 will focus most closely on data gaps associated with either of these two possibilities.

Because risk estimates were highest for the seafood consumption scenario, reducing uncertainties associated with this pathway will be the primary goal of additional data collection efforts. Identification of data gaps was one of the primary objectives of the Phase 1 HHRA. The data gaps will be discussed in a separate technical memorandum. Uncertainties associated with the seafood consumption scenario are higher than uncertainties associated with the other scenarios because fewer site-specific data are available on tissue chemistry and the extent and nature of human site use (i.e., fishing) compared to the direct sediment pathways.

The relative increase in concentrations of arsenic over background concentrations is also uncertain. Based on a preliminary analysis of existing data, concentrations of arsenic in English sole and shellfish from non-urban areas are not greatly different than arsenic concentrations in these organisms obtained from the LDW. Thus, it is uncertain whether actions to reduce arsenic concentrations within the LDW would be effective in limiting exposure to concentrations lower than those typically detected in

[28] Other exposure scenarios, such as swimming, were evaluated qualitatively

[29] A COC has a cancer risk estimate greater than 1E-6 or a HQ greater than 1

Puget Sound sediments or in seafood from typical sources nationally. Moreover, because there were no site-specific data on the concentrations of cPAHs in fish, it was assumed that the concentrations of cPAHs in fish are the same as those in mussels. If the concentrations of cPAHs in fish were assumed to be negligible, as is typically assumed due to the relatively rapid metabolism and elimination of cPAHs in fish (Eisler 1987), the risk estimate for cPAHs in the seafood consumption pathway would be reduced from 1E-4 to 2E-5 (i.e., estimated risks would decrease by a factor of approximately 5).

The risk estimates made in this HHRA for consumption of fish and shellfish exceed levels identified by EPA as the upper end of the acceptable risk range and thus suggest that remedial action may be warranted in the LDW. The results of the Phase 1 HHRA will be used to identify candidate sites for early remedial action. Recommended candidate sites will be described in a separate technical memorandum. It is likely that early remedial actions undertaken within the LDW will reduce the risks described in this HHRA. The Phase 2 HHRA will include a baseline risk assessment for two exposure regimes: 1) sediment conditions at the time the baseline risk assessment is conducted and 2) residual sediment conditions accounting for the effects of the planned early action projects. Identifying the extent of remediation that may be necessary based on the seafood consumption risk estimates will require that a linkage be derived or assumed between sediment and tissue concentrations. It is likely that some type of quantitative modeling of this linkage will be performed as part of the Phase 2 RI.

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GIS Maps

In the electronic version of this HHRA, GIS maps are found in the separate file HHRA_maps.pdf. Their titles are listed here for reference.

Map B-1. Lower Duwamish Waterway study area

Map B-2. Lower Duwamish Waterway surface sediment sampling locations (subtidal and intertidal)

Map B-3. Lower Duwamish Waterway intertidal surface sediment sampling locations

Map B-4. Lower Duwamish Waterway tissue collection locations

Map B-5. Total PCB concentration by Thiessen polygon

Subappendix B.1 Summary of King County Water Quality Assessment HHRA – Direct exposure pathways

INTRODUCTION

King County (1999b) conducted the Combined Sewer Overflow (CSO) Water Quality Assessment (WQA) for the Duwamish River and Elliott Bay to determine whether unacceptable human health risks are associated with CSO discharges. The results of the HHRA were reviewed during the preparation of the Phase 1 HHRA for the LDW. Several direct exposure pathways were quantitatively evaluated in the WQA that are not being evaluated in the LDW Phase 1 HHRA. This subappendix summarizes the methods and results from the WQA for these direct exposure pathways and provides a rationale for the exclusion of these pathways from the LDW Phase 1 HHRA. This subappendix does not include any discussion of the other components of the WQA HHRA, specifically the fish consumption pathway and the pathogen risk assessment.

EXPOSURE ASSESSMENT

Potentially exposed populations and exposure pathways

King County characterized the potentially exposed populations and described exposure pathways for them in Issue Paper 3 (Human site use) of the WQA. The following direct exposure pathways were identified:

- Swimming and wading – may occur from Duwamish Park in LDW; not popular compared to other locations such as Alki Beach
- SCUBA diving – none identified in the LDW
- Boating and sailing – little or no activity in LDW
- Wind surfing – little or no activity in LDW
- Jet skiing – little or no activity in LDW
- Canoeing and kayaking – occurs in LDW but not quantified
- Water skiing – little or no activity in LDW
- Parasailing – little or no activity in LDW
- Occupational exposures – occurs in LDW, but number of people and exposure frequency not known
- Line fishing and net fishing – preferred collection method for Elliott Bay, but frequency for LDW not known
- Collecting organisms other than fish (e.g., crabs, and squid) – frequency very low in LDW compared to Elliott Bay

Exposure parameters

Direct exposures to water and sediment were assessed for 1) swimming, 2) SCUBA diving, 3) windsurfing, and 4) net fishing. Net fishing will not be discussed further in this subappendix because this scenario is included in the LDW Phase 1 HHRA. SCUBA diving and windsurfing were not quantified for the LDW since these activities are not expected to occur in the LDW. Exposure pathways evaluated for the three other activities besides net fishing are described in Table 1.

Table 1. Exposure pathways evaluated used for estimating direct exposures to water and sediment in WQA HHRA

Activity	Exposure Pathways			
	Incidental Water Ingestion	Skin Contact with Water	Incidental Sediment Ingestion	Skin Contact with Sediment
Swimming – LDW and Elliott Bay	X	X	X	X
SCUBA diving – Elliott Bay	X	X		
Windsurfing – Elliott Bay	X	X		

Some of the direct exposure pathways described in the section above were not quantified. Direct exposures via sailing, boating, kayaking, parasailing, water skiing and jet skiing were not evaluated because exposures from these activities are expected to be similar or less than exposures occurring while windsurfing. Similarly, direct exposures while wading were not assessed because exposures while swimming are expected to be larger and provide a more health protective estimate of exposure. Finally, direct exposures while line fishing and gathering shellfish and other organisms were not assessed because these exposures were assumed to be smaller than those experienced while net fishing because of the much lower exposure frequency associated with the former activities.

Direct exposure pathways were quantified using three different estimates of exposure magnitude (low, medium, and high) based on King County's understanding of human site use of the LDW and Elliott Bay. King County believed that fewer people would engage in these activities at the high exposure frequency than at the low or medium exposure frequencies. Children were assessed separately for the swimming exposure pathway. Children were not assessed for SCUBA diving or windsurfing because it was believed that few children engage in these activities.

Exposure parameters, with the exception of exposure point concentrations, which are described in a later section, are shown in Tables 2 (general assumptions), 3 (direct pathways), and 4 (chemical-specific parameters used in the dermal risk assessment). The exposure parameters for the LDW HHRA are similar to the "high value" exposure levels in Tables 2 and 3 for most parameters.

Table 2. General human health exposure assumptions

Exposure Parameter	Exposure Level			Units	Source
	Low Value	Medium Value	High Value		
Adult body weight	79	70	60	kg	EPA (1991b)
Child age 1 to 6 body weight	18	17	15	kg	EPA (1991b)
Child age 7 to 12 body weight	37	35	29	kg	EPA (1991b)
Child age 13 to 18 body weight	64	59	52	kg	EPA (1991b)
Adult exposure duration	9	33	75	yr	EPA (1991b)
Child exposure duration	6	6	6	yr	best professional judgment
Adult averaging time for non-carcinogens	9	33	75	yr	EPA (1996)
Child averaging time for non-carcinogens	6	6	6	yr	best professional judgment
Lifespan	75	75	75	yr	EPA (1996)

Table 3. Human health exposure assumptions for direct pathways

Exposure Parameter	Exposure Level			Units	Source
	Low Value	Medium Value	High Value		
General Exposure Pathways					
Adult incidental water ingestion rate	25	50	75	ml/hr	EPA (1991b)
Adult incidental sediment ingestion rate	25	50	75	mg/event	EPA (1988)
Adult sediment deposition rate to skin	0.16	0.036	0.66	mg/cm^2	EPA (1991b)
Child sediment deposition rate	5	16	25	mg/cm^2	EPA (1996)
Absorption fraction from sediment for inorganics	0.001	0.005	0.01	unitless	EPA (1992)
Absorption fraction from sediment for organics	0.01	0.05	0.1	unitless	EPA (1992)
Scuba Diver Scenario					
Adult scuba diving frequency	2	12	24	event/yr	EPA (1988); best professional judgment
Adult scuba diving event time	0.17	1	2.6	hr/event	EPA (1988); best professional judgment
Adult skin surface area exposed to water	4,900	19,400	21,800	cm^2	EPA (1996)
Windsurfer Scenario					
Adult wind surfing frequency	2	12	24	event/yr	EPA (1988); best professional judgment
Adult wind surfing event time	0.17	1	2.6	hr/event	EPA (1988); best professional judgment
Adult skin surface area exposed to water	4,900	19,400	21,800	cm^2	EPA (1996)
Swimming Scenario					
Adult swimming frequency	2	12	24	event/yr	EPA (1996); best professional judgment

	Exposure Level				
Child swimming frequency	2	12	24	event/yr	EPA (1996); best professional judgment
Adult swimming event time	0.17	1	2.6	hr/event	EPA (1988); best professional judgment
Child swimming event time	0.25	1	2.6	hr/event	EPA (1988); best professional judgment
Adult skin surface area exposed to water	4,900	19,400	21,800	cm^2	EPA (1991b)
Child age 3 to 6 skin surface area exposed to water	6,200	7,200	8,400	cm^2	EPA (1991b)
Child age 7 to 12 skin surface area exposed to water	9,000	10,400	12,500	cm^2	EPA (1991b)
Child age 13 to 18 skin surface area exposed to water	13,800	15,800	18,400	cm^2	EPA (1991b)
Adult skin surface area exposed to sediment	4,900	9,300	17,000	cm^2	EPA (1991b)
Child age 3 to 6 skin surface area exposed to sediment	6,200	7,200	8,400	cm^2	EPA (1991b)
Child age 7 to 12 skin surface area exposed to sediment	9,000	10,400	12,500	cm^2	EPA (1991b)
Child age 13 to 18 skin surface area exposed to sediment	12,700	14,500	16,900	cm^2	EPA (1991b)

Table 4. Chemical specific parameters used in dermal exposure assessment

COPC	K_P (cm/hr)	T (hr)	Tss (hr)	B (unitless)
Metals/Metalloids				
Arsenic	0.001	n/a	n/a	n/a
Cadmium	0.001	n/a	n/a	n/a
Copper	0.001	n/a	n/a	n/a
Lead	0.000004	n/a	n/a	n/a
Mercury	0.001	n/a	n/a	n/a
Nickel	0.0001	n/a	n/a	n/a
Zinc	0.0006	n/a	n/a	n/a
Organometallics				
Tributyltin	0.00596	5.14	18.2	0.155
Polychlorinated Biphenyls				
Aroclor 1016	0.988	3.28	15.5	110
Aroclor 1221	2.20	1.47	6.95	110
Aroclor 1232	1.42	2.28	10.7	110
Aroclor 1242	0.870	3.72	17.6	110
Aroclor 1248	0.548	5.91	27.9	110
Aroclor 1254	0.369	8.76	41.4	110
Aroclor 1260	0.188	17.2	81.2	110
Total PCBs	0.369	8.76	41.4	110
Semivolatile Organics				
1,4-Dichlorobenzene	0.0732	0.690	3.70	0.313

COPC	K_P (cm/hr)	T (hr)	T_{ss} (hr)	B (unitless)
4-Methylphenol	0.0180	0.399	0.957	0.00933
Benzo(a)anthracene	0.892	2.15	10.2	52.5
Benzo(a)pyrene	1.12	3.01	14.2	116
Benzo(b)fluoranthene	1.69	3.01	14.2	208
Benzo(e)pyrene	1.12	3.01	14.2	116
Benzo(g,h,i)perylene	3.24	4.22	19.9	835
Benzo(k)fluoranthene	2.09	3.01	14.2	280
BEHP	0.0225	21.2	104	7.41
Chrysene	0.886	2.15	10.2	52.0
Dibenzo(a,h)anthracene	1.56	4.34	20.5	310
Fluoranthene	0.550	1.49	7.14	15.9
Phenanthrene	0.224	1.07	5.59	2.79
Pyrene	0.434	1.49	7.19	11.5
Indeno(1,2,3-cd)pyrene	4.34	4.22	19.9	1,260

n/a – not applicable

Chemicals of potential concern

A multi-step screening process was conducted to identify COPCs to be evaluated for both the human health and ecological risk assessments in the WQA. Details of the screening process are outlined in Subappendix A2 (Analysis Plan) in Section 4.5 (Identification of Candidate Chemical Stressors) of King County (1999c).

Selecting candidate COPCs involved: 1) determining the presence and quantity of chemicals in the study area as well as analytical detection limit goals, 2) identifying water and sediment criteria for use in screening these values, and 3) developing surrogate approaches to handle the number of chemicals that were eventually selected. King County's 1997 sampling program analyzed 45 candidate chemical COPCs, as well as fecal coliform bacteria. Chemicals were initially screened for their ability to cause human cancer. Chemicals with this characteristic were automatically included as COPCs. Non-cancer-causing chemicals were then screened for frequency of detection. Infrequently detected chemicals (i.e., frequency of detection ≤5%) were then further evaluated to determine if the method detection limit (MDL) was less than the detection goal.[30]

Infrequently detected chemicals with MDLs greater than detection goals were not selected as candidate COPCs, and instead were identified as posing uncertain risk. Chemicals frequently detected in the water column were compared with freshwater and saltwater State of Washington water quality standards, US EPA water quality criteria, or literature-based toxicity values when no standards or criteria were available. Chemicals frequently detected in sediments were compared with freshwater

[30] The detection goal is the lowest concentration of this chemical that would produce a hazard quotient of one for the most sensitive receptor, based on the identified exposure pathway for that receptor.

guidelines (i.e., US EPA sediment quality criteria or US EPA ecotox thresholds, or literature-based toxicity values when no freshwater guidelines were available) and saltwater guidelines or standards (i.e., Washington State Sediment Management Standards, US EPA Ecotox Thresholds, literature-based values, or application of equilibrium partitioning to acute water quality criteria after applying an appropriate safety factor). The hierarchy for selecting screening criteria is presented in Tables 4-1 and 4-2 in Subappendix A2 of King County (1999c). Chemical screening was conducted using the sample 95th percentile for water chemistry data and the 95th percentile upper confidence limit on the mean for sediment chemistry data. The sample 95th percentile for water chemistry data was selected to be protective for potential effects to aquatic life from acute exposures, and was also expected to be health protective for the purposes of the WQA HHRA. The 95th percentile upper confidence limit on the mean for sediment chemistry data was selected to be health protective for long-term water column exposure to sediment constituents (dissolved and particulate) in the WQA HHRA.

Chemicals with percentiles exceeding the criteria were selected as being candidate COPCs requiring further evaluation in the detailed risk assessment. Those with percentiles less than criteria were not selected for further evaluation because they posed insignificant risks.

COPCs selected by King County for the WQA HHRA are identified in Table 5, along with the rationale for their selection. Chemicals targeted by King County in their 1997 sampling program that were not selected as COPCs are listed in Table 6.

The King County COPCs list differs from the LDW COPCs list due to differences in the way in which the COPCs were selected. Both lists, however, include COPCs that were identified by King County as risk drivers for human health, specifically arsenic and total PCBs.

Table 5. Chemicals selected as candidate COPCs

CHEMICAL	REASON FOR SELECTION
1,4-Dichlorobenzene	Exceeded sediment criterion, 1997 sampling data
4-Methylphenol	Exceeded sediment criterion, Duwamish/Diagonal Study
Arsenic	Exceeded sediment criterion, Duwamish/Diagonal Study; present in mussel tissue, 1996-1997 sampling data
Benzo(a)anthracene	Known human carcinogen, present in mussel tissue, 1996-1997 sampling data
Benzo(a)pyrene	Known human carcinogen
Benzo(b)fluoranthene	Known human carcinogen, present in mussel tissue, 1996-1997 sampling data
Benzo(g,h,i)perylene	Exceeded sediment criterion, Norfolk Study
Benzo(k)fluoranthene	Known human carcinogen
BEHP	Exceeded sediment criterion, 1997 sampling data, present in mussel tissue, 1996-1997 sampling data
Cadmium	CSO concentrations exceeded water criterion and present in mussel tissue, 1996-1997 sampling data

CHEMICAL	REASON FOR SELECTION
Chrysene	Known human carcinogen, present in mussel tissue, 1996-1997 sampling data
Copper	CSO concentrations exceeded water criterion and present in mussel tissue, 1996-1997 sampling data
Dibenzo(a,h)anthracene	Known human carcinogen
Fluoranthene	Exceeded sediment criterion and present in mussel tissue, 1996-1997 sampling data
Indeno(1,2,3-c,d)pyrene	Exceeded sediment criterion, 1997 sampling data
Lead	CSO concentrations exceeded water criterion and present in mussel tissue, 1996-1997 sampling data
Mercury	Exceeded sediment criterion, Duwamish/Diagonal Study; present in mussel tissue, 1996-1997 sampling data
Nickel	CSO concentrations exceeded water criterion and present in mussel tissue, 1996-1997 sampling data
Phenanthrene	Exceeded sediment criterion and present in mussel tissue, 1996-1997 sampling data
Pyrene	Present in mussel tissue, 1996-1997 sampling data
Total PCBs	Exceeded sediment criterion, 1997 sampling data
Tributyltin	Present in mussel tissue, 1996-1997 sampling data
Zinc	CSO concentrations exceeded water criterion and present in mussel tissue, 1996-1997 sampling data; Sediment exceedance, Duwamish/Diagonal Study

Table 6. Chemicals not selected as COPCs

CHEMICAL	REASON NOT SELECTED	COMMENT
2-Methylphenol	Not detected in sediment or water	Present in mussel tissue, 1996-1997
Aldrin	Not detected in any media	Exceeded detection limit goals
Antimony	No state criteria for evaluation	Draft EPA 1988 acute AWQC = 30 µg/L; chronic AWQC = 88 µg/L
Aroclor 1254	Not detected in sediment or water	Present in mussel tissues, 1997 sampling
Barium	Overall toxicity is low	Exceeds water criterion in CSO effluent
Benzidine	Overall toxicity is low	Exceeds 1997 sampling data detection limit goals in background water, CSO effluent, mussel tissue
Benzo(e)pyrene	Not detected in any media	Exceeded detection limit goals
Benzoic acid	Overall toxicity is low	Sediment exceedance in Norfolk Study, detected in mussel tissue, 1997 sampling data
Benzyl alcohol	Not detected in sediment or water	Identified in 1995 WQA report, present in mussel tissue, 1996-1997 sampling data
Benzyl butyl phthalate	Not detected in sediment or water	Sediment exceedance in Duwamish/Diagonal Study, present in mussel tissue, 1996-1997 sampling data
Beryllium	Not detected in any media	Exceeded minimum detection limit goals in background water and CSO effluent
Chromium (total)	Not detected in sediment or water	Only present in mussel tissue as chromium III

CHEMICAL	REASON NOT SELECTED	COMMENT
Dibenzofuran	Not detected in any media	All non-detects in sediment, background water and CSO effluent, 1996-1997 sampling data.
Dieldrin	Not detected in any media	Exceeded detection limit goals
Gamma-BHC	Not detected in any media	Exceeded detection limit goals
Heptachlor	Not detected in any media	Exceeded detection limit goals
Hexachlorobenzene	Not detected in any media	Exceeded detection limit goals and exceeded sediment criterion in the Duwamish/Diagonal Study
Iron	Infrequently detected	What data is available has 95th percentile greater than criteria and exceeded background water detection limit goals.
Pentachlorophenol	Not detected in any media	Exceeded detection limit goals in background water and CSO effluent
Silver	Not detected in any media	Identified in 1995 WQA report
Total HPAHs	Not detected in any media	Identified in sediments in Duwamish/Diagonal Study and 1995 WQ report; assessed as individual chemicals
Vanadium	Overall toxicity is low	Exceeded water criterion in CSO effluent

NOTE: HPAH – high-molecular-weight polycyclic aromatic hydrocarbon

Exposure point concentrations

EPCs in water and sediment were estimated using the results from a three-dimensional fate and transport hydrodynamic model. The model was calibrated to the results of the sampling and analysis program, which included the collection of about 2,000 samples and about 13,000 chemical analyses. The model divided the river (north of the Interstate 405 Bridge) and Elliott Bay into 512 cells, which were then divided into 10 layers resulting in 5,120 cell-layers. Sediments were also modeled for each cell. Chemical inputs from the Green River upstream of the study area, the Puget Sound boundary, CSOs, sediments, and other sources were accounted for within the model.

Chemical exposure concentrations in water and sediment were estimated as the annual mean concentrations for each exposure location. When a location included more than one cell (e.g., all cells within Elliott Bay), the exposure concentrations were calculated as the mean of the annual mean concentration of each cell. Mean concentrations based on thousands of model simulations were used to calculate EPCs. The exposure locations and the use of the model for calculating EPCs are presented in Table 7.

Table 7. Locations of direct human exposure to water and sediment and use of cell-layers from EFDC hydrodynamic model

ACTIVITY	LOCATION	WATER CELLS USED	SEDIMENT CELLS USED
Swimming	Duwamish River at Duwamish Park	Surface cells only	Yes
	Elliott Bay at Duwamish Head	Surface cells only	Yes
SCUBA diving	Elliott Bay at Seacrest Park	All depths	No
Windsurfing	Elliott Bay – entire bay	Surface cells only	No

The modeled EPCs used in the KC HHRA are shown in Table 8. The water EPCs were based on both water and sediment (i.e., resuspension) sources. Water quality data collected during the WQA were used to calibrate the model-derived EPCs for most COPCs, including arsenic. Water samples were also analyzed for organic compounds, but many compounds, including PAHs, were never detected.[31] For certain compounds (i.e., PCBs and PAHs), water concentrations were estimated using the results from the deployment and subsequent analysis of semi-permeable membrane devices. For other chemicals, such as TBT, resident and transplanted mussel samples were used to estimate water concentrations.

[31] Semi-volatile organic compounds were analyzed using EPA Method 625.Method detection limits (MDLs) ranged from 0.1 to 1 µg/L, well within the target MDLs specified in the Quality Assurance Project Plan (see Table A-5 in Subappendix A in King County Combined Sewer Overflow Water Quality Assessment for the Duwamish River and Elliott Bay. Appendix A: Problem Formulation, Analysis Plan, and Field Sampling Work Plan, February 1999).

Table 8. Baseline modeled EPCs in water and sediment used in the KC WQA HHRA

COPC	Swimming				SCUBA	Windsurfing
	Duwamish Park		Duwamish Head		Seacrest	Elliott Bay
	Water (mg/L)	Sediment (mg/kg ww)	Water (mg/L)	Sediment (mg/kg ww)	Water (mg/L)	Water (mg/L)
Metals/Metalloids						
Arsenic	7.4E-4	3.6	0.0012	6.3	0.0012	0.0012
Cadmium	4.1E-5	0.13	6.3E-5	0.78	6.5E-5	6.2E-5
Copper	0.0021	15	7.9E-4	41	5.5E-4	8.0E-4
Lead	7.5E-4	55	2.2E-4	28	1.1E-4	2.0E-4
Mercury	6.1E-6	0.0065	1.5E-6	0.18	8.3E-7	1.5E-6
Nickel	0.0011	6.3	6.7E-4	15	5.8E-4	6.6E-4
Zinc	0.0047	24	0.0015	70	8.1E-4	0.0014
Organometallics						
Tributyltin	1.4E-6	0.0040	3.1E-7	0.27	1.1E-7	3.0E-7
Polychlorinated biphenyls						
Total PCBs	1.4E-5	0.061	1.2E-5	0.12	6.1E-6	1.5E-5
Semivolatile organics						
1,4-Dichlorobenzene	5.7E-6	5.4E-4	2.1E-6	0.048	8.9E-7	2.5E-6
4-Methylphenol	6.0E-5	0.0029	1.4E-5	0.061	5.4E-6	1.6E-5
Benzo(a)anthracene	1.3E-6	0.0088	7.1E-8	0.21	2.8E-8	7.2E-8
Benzo(a)pyrene	1.5E-7	0.0010	8.2E-9	0.024	3.2E-9	8.3E-9
Benzo(b)fluoranthene	2.9E-6	0.035	4.7E-7	0.18	1.7E-7	4.5E-7
Benzo(g,h,i)perylene	1.1E-6	0.013	1.8E-7	0.068	6.4E-8	1.7E-7
Benzo(k)fluoranthene	9.9E-7	0.015	1.7E-7	0.21	6.2E-8	1.6E-7
BEHP	1.2E-4	0.26	7.8E-5	0.098	6.9E-5	7.9E-5
Chrysene	1.4E-6	0.0092	7.4E-8	0.22	2.9E-8	7.5E-8
Dibenzo(a,h)anthracene	1.2E-7	0.0014	1.9E-8	0.0071	6.8E-9	1.8E-8
Fluoranthene	1.1E-5	0.033	1.4E-6	0.33	5.2E-7	1.4E-6
Phenanthrene	2.6E-5	0.0084	2.6E-6	0.20	9.5E-7	2.4E-6
Pyrene	2.3E-5	0.22	5.6E-7	0.36	4.9E-7	5.3E-7
Indeno(1,2,3-c,d)pyrene	4.4E-7	0.0052	7.1E-8	0.027	2.5E-8	6.7E-8

RISK CHARACTERIZATION

King County's risk characterization was divided into a discussion of non-carcinogenic effects, using hazard quotients (HQs), and carcinogenic effects, using estimates of excess cancer risk.

Non-carcinogenic health effects

No HQs exceeding one were predicted for any of the three exposure scenarios shown in Table 1 for either adults or children at any exposure level. To assess the potential for cumulative health effects associated with multiple chemical exposures, a hazard index

was calculated for each exposure pathway as the sum of the chemical-specific HQs. All hazard indices were less than 1.0, indicating negligible non-carcinogenic health effects by direct exposure to sediment or water to swimmers, SCUBA divers or windsurfers, regardless of age during which the exposures may occur and the frequency that exposure occurs.

Cancer risk estimates

When risks are evaluated over all exposure levels and COPCs, cancer risk predictions were less than one in one million (0.000001) for windsurfers and SCUBA divers at all exposure levels and for swimmers at medium and low exposure levels. Total incremental carcinogenic risks across all COPCs were also predicted to be less than one in one million for these scenarios. Risks exceeding one chance in a million were predicted for people swimming at Duwamish Head in Elliott Bay or in the Duwamish River at Duwamish Park at high exposure levels. Excess cancer risks were highest for arsenic and total PCBs; risks from other COPCs (not shown) were several orders of magnitude less. Table 9 shows the risk estimates for arsenic and PCBs; estimates were 60-100% higher for Elliott Bay.

Table 9. Predicted incremental cancer risks (x 10^{-6}) from arsenic and PCBs to highly exposed adult and child swimmers at Duwamish Park in the Duwamish River

COPC	Age Group	Water		Sediment		Total
		Ingestion	Dermal	Ingestion	Dermal	
Arsenic	1 to 6	0.08	0.009	0.4	4.0	4.4
	7 to 12	0.04	0.006	0.2	3.1	3.3
	13 to 18	0.02	0.005	0.1	2.3	2.4
	Adult	0.2	0.07	0.4	0.7	1.1
PCBs	1 to 6	0.0004	0.08	0.01	0.9	0.9
	7 to 12	0.0002	0.006	0.004	0.7	0.7
	13 to 18	0.0001	0.05	0.002	0.5	0.5
	Adult	0.001	0.7	0.01	0.2	0.2

The results shown in Table 8 indicate that the contribution to the combined risk estimate from the water pathway is insignificant compared to the sediment pathway.

Applicability of King County WQA Results to LDW HHRA

The Phase 1 HHRA for the LDW includes two scenarios that characterize direct exposure to sediments: beach play and netfishing. Based on the risk characterization presented above, the LDW HHRA does not include any of the direct pathways identified in Table 1. The scenarios characterized in the LDW HHRA should be protective of current and future uses of the LDW.

The exclusion of these alternate exposure pathways that were characterized in the KC assessment from the LDW HHRA is appropriate for the following reasons:

- The water-only scenarios (i.e., SCUBA diving and wind surfing) are expected to occur rarely, if at all, in the LDW
- The risks associated with the water component (which is not quantified for any LDW HHRA exposure scenario) of the swimming scenario are insignificant compared to risks associated with the sediment component, even though the hydrodynamic model used by King County to generate EPCs accounted for sediment resuspension
- Although the COPCs identified in the two risk assessments are slightly different, both assessments include arsenic and PCBs, which were associated with almost all the identified excess cancer risk in the King County HHRA
- The exposure parameters for the RME beach play scenario in the LDW HHRA are more health protective than the exposure parameters for the high-level swimming scenario in the King County HHRA, as shown in Table 10

Table 10. Comparison of selected exposure parameters for LDW HHRA beach play scenario and King County HHRA swimming scenario

EXPOSURE PARAMETER	LDW HHRA (RME scenario)	KING COUNTY HHRA (high-level exposure scenario)
Exposure frequency	41 days/yr	24 days/yr
Incidental sediment ingestion rate	200 mg dw/day	53 mg dw/day [a]
EPC (arsenic)	11 mg/kg dw (Kellogg Island) 11 mg/kg dw (southeast) 9.4 mg/kg dw (southwest)	5.1 mg/kg dw [a]
EPC (PCBs)	0.15 mg/kg dw (Kellogg Island) 1.3 mg/kg dw (southeast) 0.65 mg/kg dw (southwest)	0.087 mg/kg dw [a]

[a] Incidental sediment ingestion rates and EPCs presented by King County were in wet weight. The values presented in this table were converted to dry weight units to facilitate comparison with the LDW HHRA convention, assuming sediment was 70% solids

Subappendix B.2 Occurrence, Distribution, and Selection of Chemicals of Potential Concern

This subappendix documents the selection process for COPCs using the table format suggested in RAGS Part D (EPA 1998a). This information is summarized in Section B.3.3. Table 1 (netfishing scenario) is based on both intertidal and subtidal surface chemistry data and uses soil RBCs from EPA Region 9 (EPA 1999a) for an industrial exposure scenario. Table 2 (beach play scenario) is based on intertidal surface chemistry data only and uses soil RBCs from EPA Region 9 (EPA 1999a) for a residential exposure scenario. Table 3 includes tissue chemistry data from the composite samples of English sole, perch, crab, and mussels summarized in Table B-2 in the main document and uses RBCs from EPA Region 3 (EPA 2001a). Table 4 includes a list of chemicals measured in sediment (Table 1), but not in tissue (Table 3). Data in Tables 1 and 2 are summarized by location to match the maps in the RI map folio. Data in Table 3 are summarized by sample.

Table 1. Occurrence, distribution, and selection of chemicals of potential concern for sediment in the netfishing exposure scenario

CAS Number	Chemical	Detection frequency (location)	Min. Detected Conc.	Max. detected Conc.	Units	Range of detection limits	Conc. Used for Screening	Background conc. [a]	RBC [b]	COPC Flag	Rationale for COPC Selection or Exclusion
630-20-6	1,1,1,2-Tetrachloroethane	0/44	nd	nd	µg/kg dw	1.5 - 533	533	not eval.	7000 ca	no	bsl
71-55-6	1,1,1-Trichloroethane	0/49	nd	nd	µg/kg dw	1.4 - 533	533	not eval.	1,400,000 sat	no	bsl
79-34-5	1,1,2,2-Tetrachloroethane	0/49	nd	nd	µg/kg dw	1.4 - 533	533	not eval.	900 ca	no	bsl
79-00-5	1,1,2-Trichloroethane	0/49	nd	nd	µg/kg dw	1.4 - 1060	1060	not eval.	1900 ca*	no	bsl
76-13-1	1,1,2-Trichlorotrifluoroethane	0/47	nd	nd	µg/kg dw	1.5 - 1060	1060	not eval.	5,600,000 sat	no	bsl
513-88-2	1,1-Dichloroacetone	0/42	nd	nd	µg/kg dw	3.0 - 2660	2660	not eval.	na	no	ntx
75-34-3	1,1-Dichloroethane	0/49	nd	nd	µg/kg dw	1.4 - 533	533	not eval.	210,000 nc	no	bsl
75-35-4	1,1-Dichloroethene	0/49	nd	nd	µg/kg dw	1.4 - 1060	1060	not eval.	120 ca	yes	ifd [c]
563-58-6	1,1-Dichloropropene	0/44	nd	nd	µg/kg dw	1.5 - 533	533	not eval.	na	no	teq
35822-46-9	1,2,3,4,6,7,8-HpCDD	27/29	48	6600	ng/kg dw	0.99 - 1.1	6600	not eval.	na	no	teq
67562-39-4	1,2,3,4,6,7,8-HpCDF	26/29	8.3	1600	ng/kg dw	0.62 - 7.7	1600	not eval.	na	no	teq
55673-89-7	1,2,3,4,7,8,9-HpCDF	11/29	3.9	270	ng/kg dw	0.77 - 4.2	270	not eval.	na	no	teq
39227-28-6	1,2,3,4,7,8-HxCDD	2/29	27	72	ng/kg dw	0.72 - 5.4	72	not eval.	na	no	teq
70648-26-9	1,2,3,4,7,8-HxCDF	14/29	4.2	540	ng/kg dw	0.29 - 4.2	540	not eval.	na	no	teq
57653-85-7	1,2,3,6,7,8-HxCDD	20/29	5.6	290	ng/kg dw	0.74 - 4.3	290	not eval.	na	no	teq
57117-44-9	1,2,3,6,7,8-HxCDF	2/29	20	74	ng/kg dw	0.22 - 4.3	74	not eval.	na	no	teq
19408-74-3	1,2,3,7,8,9-HxCDD	15/29	4.8	120	ng/kg dw	0.84 - 4.8	120	not eval.	400 ca	no	teq
72918-21-9	1,2,3,7,8,9-HxCDF	1/29	16	16	ng/kg dw	0.12 - 2.4	16	not eval.	na	no	teq
40321-76-4	1,2,3,7,8-PeCDD	2/29	12	22	ng/kg dw	0.53 - 4.1	22	not eval.	na	no	teq
57117-41-6	1,2,3,7,8-PeCDF	1/29	54	54	ng/kg dw	0.28 – 5.0	54	not eval.	na	no	teq
87-61-6	1,2,3-Trichlorobenzene	0/44	nd	nd	µg/kg dw	1.8 – 1060	1060	not eval.	na	no	ntx
96-18-4	1,2,3-Trichloropropane	0/44	nd	nd	µg/kg dw	1.5 – 1060	1060	not eval.	3.1 ca	yes	asl
120-82-1	1,2,4-Trichlorobenzene	7/557	0.76	191	µg/kg dw	0.35 - 2100	2100	not eval.	3,000,000 sat	no	bsl
95-63-6	1,2,4-Trimethylbenzene	2/44	0.54	1.5	µg/kg dw	1.5 – 533	533	not eval.	5700 sat	no	bsl

CAS Number	Chemical	Detection frequency (location)	Min. Detected Conc.	Max. detected Conc.	Units	Range of detection limits	Conc. Used for Screening	Background conc. [a]	RBC [b]	COPC Flag	Rationale for COPC Selection or Exclusion
96-12-8	1,2-Dibromo-3-chloropropane	0/44	nd	nd	µg/kg dw	3.7 – 1060	1060	not eval.	4000 ca**	no	bsl
106-93-4	1,2-Dibromoethane (EDB)	0/44	nd	nd	µg/kg dw	1.5 – 1060	1060	not eval.	48 ca*	yes	ifd [d]
95-50-1	1,2-Dichlorobenzene	35/557	1.3	555	µg/kg dw	0.35 - 2100	2100	not eval.	370,000 sat	no	bsl
107-06-2	1,2-Dichloroethane	0/49	nd	nd	µg/kg dw	1.4 – 533	533	not eval.	760 ca*	no	bsl
540-59-0	1,2-Dichloroethene (total)	0/2	nd	nd	µg/kg dw	23 – 24	24	not eval.	na	no	ntx
78-87-5	1,2-Dichloropropane	0/49	nd	nd	µg/kg dw	1.4 – 533	533	not eval.	770 ca*	no	bsl
122-66-7	1,2-Diphenylhydrazine	0/87	nd	nd	µg/kg dw	13 – 120	120	not eval.	3100 ca	no	bsl
108-67-8	1,3,5-Trimethylbenzene	1/44	1.4	1.4	µg/kg dw	1.5 – 533	533	not eval.	7000 nc	no	bsl
541-73-1	1,3-Dichlorobenzene	9/550	0.83	99.2	µg/kg dw	0.35 - 2100	2100	not eval.	5200 nc	no	bsl
142-28-9	1,3-Dichloropropane	0/44	nd	nd	µg/kg dw	1.5 – 533	533	not eval.	na	no	ntx
106-46-7	1,4-Dichlorobenzene	69/557	0.74	1900	µg/kg dw	0.18 - 2100	2200	not eval.	8100 ca	no	bsl
109-69-3	1-Chlorobutane	0/44	nd	nd	µg/kg dw	1.5 – 533	533	not eval.	480,000 sat	no	bsl
90-12-0	1-Methylnaphthalene	3/3	13	41	µg/kg dw	na	41	not eval.	na	no	ntx
832-69-9	1-Methylphenanthrene	3/3	27	92	µg/kg dw	na	92	not eval.	na	no	ntx
594-20-7	2,2-Dichloropropane	0/44	nd	nd	µg/kg dw	1.5 – 533	533	not eval.	na	no	ntx
60851-34-5	2,3,4,6,7,8-HxCDF	2/29	18	32	ng/kg dw	0.29 - 2.5	32	not eval.	na	no	teq
57117-31-4	2,3,4,7,8-PeCDF	2/29	8.8	58	ng/kg dw	0.44 - 5.4	58	not eval.	na	no	teq
2245-38-7	2,3,5-Trimethylnaphthalene	3/3	18	71	µg/kg dw	na	71	not eval.	na	no	ntx
1746-01-6	2,3,7,8-TCDD	3/29	2.0	3.8	ng/kg dw	0.27 - 1.1	3.8	not eval.	27 ca	no	teq
	2,3,7,8-TCDD TEQ	29/29	1.2	224	ng/kg dw	na	224	not eval.	27 ca	yes	asl
51207-31-9	2,3,7,8-TCDF	19/29	0.99	6.8	ng/kg dw	0.18 - 1.7	6.8	not eval.	na	no	teq
95-95-4	2,4,5-Trichlorophenol	0/527	nd	nd	µg/kg dw	16 – 5200	5200	not eval.	8,800,000 nc	no	bsl
88-06-2	2,4,6-Trichlorophenol	0/527	nd	nd	µg/kg dw	18 – 2100	2100	not eval.	220,000 ca	no	bsl
53-19-0	2,4'-DDD	0/3	nd	nd	µg/kg dw	0.63 - 2.9	2.9	not eval.	na	no	ntx
3424-82-6	2,4'-DDE	0/3	nd	nd	µg/kg dw	0.63 - 2.9	2.9	not eval.	na	no	ntx
789-02-6	2,4'-DDT	0/3	nd	nd	µg/kg dw	0.63 - 2.9	2.9	not eval.	na	no	ntx
120-83-2	2,4-Dichlorophenol	0/527	nd	nd	µg/kg dw	22 – 2100	2100	not eval.	260,000 nc	no	bsl

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105-67-9	2,4-Dimethylphenol	1/553	170	170	µg/kg dw	5.9 – 2100	2100	not eval.	1,800,000 nc	no	bsl
51-28-5	2,4-Dinitrophenol	0/525	nd	nd	µg/kg dw	19 – 5200	5200	not eval.	180,000 nc	no	bsl
121-14-2	2,4-Dinitrotoluene	0/527	nd	nd	µg/kg dw	4.0 – 2100	2100	not eval.	180,000 nc	no	bsl
581-42-0	2,6-Dimethylnaphthalene	3/3	33	82	µg/kg dw	na	82	not eval.	na	no	ntx
606-20-2	2,6-Dinitrotoluene	0/527	nd	nd	µg/kg dw	13 – 2100	2100	not eval.	88,000 nc	no	bsl
110-75-8	2-Chloroethyl vinyl ether	0/3	nd	nd	µg/kg dw	7.0 – 12	12	not eval.	na	no	ntx
91-58-7	2-Chloronaphthalene	0/527	nd	nd	µg/kg dw	19 - 2100	2100	not eval.	2,700,000 nc	no	bsl
95-57-8	2-Chlorophenol	0/527	nd	nd	µg/kg dw	11 - 2100	2100	not eval.	24,000 nc	no	bsl
95-49-8	2-Chlorotoluene	0/44	nd	nd	µg/kg dw	1.5 - 533	533	not eval.	57,000 nc	no	bsl
591-78-6	2-Hexanone	0/49	nd	nd	µg/kg dw	3.0 - 2130	2130	not eval.	na	no	ntx
91-57-6	2-Methylnaphthalene	87/557	2.66	2370	µg/kg dw	1.8 - 2100	2370	not eval.	na	no	ntx
2531-84-2	2-Methylphenanthrene	3/3	31	123	µg/kg dw	na	123	not eval.	na	no	ntx
95-48-7	2-Methylphenol	2/557	20	55	µg/kg dw	5.9 - 2100	2100	not eval.	4,400,000 nc	no	bsl
88-74-4	2-Nitroaniline	0/525	nd	nd	µg/kg dw	94 - 5200	5200	not eval.	5000 nc	no	ifd [e]
88-75-5	2-Nitrophenol	0/527	nd	nd	µg/kg dw	20 - 2100	2100	not eval.	na	no	ntx
79-46-9	2-Nitropropane	0/44	nd	nd	µg/kg dw	7.6 - 2660	2660	not eval.	na	no	ntx
91-94-1	3,3'-Dichlorobenzidine	0/513	nd	nd	µg/kg dw	32 - 2100	2100	not eval.	5500 ca	no	bsl
	3-Methylphenol and 4-Methylphenol Coelution	15/276	20	910	µg/kg dw	20 - 200	910	not eval.	na	no	ntx
99-09-2	3-Nitroaniline	0/517	nd	nd	µg/kg dw	110 - 5200	5200	not eval.	na	no	ntx
72-54-8	4,4'-DDD	36/102	2.0	840	µg/kg dw	0.81 - 51	840	not eval.	17,000 ca	no	sum
72-55-9	4,4'-DDE	17/102	1.0	370	µg/kg dw	0.81 - 56	370	not eval.	12,000 ca	no	sum
50-29-3	4,4'-DDT	10/102	2.0	1670	µg/kg dw	0.63 - 56	1670	not eval.	12,000 ca*	no	sum
534-52-1	4,6-Dinitro-o-cresol	0/525	nd	nd	µg/kg dw	64 - 5200	5200	not eval.	na	no	ntx
101-55-3	4-Bromophenyl phenyl ether	0/527	nd	nd	µg/kg dw	12 - 2100	2100	not eval.	na	no	ntx
59-50-7	4-Chloro-3-methylphenol	0/525	nd	nd	µg/kg dw	38 - 2100	2100	not eval.	na	no	ntx
106-47-8	4-Chloroaniline	0/495	nd	nd	µg/kg dw	56 - 2100	2100	not eval.	350,000 nc	no	bsl
7005-72-3	4-Chlorophenyl phenyl ether	0/527	nd	nd	µg/kg dw	18 - 2100	2100	not eval.	na	no	ntx

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106-43-4	4-Chlorotoluene	0/44	nd	nd	µg/kg dw	1.5 - 533	533	not eval.	na	no	ntx
106-44-5	4-Methylphenol	36/281	20	6250	µg/kg dw	7.0 - 2100	6250	not eval.	440,000 nc	no	bsl
100-01-6	4-Nitroaniline	0/516	nd	nd	µg/kg dw	94 - 5200	5200	not eval.	na	no	ntx
100-02-7	4-Nitrophenol	0/525	nd	nd	µg/kg dw	64 - 5200	5200	not eval.	700,000 nc	no	bsl
83-32-9	Acenaphthene	229/557	1.92	3300	µg/kg dw	1.79 - 2100	3300	not eval.	3,800,000 nc	no	bsl
208-96-8	Acenaphthylene	57/557	2.22	110	µg/kg dw	1.8 - 2100	2100	not eval.	na	no	ntx
67-64-1	Acetone	3/49	114	1020	µg/kg dw	11.2 - 21300	21,300	not eval.	620,000 nc	no	bsl
	Acid volatile sulfides	46/56	88	6100	mg/kg dw	48 - 89	6100	not eval.	na	no	ntx
309-00-2	Aldrin	0/100	nd	nd	µg/kg dw	0.40 - 56	56	not eval.	150 ca	no	bsl
107-05-1	Allyl Chloride	0/44	nd	nd	µg/kg dw	1.5 - 1060	1060	not eval.	4,300,000 nc	no	bsl
319-84-6	alpha-BHC	0/100	nd	nd	µg/kg dw	0.40 - 56	56	not eval.	590 ca	no	bsl
5103-71-9	alpha-Chlordane	1/55	26	26	µg/kg dw	0.81 - 37	37	not eval.	na	no	ntx
959-98-8	alpha-Endosulfan	1/56	2.0	2.0	µg/kg dw	0.40 - 100	100	not eval.	530,000 nc [l]	no	bsl
7429-90-5	Aluminum	450/450	2800	110,000	mg/kg dw	na	110,000	12,000/21,000	100,000 max	yes	asl
7664-41-7	Ammonia	18/18	5.4	20	mg/kg dw	na	20	not eval.	na	no	ntx
62-53-3	Aniline	0/54	nd	nd	µg/kg dw	64 - 120	120	not eval.	430,000 ca*	no	bsl
120-12-7	Anthracene	401/557	2.01	9300	µg/kg dw	5.4 - 2000	9300	not eval.	1E8 max	no	bsl
7440-36-0	Antimony	97/389	0.22	110	mg/kg dw	0.20 - 31	110	0.23/0.44	82 nc	yes	asl
12674-11-2	Aroclor-1016	0/652	nd	nd	µg/kg dw	0.87 - 2000	2000	not eval.	29,000 ca**	no	sum
11104-28-2	Aroclor-1221	0/515	nd	nd	µg/kg dw	1.89 - 1600	1600	not eval.	1000 ca	no	sum
11141-16-5	Aroclor-1232	0/515	nd	nd	µg/kg dw	0.87 - 1600	1600	not eval.	1000 ca	no	sum
53469-21-9	Aroclor-1242	75/652	7.8	2400	µg/kg dw	0.87 - 6100	6100	not eval.	1000 ca	no	sum
12672-29-6	Aroclor-1248	101/652	8.3	219,000	µg/kg dw	0.87 - 2300	219,000	not eval.	1000 ca	no	sum
11097-69-1	Aroclor-1254	530/654	2.17	14,000	µg/kg dw	1.31 - 4300	14,000	not eval.	1000 ca*	no	sum
11096-82-5	Aroclor-1260	564/653	1.24	26,000	µg/kg dw	5.6 - 1700	26,000	not eval.	1000 ca	no	sum
37324-23-5	Aroclor-1262	2/2	270	840	µg/kg dw	na	840	not eval.	na	no	ntx, sum
11100-14-4	Aroclor-1268	1/1	460	460	µg/kg dw	na	460	not eval.	na	no	ntx, sum

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7440-38-2	Arsenic	525/575	1.8	99.3	mg/kg dw	3.1 - 31	99.3	5.03/10.4	2.7 ca	yes	asl
7440-39-3	Barium	430/430	9.4	7380	mg/kg dw	na	7380	24.0/55.5	100,000 max	no	bsl
71-43-2	Benzene	1/49	0.87	0.87	µg/kg dw	1.4 - 533	533	not eval.	1500 ca*	no	bsl
92-87-5	Benzidine	0/8	nd	nd	µg/kg dw	930 - 1500	1500	not eval.	11 ca	yes	asl
56-55-3	Benzo(a)anthracene	511/557	3.0	21,000	µg/kg dw	13 - 130	21,000	not eval.	2900 ca	no	teq
50-32-8	Benzo(a)pyrene	511/557	11	21,000	µg/kg dw	4.0 - 130	21,000	not eval.	290 ca	no	teq
205-99-2	Benzo(b)fluoranthene	510/552	14.3	18,000	µg/kg dw	4.0 - 4400	18,000	not eval.	2900 ca	no	teq
192-97-2	Benzo(e)pyrene	3/3	164	778	µg/kg dw	na	778	not eval.	na	no	ntx
191-24-2	Benzo(g,h,i)perylene	489/557	6.05	14,000	µg/kg dw	11 - 2100	14,000	not eval.	na	no	ntx
207-08-9	Benzo(k)fluoranthene	511/550	14	14,000	µg/kg dw	4.0 - 450	14,000	not eval.	29,000 ca	no	teq
56832-73-6	Benzofluoranthenes (total-calc'd)	511/550	20	32,000	µg/kg dw	4.0 - 450	32,000	not eval.	na	no	ntx
65-85-0	Benzoic acid	30/549	67.6	5930	µg/kg dw	13 - 2000	5930	not eval.	1E8 max	no	bsl
100-51-6	Benzyl alcohol	7/549	80	1700	µg/kg dw	16 - 690	1700	not eval.	1E8 max	no	bsl
7440-41-7	Beryllium	449/459	0.095	0.73	mg/kg dw	0.10 - 0.70	0.73	not eval.	2200 ca**	no	bsl
319-85-7	beta-BHC	1/100	13	13	µg/kg dw	0.40 - 56	56	not eval.	2100 ca	no	bsl
33213-65-9	beta-Endosulfan	1/56	2.85	2.85	µg/kg dw	0.63 - 200	200	not eval.	530,000 nc [l]	no	bsl
92-52-4	Biphenyl	2/2	7.5	31	µg/kg dw	na	31	not eval.	350,000 sat	no	bsl
111-91-1	bis(2-chloroethoxy)methane	1/527	40	40	µg/kg dw	19 - 2100	2100	not eval.	na	no	ntx
111-44-4	bis(2-chloroethyl)ether	0/527	nd	nd	µg/kg dw	19 - 2100	2100	not eval.	620 ca	no	ifd [f]
39638-32-9	bis(2-chloroisopropyl)ether	0/352	nd	nd	µg/kg dw	19 - 400	400	not eval.	8100 ca	no	bsl
117-81-7	bis(2-ethylhexyl)phthalate	466/561	5.42	13,000	µg/kg dw	19 - 1790	13,000	not eval.	180,000 ca	no	bsl
108-60-1	bis-chloroisopropyl ether	0/177	nd	nd	µg/kg dw	19 - 2100	2100	not eval.	35,000 ca	no	bsl
108-86-1	Bromobenzene	0/44	nd	nd	µg/kg dw	1.5 - 533	533	not eval.	9200 nc	no	bsl
74-97-5	Bromochloromethane	0/44	nd	nd	µg/kg dw	1.5 - 1060	1060	not eval.	na	no	ntx
75-27-4	Bromodichloromethane	0/49	nd	nd	µg/kg dw	1.4 - 533	533	not eval.	2400 ca	no	bsl
75-25-2	Bromoform	0/49	nd	nd	µg/kg dw	1.4 - 2660	2660	not eval.	310,000 ca*	no	bsl
74-83-9	Bromomethane	0/49	nd	nd	µg/kg dw	2.8 - 5330	5330	not eval.	1300 nc	no	ifd [c]

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85-68-7	Butyl benzyl phthalate	336/561	2.01	7100	µg/kg dw	16 - 2100	7100	not eval.	1E8 max	no	bsl
	Butyltin (total)	33/44	14	420	µg/kg dw	15 - 25	420	not eval.	na	no	ntx
7440-43-9	Cadmium	430/567	0.070	120	mg/kg dw	0.040 - 1.6	120	0.360/1.12	81 nc	no	bsl
58-08-2	Caffeine	0/16	nd	nd	µg/kg dw	7.1 - 2100	2100	not eval.	na	no	ntx
7440-70-2	Calcium	429/429	1760	48,900	mg/kg dw	na	48,900	not eval.	na	no	ntx
86-74-8	Carbazole	307/527	12	7500	µg/kg dw	9.4 - 2100	7500	not eval.	120,000 ca	no	bsl
75-15-0	Carbon disulfide	16/49	0.84	4.0	µg/kg dw	1.4 - 1060	1060	not eval.	720,000 sat	no	bsl
56-23-5	Carbon tetrachloride	0/49	nd	nd	µg/kg dw	1.4 - 533	533	not eval.	530 ca*	no	ifd [c]
	Carcinogenic PAHs (calc'd)	531/557	5.2	30,900	µg/kg dw	17 - 109	30,900	not eval.	290 ca [m]	yes	asl
57-74-9	Chlordane	5/45	25	50	µg/kg dw	8.3 - 330	330	not eval.	11,000 ca*	no	bsl
107-14-2	Chloroacetonitrile	0/2	nd	nd	µg/kg dw	7.6 - 23.8	23.8	not eval.	na	no	ntx
108-90-7	Chlorobenzene	0/49	nd	nd	µg/kg dw	1.4 - 533	533	not eval.	54,000 nc	no	bsl
75-00-3	Chloroethane	0/49	nd	nd	µg/kg dw	2.8 – 10,600	10,600	not eval.	6500 ca	no	ifd [c]
67-66-3	Chloroform	0/49	nd	nd	µg/kg dw	1.4 - 533	533	not eval.	520 ca**	no	ifd [c]
74-87-3	Chloromethane	0/49	nd	nd	µg/kg dw	1.5 - 1060	1060	not eval.	2700 ca	no	bsl
2921-88-2	Chlorpyriphos	0/3	nd	nd	µg/kg dw	10 - 46	46	not eval.	260,000 nc	no	bsl
7440-47-3	Chromium	571/571	5.0	1100	mg/kg dw	na	1100	not eval.	448 ca	yes	asl
18540-29-9	Chromium VI	1/8	11.8	11.8	mg/kg dw	1.0 - 10	11.8	not eval.	64 ca	no	bsl
218-01-9	Chrysene	529/557	20	21,000	µg/kg dw	5.4 - 120	21,000	not eval.	290,000 ca	no	teq
156-59-2	cis-1,2-Dichloroethene	0/47	nd	nd	µg/kg dw	1.4 - 533	533	not eval.	15,000 nc	no	bsl
10061-01-5	cis-1,3-Dichloropropene	0/49	nd	nd	µg/kg dw	1.4 - 564	564	not eval.	na	no	ntx
5103-73-1	cis-Nonachlor	0/3	nd	nd	µg/kg dw	0.63 - 2.9	2.9	not eval.	na	no	ntx
7440-48-4	Cobalt	372/372	3.0	140	mg/kg dw	na	140	not eval.	100,000 max	no	bsl
7440-50-8	Copper	575/575	5.0	12,000	mg/kg dw	na	12,000	21.3/50.8	7600 nc	yes	asl
360-68-9	Coprostanol	43/95	240	49,500	µg/kg dw	19 - 2100	49,500	not eval.	na	no	ntx
57-12-5	Cyanide	0/4	nd	nd	mg/kg dw	0.44 - 0.51	0.51	not eval.	1800 nc	no	bsl
99-87-6	Cymene	3/44	1.6	25	µg/kg dw	1.5 - 533	533	not eval.	na	no	ntx

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	DDTs (total-calc'd)	42/102	1.0	2880	µg/kg dw	0.81 - 51	2880	not eval.	12,000 ca [n]	no	bsl
319-86-8	delta-BHC	1/56	6.7	6.7	µg/kg dw	0.40 - 56	56	not eval.	na	no	ntx
53-70-3	Dibenzo(a,h)anthracene	330/557	2.2	7200	µg/kg dw	5.9 - 2100	7200	not eval.	290 ca	no	teq
132-64-9	Dibenzofuran	188/556	2.25	2300	µg/kg dw	1.7 - 2100	2300	not eval.	510,000 nc	no	bsl
132-65-0	Dibenzothiophene	3/3	17	59	µg/kg dw	na	59	not eval.	na	no	ntx
124-48-1	Dibromochloromethane	0/49	nd	nd	µg/kg dw	1.4 - 2660	2660	not eval.	2700 ca	no	bsl
74-95-3	Dibromomethane	0/44	nd	nd	µg/kg dw	1.5 - 1060	1060	not eval.	24,000 nc	no	bsl
1002-53-5	Dibutyltin as ion	59/86	1.0	210	µg/kg dw	1.0 - 49	210	not eval.	na	no	ntx
75-71-8	Dichlorodifluoromethane	0/8	nd	nd	µg/kg dw	1.5 - 3.3	3.3	not eval.	31,000 nc	no	bsl
75-09-2	Dichloromethane	1/49	1610	1610	µg/kg dw	2.8 - 21	1610	not eval.	21,000 ca	no	bsl
60-57-1	Dieldrin	5/100	2.6	280	µg/kg dw	0.63 - 56	280	not eval.	150 ca	yes	asl
60-29-7	Diethyl ether	0/44	nd	nd	µg/kg dw	1.5 - 1060	1060	not eval.	1,800,000 sat	no	bsl
84-66-2	Diethyl phthalate	8/561	21	140	µg/kg dw	1.79 - 2100	2100	not eval.	1E8 max	no	bsl
131-11-3	Dimethyl phthalate	109/561	19	200	µg/kg dw	1.79 - 2100	2100	not eval.	1E8 max	no	bsl
84-74-2	Di-n-butyl phthalate	183/561	13	3800	µg/kg dw	1.79 - 2100	3800	not eval.	8,800,000 nc	no	bsl
117-84-0	Di-n-octyl phthalate	43/561	1.84	570	µg/kg dw	1.79 - 2100	2100	not eval.	1E7 sat	no	bsl
115-29-7	Endosulfan	0/45	nd	nd	µg/kg dw	1.6 - 56	56	not eval.	530,000 nc	no	bsl
1031-07-8	Endosulfan sulfate	1/100	6.1	6.1	µg/kg dw	0.81 - 200	200	not eval.	na	no	ntx
72-20-8	Endrin	0/100	nd	nd	µg/kg dw	0.63 - 200	200	not eval.	26,000 nc	no	bsl
7421-93-4	Endrin aldehyde	3/89	4.6	130	µg/kg dw	0.63 - 56	130	not eval.	na	no	ntx
53494-70-5	Endrin ketone	1/56	2.8	2.8	µg/kg dw	0.81 - 200	200	not eval.	na	no	ntx
97-63-2	Ethyl Methacrylate	0/44	nd	nd	µg/kg dw	1.5 - 1060	1060	not eval.	140,000 sat	no	bsl
100-41-4	Ethylbenzene	1/49	0.49	0.49	µg/kg dw	1.4 - 533	533	not eval.	230,000 sat	no	bsl
206-44-0	Fluoranthene	540/557	20	62,000	µg/kg dw	11 - 1400	62,000	not eval.	3,000,000 nc	no	bsl
86-73-7	Fluorene	299/557	2.01	4400	µg/kg dw	1.79 - 2000	4400	not eval.	3,300,000 nc	no	bsl
58-89-9	gamma-BHC	3/100	4.9	8.6	µg/kg dw	0.40 - 56	56	not eval.	2900 ca	no	bsl
5103-74-2	gamma-Chlordane	3/56	1.0	204	µg/kg dw	0.81 - 37	204	not eval.	11,000 ca* [k]	no	bsl

CAS Number	Chemical	Detection frequency (location)	Min. Detected Conc.	Max. detected Conc.	Units	Range of detection limits	Conc. Used for Screening	Background conc. [a]	RBC [b]	COPC Flag	Rationale for COPC Selection or Exclusion
8006-61-9	Gasoline	0/8	nd	nd	mg/kg dw	10 - 10	10	not eval.	na	no	ntx
76-44-8	Heptachlor	4/100	1.1	2.8	µg/kg dw	0.40 - 56	56	not eval.	550 ca	no	bsl
1024-57-3	Heptachlor epoxide	2/100	1.0	2.0	µg/kg dw	0.40 - 100	100	not eval.	270 ca*	no	bsl
118-74-1	Hexachlorobenzene	41/557	0.40	690	µg/kg dw	0.11 - 2100	2100	not eval.	1500 ca	no	ifd [g]
87-68-3	Hexachlorobutadiene	0/557	nd	nd	µg/kg dw	1.1 - 2100	2100	not eval.	32,000 ca**	no	bsl
77-47-4	Hexachlorocyclopentadiene	1/475	100	100	µg/kg dw	32 - 2100	2100	not eval.	590,000 nc	no	bsl
67-72-1	Hexachloroethane	0/546	nd	nd	µg/kg dw	1.5 - 2100	2100	not eval.	180,000 ca**	no	bsl
193-39-5	Indeno(1,2,3-cd)pyrene	492/557	7.7	15,000	µg/kg dw	12 - 2100	15,000	not eval.	2900 ca	no	teq
74-88-4	Iodomethane	0/44	nd	nd	µg/kg dw	1.5 - 1060	1060	not eval.	na	no	ntx
7439-89-6	Iron	448/448	8100	160,000	mg/kg dw	na	160,000	17,500/28,700	100,000 max	yes	asl
78-59-1	Isophorone	0/527	nd	nd	µg/kg dw	19 - 2100	2100	not eval.	2,600,000 ca*	no	bsl
98-82-8	iso-Propylbenzene	0/44	nd	nd	µg/kg dw	1.8 - 533	533	not eval.	52,000 nc	no	bsl
7439-92-1	Lead	575/575	2.0	23,000	mg/kg dw	na	23,000	15/45	100 nc	yes	asl
	Lube Oils	0/8	nd	nd	mg/kg dw	10 - 10	10	not eval.	na	no	ntx
7439-95-4	Magnesium	439/439	2000	17,200	mg/kg dw	na	17,200	not eval.	na	no	ntx
7439-96-5	Manganese	445/445	78	3300	mg/kg dw	na	3300	279/1010	3200 nc	yes	asl
7439-97-6	Mercury	501/572	0.020	4.6	mg/kg dw	0.020 - 0.22	4.6	0.0981/0.327	8.8 nc [o]	no	bsl
126-98-7	Methacrylonitrile	0/44	nd	nd	µg/kg dw	3.7 - 1060	1060	not eval.	880 nc	no	ifd [d]
72-43-5	Methoxychlor	6/100	2.0	99	µg/kg dw	1.0 - 330	330	not eval.	440,000 nc	no	bsl
96-33-3	Methyl Acrylate	0/44	nd	nd	µg/kg dw	2.3 - 533	533	not eval.	23,000 nc	no	bsl
78-93-3	Methyl ethyl ketone	17/49	5.3	34.5	µg/kg dw	3.0 - 1060	1060	not eval.	2,800,000 nc	no	bsl
108-10-1	Methyl iso-butyl ketone	0/49	nd	nd	µg/kg dw	3.0 - 1060	1060	not eval.	290,000 nc	no	bsl
80-62-6	Methyl Methacrylate	0/44	nd	nd	µg/kg dw	1.8 - 533	533	not eval.	2,700,000 sat	no	bsl
22967-92-6	Methylmercury	19/19	0.11	3.4	µg/kg dw	na	3.4	not eval.	8800 nc	no	bsl
2385-85-5	Mirex	0/3	nd	nd	µg/kg dw	0.63 - 2.9	2.9	not eval.	1400 ca	no	bsl
7439-98-7	Molybdenum	8/65	2.1	9.6	mg/kg dw	1.2 - 4.5	9.6	not eval.	1000 nc	no	bsl

CAS Number	Chemical	Detection frequency (location)	Min. Detected Conc.	Max. detected Conc.	Units	Range of detection limits	Conc. Used for Screening	Background conc. [a]	RBC [b]	COPC Flag	Rationale for COPC Selection or Exclusion
78763-54-9	Monobutyltin as ion	5/12	15	26.3	µg/kg dw	11 - 13	26.3	not eval.	na	no	ntx
91-20-3	Naphthalene	91/557	4.3	2100	µg/kg dw	1.5 - 2100	2100	not eval.	19,000 nc	no	bsl
104-51-8	n-Butylbenzene	0/44	nd	nd	µg/kg dw	1.5 - 533	533	not eval.	240,000 sat	no	bsl
2406-65-7	n-Butyltin	54/80	1.0	96	µg/kg dw	1.0 - 85	96	not eval.	na	no	ntx
7440-02-0	Nickel	563/565	5.0	910	mg/kg dw	29 - 32	910	26.8/41.7	4100 nc	no	bsl
98-95-3	Nitrobenzene	0/527	nd	nd	µg/kg dw	19 - 2100	2100	not eval.	11,000 nc	no	bsl
62-75-9	N-Nitrosodimethylamine	0/87	nd	nd	µg/kg dw	27 - 250	250	not eval.	48 ca	yes	asl
621-64-7	N-Nitroso-di-n-propylamine	0/527	nd	nd	µg/kg dw	12 - 3900	3900	not eval.	350 ca	no	ifd [h]
86-30-6	N-Nitrosodiphenylamine	8/557	41	190	µg/kg dw	1.79 - 2100	2100	not eval.	500,000 ca	no	bsl
103-65-1	n-Propylbenzene	0/44	nd	nd	µg/kg dw	1.5 - 533	533	not eval.	240,000 sat	no	bsl
3268-87-9	OCDD	29/29	7.8	91,000	ng/kg dw	na	91,000	not eval.	na	no	teq
39001-02-0	OCDF	28/29	22	3600	ng/kg dw	0.74 - 0.74	3600	not eval.	na	no	teq
27304138	Oxychlordane	0/3	nd	nd	µg/kg dw	0.63 - 2.9	2.9	not eval.	na	no	ntx
37680-73-2	PCB-101	524/581	0.41	5600	µg/kg dw	0.12 - 10	5600	not eval.	na	no	teq
32598-14-4	PCB-105	415/578	0.25	560	µg/kg dw	0.12 - 19	560	not eval.	na	no	sum
38380-03-9	PCB-110	269/304	0.22	3000	µg/kg dw	0.12 - 6.6	3000	not eval.	na	no	sum
74472-37-0	PCB-114	6/276	1.0	5.0	µg/kg dw	1.0 - 20	20	not eval.	na	no	sum
31508-00-6	PCB-118	479/582	0.42	2200	µg/kg dw	0.12 - 8.3	2200	not eval.	na	no	sum
65510-44-3	PCB-123	0/276	nd	nd	µg/kg dw	1.0 - 31	31	not eval.	na	no	sum
57465-28-8	PCB-126	11/582	0.65	3.0	µg/kg dw	0.10 - 50	50	not eval.	na	no	sum
38380-07-3	PCB-128	324/578	0.35	620	µg/kg dw	0.13 - 13	620	not eval.	na	no	sum
35065-28-2	PCB-138	514/583	0.21	1400	µg/kg dw	0.13 - 40	1400	not eval.	na	no	sum
35065-27-1	PCB-153	529/580	0.48	3000	µg/kg dw	0.12 - 11	3000	not eval.	na	no	sum
38380-08-4	PCB-156	231/580	0.33	160	µg/kg dw	0.080 – 3.0	160	not eval.	na	no	sum
69782-90-7	PCB-157	71/578	0.31	56	µg/kg dw	0.080 – 27	56	not eval.	na	no	sum
52663-72-6	PCB-167	44/276	1.0	30	µg/kg dw	1.0 – 2.0	30	not eval.	na	no	sum
32774-16-6	PCB-169	0/580	nd	nd	µg/kg dw	0.25 – 10	10	not eval.	na	no	sum

CAS Number	Chemical	Detection frequency (location)	Min. Detected Conc.	Max. detected Conc.	Units	Range of detection limits	Conc. Used for Screening	Background conc. [a]	RBC [b]	COPC Flag	Rationale for COPC Selection or Exclusion
35065-30-6	PCB-170	448/583	0.19	560	µg/kg dw	0.080 - 14	560	not eval.	na	no	sum
37680-65-2	PCB-18	85/264	1.0	170	µg/kg dw	0.81 - 24	170	not eval.	na	no	sum
35065-29-3	PCB-180	482/583	0.19	965	µg/kg dw	0.11 - 9.5	965	not eval.	na	no	sum
52663-68-0	PCB-187	235/279	1.0	360	µg/kg dw	1.0 – 6.0	360	not eval.	na	no	sum
39635-31-9	PCB-189	29/580	0.78	11.5	µg/kg dw	0.11 – 5.0	11.5	not eval.	na	no	sum
52663-78-2	PCB-195	41/279	0.76	49	µg/kg dw	1.0 – 2.0	49	not eval.	na	no	sum
40186-72-9	PCB-206	52/279	0.58	27	µg/kg dw	1.0 – 1.0	27	not eval.	na	no	sum
2051-24-3	PCB-209	15/279	0.40	3.0	µg/kg dw	1.0 – 1.0	3.0	not eval.	na	no	sum
7012-37-5	PCB-28	155/279	1.0	160	µg/kg dw	0.81 – 8.0	160	not eval.	na	no	sum
41464-39-5	PCB-44	190/279	1.0	190	µg/kg dw	1.0 – 2.0	190	not eval.	na	no	sum
35693-99-3	PCB-52	3/3	4.4	22	µg/kg dw	na	22	not eval.	na	no	ntx
35693-99-3	PCB-55	204/276	1.0	890	µg/kg dw	1.0 - 13	890	not eval.	na	no	ntx
32598-10-0	PCB-66	188/279	1.0	440	µg/kg dw	1.0 - 300	440	not eval.	na	no	ntx
32598-13-3	PCB-77	20/583	0.70	26	µg/kg dw	0.11 - 35	35	not eval.	na	no	ntx
34883-43-7	PCB-8	1/3	1.7	1.7	µg/kg dw	0.81 - 2.9	2.9	not eval.	na	no	ntx
70362-50-4	PCB-81	0/276	nd	nd	µg/kg dw	1.0 - 10	10	not eval.	na	no	ntx
	PCBs (total-calc'd)	905/957	1.6	222,600	µg/kg dw	0.56 - 50	222,600	not eval.	1000 ca [p]	yes	asl
	PCBs + PCTs (total)	301/304	1.6	26,000	µg/kg dw	0.56 - 0.63	26,000	not eval.	na	no	ntx
	PCTs (total)	265/306	1.8	5600	µg/kg dw	1.6 - 8.1	5600	not eval.	na	no	ntx
76-01-7	Pentachloroethane	0/44	nd	nd	µg/kg dw	1.5 - 1060	1060	not eval.	na	no	ntx
87-86-5	Pentachlorophenol	5/506	100	527	µg/kg dw	6.7 - 5200	5200	not eval.	11,000 ca	no	bsl
198-55-0	Perylene	3/3	116	949	µg/kg dw	na	949	not eval.	na	no	ntx
85-01-8	Phenanthrene	520/557	7.05	43,000	µg/kg dw	5.4 - 850	43,000	not eval.	na	no	ntx
108-95-2	Phenol	197/557	15	3600	µg/kg dw	12 - 2000	3600	not eval.	1E8 max	no	bsl
104-40-5	Phenol, 4-Nonyl-	0/3	nd	nd	µg/kg dw	5.9 - 26	26	not eval.	na	no	ntx
7440-09-7	Potassium	439/439	380	11,100	mg/kg dw	na	11100	not eval.	na	no	ntx
129-00-0	Pyrene	531/557	5.9	48,000	µg/kg dw	5.4 - 5400	48,000	not eval.	5,400,000 nc	no	bsl

CAS Number	Chemical	Detection frequency (location)	Min. Detected Conc.	Max. detected Conc.	Units	Range of detection limits	Conc. Used for Screening	Background conc. [a]	RBC [b]	COPC Flag	Rationale for COPC Selection or Exclusion
483-65-8	Retene	3/11	33	267	µg/kg dw	290 - 2100	2100	not eval.	na	no	ntx
135-98-8	sec-Butylbenzene	0/44	nd	nd	µg/kg dw	1.5 - 533	533	not eval.	220,000 sat	no	bsl
7782-49-2	Selenium	269/454	0.40	28	mg/kg dw	0.30 - 34	34	not eval.	1000 nc	no	bsl
7440-21-3	Silicon	3/3	251,000	271,000	mg/kg dw	na	271,000	not eval.	na	no	ntx
7440-22-4	Silver	408/567	0.040	270	mg/kg dw	0.20 - 3.3	270	0.28/0.74	1000 nc	no	bsl
7440-23-5	Sodium	431/431	580	22,700	mg/kg dw	na	22700	not eval.	na	no	ntx
100-42-5	Styrene	0/49	nd	nd	µg/kg dw	1.4 - 1060	1060	not eval.	1,700,000 sat	no	bsl
	Sulfides (total)	42/76	2.0	2300	mg/kg dw	0.68 – 3.7	2300	not eval.	na	no	ntx
1634-04-4	Tert-butyl methyl ether	0/44	nd	nd	µg/kg dw	1.5 – 533	533	not eval.	na	no	ntx
98-06-6	tert-Butylbenzene	0/44	nd	nd	µg/kg dw	1.5 – 533	533	not eval.	390,000 sat	no	bsl
1461-25-2	Tetrabutyltin as ion	7/92	1.5	7.0	µg/kg dw	0.60 - 20	20	not eval.	na	no	ntx
127-18-4	Tetrachloroethene	2/49	0.21	0.52	µg/kg dw	1.4 - 533	533	not eval.	19,000 ca*	no	bsl
109-99-9	Tetrahydrofuran	0/2	nd	nd	µg/kg dw	7.6 - 7.8	7.8	not eval.	320,000 ca	no	bsl
7440-28-0	Thallium	302/458	0.010	30	mg/kg dw	0.030 - 45	45	0.252/1.79	16 nc	yes	asl
7440-31-5	Tin	185/279	1.0	466	mg/kg dw	1.0 – 9.0	466	not eval.	100,000 max	no	bsl
7440-32-6	Titanium	3/3	650	985	mg/kg dw	na	985	not eval.	na	no	ntx
108-88-3	Toluene	5/49	0.34	6.4	µg/kg dw	1.4 – 533	533	not eval.	520,000 sat	no	bsl
	Total HPAH (calc'd)	544/557	3.0	241,000	µg/kg dw	20 – 78	241,200	not eval.	na	no	ntx
	Total LPAH (calc'd)	522/557	9.1	60,200	µg/kg dw	20 – 130	60,230	not eval.	na	no	ntx
8001-35-2	Toxaphene	0/100	nd	nd	µg/kg dw	1.0 - 3700	3700	not eval.	2200 ca	no	ifd [i]
	TPH	50/56	23	23,000	mg/kg dw	20 - 20	23000	not eval.	na	no	ntx
68334-30-5	TPH - Diesel #2 Range	0/8	nd	nd	mg/kg dw	10 - 10	10	not eval.	na	no	ntx
	TPH - Diesel Range	2/2	105.5	164	mg/kg dw	na	164	not eval.	na	no	ntx
	TPH - Gasoline Range	0/2	nd	nd	mg/kg dw	20 - 20	20	not eval.	na	no	ntx
	TPH - Heavy Fuel Oil Range	2/2	250	370	mg/kg dw	na	370	not eval.	na	no	ntx
156-60-5	trans-1,2-Dichloroethene	0/47	nd	nd	µg/kg dw	1.4 - 533	533	not eval.	21,000 nc	no	bsl
10061-02-6	trans-1,3-Dichloropropene	0/49	nd	nd	µg/kg dw	1.4 - 501	501	not eval.	na	no	ntx

CAS Number	Chemical	Detection frequency (location)	Min. Detected Conc.	Max. detected Conc.	Units	Range of detection limits	Conc. Used for Screening	Background conc. [a]	RBC [b]	COPC Flag	Rationale for COPC Selection or Exclusion
110-57-6	trans-1,4-Dichloro-2-butene	0/42	nd	nd	µg/kg dw	7.6 - 2660	2660	not eval.	na	no	ntx
39765-80-5	Trans-Nonachlor	0/3	nd	nd	µg/kg dw	0.81 - 2.9	2.9	not eval.	na	no	ntx
688-73-3	Tributyltin as ion	88/94	1.0	358	µg/kg dw	1.0 - 1.0	358	not eval.	13,000 nc [q]	no	bsl
79-01-6	Trichloroethene	0/49	nd	nd	µg/kg dw	1.4 - 533	533	not eval.	6100 ca*	no	bsl
75-69-4	Trichlorofluoromethane	0/47	nd	nd	µg/kg dw	1.5 - 5330	5330	not eval.	2,000,000 sat	no	bsl
7440-62-2	Vanadium	372/372	15	150	mg/kg dw	na	150	36.0/59.6	1400 nc	no	bsl
108-05-4	Vinyl acetate	0/3	nd	nd	µg/kg dw	7.0 – 12	12	not eval.	140,000 nc	no	bsl
75-01-4	Vinyl chloride	0/49	nd	nd	µg/kg dw	1.5 - 2660	2660	not eval.	49 ca	no	ifd [c]
108-38-3/106-42-3	Xylene (meta & para)	1/47	1.4	1.4	µg/kg dw	1.4 - 1060	1060	not eval.	210,000 sat [r]	no	ntx
95-47-6	Xylene (ortho)	1/47	1.1	1.1	µg/kg dw	1.4 - 533	533	not eval.	210,000 sat [r]	no	ntx
1330-20-7	Xylene (total)	0/2	nd	nd	µg/kg dw	23 – 24	24	not eval.	210,000 sat	no	bsl
7440-66-6	Zinc	573/575	16	9700	mg/kg dw	128 - 340	9700	52.6/98.5	100,000 max	no	bsl

[a] Background concentrations obtained from joint Ecology/PSAMP 1998 study entitled “Sediment Quality in Puget Sound. Year 2- Central Puget Sound” (Ecology 2000). Reported concentrations are mean and maximum from 52 samples collected from the following strata: South Port Townsend, Port Townsend, North Admiralty Inlet, South Admiralty Inlet, Possession Sound, Central Basin, Port Madison, West Point, East Passage, Liberty Bay, Keyport, Northwest Bainbridge Island, Southwest Bainbridge Island, Rich Passage, Port Orchard, and Port Washington Narrows

[b] Risk-based concentrations (RBCs) are derived from EPA Region 9 Preliminary Remediation Goals (PRGs) for industrial soil (last updated October 1999). PRGs associated with a non-cancer endpoint (abbreviated “nc”) were divided by 10 for this screening, reflecting the different target hazard quotients used in Region 9 (HQ = 1) and Region 10 (HQ = 0.1). All other PRGs were not modified for this screening. Abbreviations: ca = cancer endpoint, nc = non-cancer endpoint, sat = soil saturation, m = ceiling limit, * = nc < 100X ca, ** = nc < 10X ca

[c] 48 of 49 detection limits were less than RBC

[d] 43 of 44 detection limits were less than RBC

[e] 524 of 525 detection limits were less than RBC

[f] 520 of 527 detection limits were less than RBC

[g] 552 of 557 concentrations (all 41 detections) were less than RBC

[h] 515 of 527 detection limits were less than RBC

[i] 99 of 100 detection limits were less than RBC

[j] RBC is for 2,3,7,8-TCDD

[k] RBC is for chlordane

[l] RBC is for endosulfan

[m] RBC is for benzo(a)pyrene

[n] RBC is for 4,4'-DDT

[o] RBC is for methylmercury

[p] RBC is for Aroclor 1254

[q] RBC for tributyltin oxide multiplied by 0.49 to account for differences in molecular weight

[r] RBC is for total xylenes

Other abbreviations: nd = not detected, na = not applicable; HPAH = high-molecular-weight polycyclic aromatic hydrocarbon; LPAH = low-molecular-weight polycyclic aromatic hydrocarbon

Rationale codes	Selection reason:	above screening level (asl)
	Exclusion reason:	infrequent detection (ifd)
		no toxicity information (ntx)
		below screening level (bsl)
		chemical included in sum and is not evaluated separately (sum)
		chemical included in TEQ calculation and is not evaluated separately (teq)

Table 2. Occurrence, distribution, and selection of chemicals of potential concern for sediment in the beach play exposure scenario

CAS Number	Chemical	Min. Detected Conc.	Max. detected Conc.	Units	Detection frequency (location)	Range of detection limits	Conc. Used for Screening	Background conc.[a]	RBC[b]	COPC Flag	Rationale for Selection or Exclusion
630-20-6	1,1,1,2-Tetrachloroethane	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	3000 ca	no	bsl
71-55-6	1,1,1-Trichloroethane	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	77,000 nc	no	bsl
79-34-5	1,1,2,2-Tetrachloroethane	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	380 ca	no	bsl
79-00-5	1,1,2-Trichloroethane	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	840 ca*	no	bsl
76-13-1	1,1,2-Trichlorotrifluoroethane	nd	nd	µg/kg dw	0/14	2.3 - 73.8	73.8	not eval.	5,600,000 sat	no	bsl
513-88-2	1,1-Dichloroacetone	nd	nd	µg/kg dw	0/10	4.6 - 36.9	36.9	not eval.	na	no	ntx
75-34-3	1,1-Dichloroethane	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	59,000 nc	no	bsl
75-35-4	1,1-Dichloroethene	nd	nd	µg/kg dw	0/14	1.4 - 7.4	7.4	not eval.	54 ca	no	bsl
563-58-6	1,1-Dichloropropene	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	na	no	ntx
35822-46-9	1,2,3,4,6,7,8-HpCDD	48	5200	ng/kg dw	8/8	na	5200	not eval.	na	no	teq
67562-39-4	1,2,3,4,6,7,8-HpCDF	8.3	1600	ng/kg dw	7/8	7.7 - 7.7	1600	not eval.	na	no	teq
55673-89-7	1,2,3,4,7,8,9-HpCDF	5.2	270	ng/kg dw	4/8	0.77 - 2.8	270	not eval.	na	no	teq
39227-28-6	1,2,3,4,7,8-HxCDD	27	27	ng/kg dw	1/8	0.86 - 4.4	27	not eval.	na	no	teq
70648-26-9	1,2,3,4,7,8-HxCDF	9.0	540	ng/kg dw	4/8	1.2 - 3.6	540	not eval.	na	no	teq
57653-85-7	1,2,3,6,7,8-HxCDD	8.6	200	ng/kg dw	5/8	2.4 - 4.3	200	not eval.	na	no	teq
57117-44-9	1,2,3,6,7,8-HxCDF	74	74	ng/kg dw	1/8	0.51 - 4.3	74	not eval.	na	no	teq
19408-74-3	1,2,3,7,8,9-HxCDD	6.9	65	ng/kg dw	5/8	2.1 - 3.3	65	not eval.	78 ca	no	teq
72918-21-9	1,2,3,7,8,9-HxCDF	16	16	ng/kg dw	1/8	0.15 - 2.4	16	not eval.	na	no	teq
40321-76-4	1,2,3,7,8-PeCDD	12	12	ng/kg dw	1/8	0.53 - 4.1	12	not eval.	na	no	teq
57117-41-6	1,2,3,7,8-PeCDF	54	54	ng/kg dw	1/8	0.28 - 2.8	54	not eval.	na	no	teq
87-61-6	1,2,3-Trichlorobenzene	nd	nd	µg/kg dw	0/11	3.7 - 6.6	6.6	not eval.	na	no	ntx
96-18-4	1,2,3-Trichloropropane	nd	nd	µg/kg dw	0/11	2.3 - 18.4	18.4	not eval.	1.4 ca	yes	asl
120-82-1	1,2,4-Trichlorobenzene	nd	nd	µg/kg dw	0/203	0.35 - 140	140	not eval.	65,000 nc	no	bsl
95-63-6	1,2,4-Trimethylbenzene	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	5700 sat	no	bsl

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96-12-8	1,2-Dibromo-3-chloropropane	nd	nd	µg/kg dw	0/11	7.4 - 16.6	16.6	not eval.	450 ca**	no	bsl
106-93-4	1,2-Dibromoethane (EDB)	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	6.9 ca	no	bsl
95-50-1	1,2-Dichlorobenzene	22	22	µg/kg dw	1/203	0.35 - 140	140	not eval.	370,000 sat	no	bsl
107-06-2	1,2-Dichloroethane	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	350 ca*	no	bsl
78-87-5	1,2-Dichloropropane	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	350 ca*	no	bsl
122-66-7	1,2-Diphenylhydrazine	nd	nd	µg/kg dw	0/24	13 - 120	120	not eval.	610 ca	no	bsl
108-67-8	1,3,5-Trimethylbenzene	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	2100 nc	no	bsl
541-73-1	1,3-Dichlorobenzene	nd	nd	µg/kg dw	0/197	0.35 - 140	140	not eval.	1300 nc	no	bsl
142-28-9	1,3-Dichloropropane	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	na	no	ntx
106-46-7	1,4-Dichlorobenzene	1.8	1300	µg/kg dw	16/203	0.18 - 140	1300	not eval.	3400 ca	no	bsl
109-69-3	1-Chlorobutane	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	480,000 sat	no	bsl
594-20-7	2,2-Dichloropropane	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	na	no	ntx
60851-34-5	2,3,4,6,7,8-HxCDF	32	32	ng/kg dw	1/8	0.44 - 2.5	32	not eval.	na	no	teq
57117-31-4	2,3,4,7,8-PeCDF	58	58	ng/kg dw	1/8	0.44 - 5.4	58	not eval.	na	no	teq
1746-01-6	2,3,7,8-TCDD	2.0	3.8	ng/kg dw	2/8	0.37 - 1.1	3.8	not eval.	3.9 ca	no	teq
	2,3,7,8-TCDD TEQ	1.7	224	ng/kg dw	8/8	na	224	not eval.	3.9 ca	yes	asl
51207-31-9	2,3,7,8-TCDF	0.99	6.8	ng/kg dw	6/8	0.81 - 0.91	6.8	not eval.	na	no	teq
95-95-4	2,4,5-Trichlorophenol	nd	nd	µg/kg dw	0/186	16 - 2000	2000	not eval.	610,000 nc	no	bsl
88-06-2	2,4,6-Trichlorophenol	nd	nd	µg/kg dw	0/186	18 - 2000	2000	not eval.	44,000 ca	no	bsl
120-83-2	2,4-Dichlorophenol	nd	nd	µg/kg dw	0/186	22 - 1200	1200	not eval.	18,000 nc	no	bsl
105-67-9	2,4-Dimethylphenol	290	290	µg/kg dw	1/201	9.4 - 520	520	not eval.	120,000 nc	no	bsl
51-28-5	2,4-Dinitrophenol	nd	nd	µg/kg dw	0/184	20 - 1400	1400	not eval.	12,000 nc	no	bsl
121-14-2	2,4-Dinitrotoluene	nd	nd	µg/kg dw	0/186	4.0 – 690	690	not eval.	12,000 nc	no	bsl
606-20-2	2,6-Dinitrotoluene	nd	nd	µg/kg dw	0/186	13 – 690	690	not eval.	6100 nc	no	bsl
110-75-8	2-Chloroethyl vinyl ether	nd	nd	µg/kg dw	0/3	7.0 – 12	12	not eval.	na	no	ntx
91-58-7	2-Chloronaphthalene	nd	nd	µg/kg dw	0/186	19 – 140	140	not eval.	490,000 nc	no	bsl
95-57-8	2-Chlorophenol	nd	nd	µg/kg dw	0/186	11 – 140	140	not eval.	6300 nc	no	bsl

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95-49-8	2-Chlorotoluene	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	16,000 nc	no	bsl
591-78-6	2-Hexanone	nd	nd	µg/kg dw	0/14	4.6 - 12	12	not eval.	na	no	ntx
91-57-6	2-Methylnaphthalene	2.69	250	µg/kg dw	20/203	1.8 - 140	250	not eval.	na	no	ntx
95-48-7	2-Methylphenol	20	20	µg/kg dw	1/203	13 - 520	520	not eval.	310,000 nc	no	bsl
88-74-4	2-Nitroaniline	nd	nd	µg/kg dw	0/184	94 - 2000	2000	not eval.	350 nc	yes	asl
88-75-5	2-Nitrophenol	nd	nd	µg/kg dw	0/186	20 - 2000	2000	not eval.	na	no	ntx
79-46-9	2-Nitropropane	nd	nd	µg/kg dw	0/11	11.5 - 18.4	18.4	not eval.	na	no	ntx
91-94-1	3,3'-Dichlorobenzidine	nd	nd	µg/kg dw	0/179	33 - 690	690	not eval.	1100 ca	no	bsl
	3-Methylphenol and 4-Methylphenol Coelution	20	100	µg/kg dw	4/50	20 - 20	100	not eval.	na	no	ntx
99-09-2	3-Nitroaniline	nd	nd	µg/kg dw	0/183	110 - 820	820	not eval.	na	no	ntx
72-54-8	4,4'-DDD	6.0	840	µg/kg dw	9/26	1.6 - 10	840	not eval.	2400 ca	no	sum
72-55-9	4,4'-DDE	3.5	370	µg/kg dw	6/26	1.0 – 5.0	370	not eval.	1700 ca	no	sum
50-29-3	4,4'-DDT	12.5	1670	µg/kg dw	3/26	1.6 – 20	1670	not eval.	1700 ca*	no	sum
534-52-1	4,6-Dinitro-o-cresol	nd	nd	µg/kg dw	0/184	65 - 1400	1400	not eval.	na	no	ntx
101-55-3	4-Bromophenyl phenyl ether	nd	nd	µg/kg dw	0/186	12 – 140	140	not eval.	na	no	ntx
59-50-7	4-Chloro-3-methylphenol	nd	nd	µg/kg dw	0/184	38 – 270	270	not eval.	na	no	ntx
106-47-8	4-Chloroaniline	nd	nd	µg/kg dw	0/176	56 – 410	410	not eval.	24,000 nc	no	bsl
7005-72-3	4-Chlorophenyl phenyl ether	nd	nd	µg/kg dw	0/186	18 – 140	140	not eval.	na	no	ntx
106-43-4	4-Chlorotoluene	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	na	no	ntx
106-44-5	4-Methylphenol	21	444	µg/kg dw	14/153	17 – 140	444	not eval.	31,000 nc	no	bsl
100-01-6	4-Nitroaniline	nd	nd	µg/kg dw	0/180	94 – 690	690	not eval.	na	no	ntx
100-02-7	4-Nitrophenol	nd	nd	µg/kg dw	0/184	65 – 690	690	not eval.	49,000 nc	no	bsl
83-32-9	Acenaphthene	1.92	760	µg/kg dw	54/203	1.79 - 140	760	not eval.	370,000 nc	no	bsl
208-96-8	Acenaphthylene	20	110	µg/kg dw	11/203	1.8 – 140	140	not eval.	na	no	ntx
67-64-1	Acetone	114	1020	µg/kg dw	3/14	23 – 148	1020	not eval.	160,000 nc	no	bsl
	Acid volatile sulfides	97	6100	mg/kg dw	8/12	50 – 55	6100	not eval.	na	no	ntx

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309-00-2	Aldrin	nd	nd	µg/kg dw	0/24	1.0 - 10	10	not eval.	29 ca*	no	bsl
107-05-1	Allyl Chloride	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	300,000 nc	no	bsl
319-84-6	alpha-BHC	nd	nd	µg/kg dw	0/24	1.0 - 10	10	not eval.	90 ca	no	bsl
5103-71-9	alpha-Chlordane	26	26	µg/kg dw	1/11	1.0 - 10	26	not eval.	1600 ca* [e]	no	bsl
959-98-8	alpha-Endosulfan	2.0	2.0	µg/kg dw	1/12	1.0 - 100	100	not eval.	37,000 nc [f]	no	bsl
7429-90-5	Aluminum	3800	110,000	mg/kg dw	151/151	na	110,000	12,000/21,000	7600 nc	yes	asl
7664-41-7	Ammonia	5.58	5.58	mg/kg dw	1/1	na	5.58	not eval.	na	no	ntx
62-53-3	Aniline	nd	nd	µg/kg dw	0/14	65 - 89	89	not eval.	85,000 ca**	no	bsl
120-12-7	Anthracene	2.01	1200	µg/kg dw	97/203	5.4 - 130	1200	not eval.	2,200,000 nc	no	bsl
7440-36-0	Antimony	2.55	110	mg/kg dw	30/89	1.8 - 28	110	0.23/0.44	3.1 nc	yes	asl
12674-11-2	Aroclor-1016	nd	nd	µg/kg dw	0/258	0.87 - 2000	2000	not eval.	390 nc	no	sum
11104-28-2	Aroclor-1221	nd	nd	µg/kg dw	0/145	1.89 - 1600	1600	not eval.	220 ca	no	sum
11141-16-5	Aroclor-1232	nd	nd	µg/kg dw	0/145	0.87 - 1600	1600	not eval.	220 ca	no	sum
53469-21-9	Aroclor-1242	7.8	2400	µg/kg dw	20/258	0.87 - 6100	6100	not eval.	220 ca	no	sum
12672-29-6	Aroclor-1248	8.3	219,000	µg/kg dw	45/258	0.87 - 2300	219,000	not eval.	220 ca	no	sum
11097-69-1	Aroclor-1254	2.17	14,000	µg/kg dw	175/260	1.31 - 4300	14,000	not eval.	220 ca**	no	sum
11096-82-5	Aroclor-1260	1.24	26,000	µg/kg dw	213/259	5.6 - 940	26,000	not eval.	220 ca	no	sum
37324-23-5	Aroclor-1262	270	840	µg/kg dw	2/2	na	840	not eval.	na	no	sum
11100-14-4	Aroclor-1268	460	460	µg/kg dw	1/1	na	460	not eval.	na	no	sum
7440-38-2	Arsenic	1.9	79.4	mg/kg dw	170/211	3.1 - 28	79.4	5.03/10.4	0.39 ca*	yes	asl
7440-39-3	Barium	9.4	1800	mg/kg dw	142/142	na	1800	24.0/55.5	540 nc	yes	asl
71-43-2	Benzene	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval	670 ca*	no	bsl
92-87-5	Benzidine	nd	nd	µg/kg dw	0/5	790 - 1500	1500	not eval.	2.1 ca	yes	asl
56-55-3	Benzo(a)anthracene	3.0	5000	µg/kg dw	173/203	20 - 130	5000	not eval.	620 ca	no	teq
50-32-8	Benzo(a)pyrene	11	5700	µg/kg dw	173/203	4.0 - 130	5700	not eval.	620 ca	no	teq
205-99-2	Benzo(b)fluoranthene	17.3	5700	µg/kg dw	177/199	4.0 - 120	5700	not eval.	620 ca	no	teq
191-24-2	Benzo(g,h,i)perylene	6.05	3900	µg/kg dw	162/203	11 - 130	3900	not eval.	na	no	ntx

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207-08-9	Benzo(k)fluoranthene	14	5500	µg/kg dw	170/199	4.0 - 120	5500	not eval.	6200 ca	no	teq
56832-73-6	Benzofluoranthenes (total-calc'd)	16.4	11,200	µg/kg dw	184/209	4.0 - 120	11,200	not eval.	na	no	ntx
65-85-0	Benzoic acid	67.6	550	µg/kg dw	13/203	13 - 1400	1400	not eval.	1E8 max	no	bsl
100-51-6	Benzyl alcohol	27	130	µg/kg dw	4/203	8.0 - 690	690	not eval.	1,800,000 nc	no	bsl
7440-41-7	Beryllium	0.10	0.60	mg/kg dw	144/153	0.10 - 0.70	0.70	not eval.	15 nc	no	bsl
319-85-7	beta-BHC	13	13	µg/kg dw	1/24	1.0 - 10	13	not eval.	320 ca	no	bsl
33213-65-9	beta-Endosulfan	2.85	2.85	µg/kg dw	1/12	2.0 - 200	200	not eval.	37,00 nc [f]	no	bsl
111-91-1	bis(2-chloroethoxy)methane	nd	nd	µg/kg dw	0/186	19 - 400	400	not eval.	na	no	ntx
111-44-4	bis(2-chloroethyl)ether	nd	nd	µg/kg dw	0/186	20 - 270	270	not eval.	210 ca	yes	asl
39638-32-9	bis(2-chloroisopropyl)ether	nd	nd	µg/kg dw	0/126	19 - 140	140	not eval.	6900 ca	no	bsl
117-81-7	bis(2-ethylhexyl)phthalate	5.42	10,000	µg/kg dw	166/203	20 - 389	10,000	not eval.	35,000 ca*	no	bsl
108-60-1	bis-chloroisopropyl ether	nd	nd	µg/kg dw	0/62	19 - 400	400	not eval.	6900 ca	no	bsl
108-86-1	Bromobenzene	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	2800 nc	no	bsl
74-97-5	Bromochloromethane	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	na	no	ntx
75-27-4	Bromodichloromethane	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	1000 ca	no	bsl
75-25-2	Bromoform	nd	nd	µg/kg dw	0/14	1.4 - 7.4	7.4	not eval.	62,000 ca*	no	bsl
74-83-9	Bromomethane	nd	nd	µg/kg dw	0/14	2.8 - 18.4	18.4	not eval.	390 nc	no	bsl
85-68-7	Butyl benzyl phthalate	2.38	7100	µg/kg dw	79/203	1.79 - 140	7100	not eval.	1,200,000 nc	no	bsl
	Butyltin (total)	91	91	µg/kg dw	1/7	15 – 20	91	not eval.	na	no	ntx
7440-43-9	Cadmium	0.070	92	mg/kg dw	143/211	0.040 - 1.6	92	0.360/1.12	3.7 nc	yes	asl
58-08-2	Caffeine	nd	nd	µg/kg dw	0/8	7.0 – 12	12	not eval.	na	no	ntx
7440-70-2	Calcium	1960	29,600	mg/kg dw	141/141	na	29,600	not eval.	na	no	ntx
86-74-8	Carbazole	12	2400	µg/kg dw	64/186	9.4 - 140	2400	not eval.	24,000 ca	no	bsl
75-15-0	Carbon disulfide	4.0	4.0	µg/kg dw	1/14	1.4 - 7.4	7.4	not eval.	36,000 nc	no	bsl
56-23-5	Carbon tetrachloride	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	240 ca**	no	bsl
	Carcinogenic PAHs (calc'd)	5.2	8620	µg/kg dw	190/203	18 - 109	8620	not eval.	62 ca [g]	yes	asl

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57-74-9	Chlordane	26	50	µg/kg dw	2/13	8.3 - 18	50	not eval.	1600 ca*	no	bsl
108-90-7	Chlorobenzene	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	15,000 nc	no	bsl
75-00-3	Chloroethane	nd	nd	µg/kg dw	0/14	2.8 - 18.4	18.4	not eval.	3000 ca	no	bsl
67-66-3	Chloroform	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	240 ca**	no	bsl
74-87-3	Chloromethane	nd	nd	µg/kg dw	0/14	2.3 - 18.4	18.4	not eval.	1200 ca	no	bsl
7440-47-3	Chromium	5.0	1100	mg/kg dw	211/211	na	1100	not eval.	210 ca	yes	asl
218-01-9	Chrysene	22	6800	µg/kg dw	188/203	5.4 - 120	6800	not eval.	62,000 ca	no	teq
156-59-2	cis-1,2-Dichloroethene	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	4300 nc	no	bsl
10061-01-5	cis-1,3-Dichloropropene	nd	nd	µg/kg dw	0/14	1.4 - 3.9	3.9	not eval.	na	no	ntx
7440-48-4	Cobalt	3.0	140	mg/kg dw	129/129	na	140	not eval.	470 nc	no	bsl
7440-50-8	Copper	5.0	12,000	mg/kg dw	211/211	na	12,000	21.3/50.8	290 nc	yes	asl
360-68-9	Coprostanol	570	2860	µg/kg dw	7/24	19 - 450	2860	not eval.	na	no	ntx
99-87-6	Cymene	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	na	no	ntx
	DDTs (total-calc'd)	4.0	2880	µg/kg dw	10/26	1.6 - 10	2880	not eval.	1700 ca [h]	yes	asl
319-86-8	delta-BHC	6.7	6.7	µg/kg dw	1/13	1.6 – 3.0	6.7	not eval.	na	no	ntx
53-70-3	Dibenzo(a,h)anthracene	2.2	2000	µg/kg dw	84/203	5.9 – 260	2000	not eval.	62 ca	no	teq
132-64-9	Dibenzofuran	20	470	µg/kg dw	39/202	1.7 – 140	470	not eval.	29,000 nc	no	bsl
124-48-1	Dibromochloromethane	nd	nd	µg/kg dw	0/14	1.4 - 7.4	7.4	not eval.	1100 ca	no	bsl
74-95-3	Dibromomethane	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	6700 nc	no	bsl
1002-53-5	Dibutyltin as ion	1.0	82	µg/kg dw	14/18	1.0 – 10	82	not eval.	na	no	ntx
75-71-8	Dichlorodifluoromethane	nd	nd	µg/kg dw	0/3	2.5 - 3.3	3.3	not eval.	9400 nc	no	bsl
75-09-2	Dichloromethane	nd	nd	µg/kg dw	0/14	2.8 - 18.4	18.4	not eval.	8900 ca	no	bsl
60-57-1	Dieldrin	2.6	280	µg/kg dw	3/24	1.6 - 10	280	not eval.	30 ca	yes	asl
60-29-7	Diethyl ether	nd	nd	µg/kg dw	0/11	2.3 - 7.4	7.4	not eval.	1,800,000 sat	no	bsl
84-66-2	Diethyl phthalate	40	130	µg/kg dw	2/203	1.79 - 200	200	not eval.	4,900,000 nc	no	bsl
131-11-3	Dimethyl phthalate	20	200	µg/kg dw	26/203	1.79 - 140	200	not eval.	1E8 max	no	bsl
84-74-2	Di-n-butyl phthalate	21	3800	µg/kg dw	69/203	1.79 - 210	3800	not eval.	610,000 nc	no	bsl

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117-84-0	Di-n-octyl phthalate	1.84	570	µg/kg dw	18/203	1.79 - 140	570	not eval.	120,000nc	no	bsl
115-29-7	Endosulfan	nd	nd	µg/kg dw	0/13	1.6 – 3.0	3.0	not eval.	37,000 nc	no	bsl
1031-07-8	Endosulfan sulfate	6.1	6.1	µg/kg dw	1/24	1.6 – 200	200	not eval.	na	no	ntx
72-20-8	Endrin	nd	nd	µg/kg dw	0/24	1.6 – 200	200	not eval.	1800 nc	no	bsl
7421-93-4	Endrin aldehyde	4.6	130	µg/kg dw	2/19	1.6 – 50	130	not eval.	na	no	ntx
53494-70-5	Endrin ketone	2.8	2.8	µg/kg dw	1/12	2.0 – 200	200	not eval.	na	no	ntx
97-63-2	Ethyl Methacrylate	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	140,000 sat	no	bsl
100-41-4	Ethylbenzene	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	230,000 sat	no	bsl
206-44-0	Fluoranthene	20	17,000	µg/kg dw	196/203	11 - 78	17,000	not eval.	230,000 nc	no	bsl
86-73-7	Fluorene	2.08	730	µg/kg dw	57/203	1.79 - 140	730	not eval.	260,000 nc	no	bsl
58-89-9	gamma-BHC	nd	nd	µg/kg dw	0/24	1.0 - 10	10	not eval.	440 ca*	no	bsl
5103-74-2	gamma-Chlordane	3.4	204	µg/kg dw	2/12	1.0 - 11	204	not eval.	1600 ca* [e]	no	ntx
76-44-8	Heptachlor	nd	nd	µg/kg dw	0/24	1.0 - 10	10	not eval.	110 ca	no	bsl
1024-57-3	Heptachlor epoxide	1.0	1.0	µg/kg dw	1/24	1.0 - 100	100	not eval.	53 ca*	yes	asl
118-74-1	Hexachlorobenzene	0.40	690	µg/kg dw	11/203	0.11 - 520	690	not eval.	300 ca	yes	asl
87-68-3	Hexachlorobutadiene	nd	nd	µg/kg dw	0/203	1.0 - 270	270	not eval.	6200 ca**	no	bsl
77-47-4	Hexachlorocyclopentadiene	nd	nd	µg/kg dw	0/174	34 - 2000	2000	not eval.	42,000 nc	no	bsl
67-72-1	Hexachloroethane	nd	nd	µg/kg dw	0/197	2.3 - 270	270	not eval.	35,000 ca**	no	bsl
193-39-5	Indeno(1,2,3-cd)pyrene	7.7	4300	µg/kg dw	163/203	12 - 130	4300	not eval.	620 ca	no	teq
74-88-4	Iodomethane	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	na	no	ntx
7439-89-6	Iron	8100	160,000	mg/kg dw	149/149	na	160,000	17,500/28,700	2300 nc	yes	asl
78-59-1	Isophorone	nd	nd	µg/kg dw	0/186	19 - 140	140	not eval.	510,000 ca*	no	bsl
98-82-8	iso-Propylbenzene	nd	nd	µg/kg dw	0/11	3.7 - 16.6	16.6	not eval.	16,000 nc	no	bsl
7439-92-1	Lead	2.0	23,000	mg/kg dw	211/211	na	23,000	15/45	40 nc	yes	asl
7439-95-4	Magnesium	2000	17,000	mg/kg dw	142/142	na	17,000	not eval.	na	no	ntx
7439-96-5	Manganese	78	3300	mg/kg dw	151/151	na	3300	279/1010	180 nc	yes	asl
7439-97-6	Mercury	0.030	4.6	mg/kg dw	165/208	0.020 - 0.22	4.6	0.0981/0.327	0.61 nc [i]	yes	asl

CAS Number	Chemical	Min. Detected Conc.	Max. detected Conc.	Units	Detection frequency (location)	Range of detection limits	Conc. Used for Screening	Background conc.[a]	RBC[b]	COPC Flag	Rationale for Selection or Exclusion
126-98-7	Methacrylonitrile	nd	nd	µg/kg dw	0/11	7.4 - 16.6	16.6	not eval.	210 nc	no	bsl
72-43-5	Methoxychlor	7.0	99	µg/kg dw	2/24	1.0 – 18	99	not eval.	31,000 nc	no	bsl
96-33-3	Methyl Acrylate	nd	nd	µg/kg dw	0/11	7.4 - 16.6	16.6	not eval.	7000 nc	no	bsl
78-93-3	Methyl ethyl ketone	8.7	16.8	µg/kg dw	4/14	4.6 - 7.4	16.8	not eval.	730,000 nc	no	bsl
108-10-1	Methyl iso-butyl ketone	nd	nd	µg/kg dw	0/14	4.6 - 12	12	not eval.	79,000 nc	no	bsl
80-62-6	Methyl Methacrylate	nd	nd	µg/kg dw	0/11	3.7 - 6.6	6.6	not eval.	220,000 nc	no	bsl
22967-92-6	Methylmercury	0.31	3.4	µg/kg dw	4/4	na	3.4	not eval.	610 nc	no	bsl
7439-98-7	Molybdenum	3.5	5.5	mg/kg dw	3/14	1.2 - 4.5	5.5	not eval.	39 nc	no	bsl
78763-54-9	Monobutyltin as ion	25.3	25.3	µg/kg dw	1/1	na	25.3	not eval.	na	no	ntx
91-20-3	Naphthalene	20	380	µg/kg dw	20/203	1.79 - 140	380	not eval.	5600 nc	no	bsl
104-51-8	n-Butylbenzene	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	14,000 nc	no	bsl
2406-65-7	n-Butyltin	1.0	66	µg/kg dw	12/18	5.0 - 45	66	not eval.	na	no	ntx
7440-02-0	Nickel	5.0	910	mg/kg dw	205/206	32 - 32	910	26.8/41.7	160 nc	yes	asl
98-95-3	Nitrobenzene	nd	nd	µg/kg dw	0/186	19 - 560	560	not eval.	2000 nc	no	bsl
62-75-9	N-Nitrosodimethylamine	nd	nd	µg/kg dw	0/24	27 - 250	250	not eval.	9.5 ca	yes	asl
621-64-7	N-Nitroso-di-n-propylamine	nd	nd	µg/kg dw	0/186	12 - 1100	1100	not eval.	69 ca	yes	asl
86-30-6	N-Nitrosodiphenylamine	76	190	µg/kg dw	3/203	1.79 - 560	560	not eval.	99,000 ca	no	bsl
103-65-1	n-Propylbenzene	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	14,000 nc	no	bsl
3268-87-9	OCDD	420	91,000	ng/kg dw	8/8	na	91,000	not eval.	na	no	teq
39001-02-0	OCDF	22	3600	ng/kg dw	8/8	na	3600	not eval.	na	no	teq
37680-73-2	PCB-101	0.41	5600	µg/kg dw	141/164	0.16 – 5.0	5600	not eval.	na	no	sum
32598-14-4	PCB-105	0.25	560	µg/kg dw	102/163	0.13 – 1.0	560	not eval.	na	no	sum
38380-03-9	PCB-110	0.22	3000	µg/kg dw	98/115	0.13 - 1.2	3000	not eval.	na	no	sum
74472-37-0	PCB-114	1.0	5.0	µg/kg dw	5/50	1.0 - 12	12	not eval.	na	no	sum
31508-00-6	PCB-118	0.42	2200	µg/kg dw	113/164	0.12 - 8.3	2200	not eval.	na	no	sum
65510-44-3	PCB-123	nd	nd	µg/kg dw	0/50	1.0 - 31	31	not eval.	na	no	sum
57465-28-8	PCB-126	0.65	3.0	µg/kg dw	2/164	0.10 – 4.0	4.0	not eval.	na	no	sum

CAS Number	Chemical	Min. Detected Conc.	Max. detected Conc.	Units	Detection frequency (location)	Range of detection limits	Conc. Used for Screening	Background conc.[a]	RBC[b]	COPC Flag	Rationale for Selection or Exclusion
38380-07-3	PCB-128	0.35	620	µg/kg dw	82/164	0.14 – 13	620	not eval.	na	no	sum
35065-28-2	PCB-138	0.21	1400	µg/kg dw	143/165	0.13 – 5.0	1400	not eval.	na	no	sum
35065-27-1	PCB-153	0.48	3000	µg/kg dw	140/164	0.13 - 4.4	3000	not eval.	na	no	sum
38380-08-4	PCB-156	0.33	160	µg/kg dw	56/165	0.080 – 1.0	160	not eval.	na	no	sum
69782-90-7	PCB-157	0.44	56	µg/kg dw	31/163	0.080 - 27	56	not eval.	na	no	sum
52663-72-6	PCB-167	1.0	30	µg/kg dw	10/50	1.0 – 1.0	30	not eval.	na	no	sum
32774-16-6	PCB-169	nd	nd	µg/kg dw	0/165	0.25 - 1.9	1.9	not eval.	na	no	sum
35065-30-6	PCB-170	0.19	560	µg/kg dw	102/165	0.080 - 12	560	not eval.	na	no	sum
37680-65-2	PCB-18	1.0	170	µg/kg dw	7/40	1.0 - 24	170	not eval.	na	no	sum
35065-29-3	PCB-180	0.19	965	µg/kg dw	118/165	0.11 - 4.9	965	not eval.	na	no	sum
52663-68-0	PCB-187	1.0	360	µg/kg dw	36/50	1.0 – 1.0	360	not eval.	na	no	sum
39635-31-9	PCB-189	0.78	11.5	µg/kg dw	18/165	0.11 – 5.0	11.5	not eval.	na	no	sum
52663-78-2	PCB-195	1.0	49	µg/kg dw	10/50	1.0 – 2.0	49	not eval.	na	no	sum
40186-72-9	PCB-206	1.0	21	µg/kg dw	9/50	1.0 – 1.0	21	not eval.	na	no	sum
2051-24-3	PCB-209	1.0	2.0	µg/kg dw	5/50	1.0 – 1.0	2.0	not eval.	na	no	sum
7012-37-5	PCB-28	1.0	160	µg/kg dw	14/50	1.0 – 2.0	160	not eval.	na	no	sum
41464-39-5	PCB-44	1.0	190	µg/kg dw	22/50	1.0 – 1.0	190	not eval.	na	no	sum
35693-99-3	PCB-55	1.0	890	µg/kg dw	32/50	1.0 – 2.0	890	not eval.	na	no	sum
32598-10-0	PCB-66	1.0	440	µg/kg dw	41/50	1.0 - 250	440	not eval.	na	no	sum
32598-13-3	PCB-77	0.93	26	µg/kg dw	15/165	0.11 - 15	26	not eval.	na	no	sum
70362-50-4	PCB-81	nd	nd	µg/kg dw	0/50	1.0 – 1.0	1.0	not eval.	na	no	sum
	PCBs (total-calc'd)	2.2	222,600	µg/kg dw	354/374	0.60 - 40	222,600	not eval.	220 ca [j]	yes	asl
	PCBs + PCTs (total)	2.2	26,000	µg/kg dw	114/115	0.60 - 0.60	26,000	not eval.	na	no	ntx
	PCTs (total)	2.2	5600	µg/kg dw	89/117	1.7 - 8.1	5600	not eval.	na	no	ntx
76-01-7	Pentachloroethane	nd	nd	µg/kg dw	0/11	2.3 - 7.4	7.4	not eval.	na	no	ntx
87-86-5	Pentachlorophenol	100	300	µg/kg dw	2/170	6.7 – 3800	3800	not eval.	3000 ca	no	ifd [c]
85-01-8	Phenanthrene	7.05	8300	µg/kg dw	177/203	5.4 – 130	8300	not eval.	na	no	ntx

CAS Number	Chemical	Min. Detected Conc.	Max. detected Conc.	Units	Detection frequency (location)	Range of detection limits	Conc. Used for Screening	Background conc.[a]	RBC[b]	COPC Flag	Rationale for Selection or Exclusion
108-95-2	Phenol	20	2100	µg/kg dw	49/203	12 – 270	2100	not eval.	3,700,000 nc	no	bsl
7440-09-7	Potassium	380	11,100	mg/kg dw	143/143	na	11,100	not eval.	na	no	ntx
129-00-0	Pyrene	24	13,000	µg/kg dw	191/203	5.4 – 79	13,000	not eval.	230,000 nc	no	bsl
135-98-8	sec-Butylbenzene	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	11,000 nc	no	bsl
7782-49-2	Selenium	0.70	20	mg/kg dw	50/152	1.0 - 34	34	not eval.	39 nc	no	bsl
7440-22-4	Silver	0.060	270	mg/kg dw	119/211	0.25 - 3.3	270	0.28/0.74	39 nc	yes	asl
7440-23-5	Sodium	580	20,800	mg/kg dw	141/141	na	20,800	not eval.	na	no	ntx
100-42-5	Styrene	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	1,700,000 sat	no	bsl
	Sulfides (total)	9.8	2100	mg/kg dw	18/52	0.68 - 3.7	2100	not eval.	na	no	ntx
1634-04-4	Tert-butyl methyl ether	nd	nd	µg/kg dw	0/11	2.3 - 7.4	7.4	not eval.	na	no	ntx
98-06-6	tert-Butylbenzene	nd	nd	µg/kg dw	0/11	2.3 - 3.7	3.7	not eval.	13,000 nc	no	bsl
1461-25-2	Tetrabutyltin as ion	2.0	2.0	µg/kg dw	1/20	0.88 - 32	32	not eval.	na	no	ntx
127-18-4	Tetrachloroethene	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	5700 ca*	no	bsl
7440-28-0	Thallium	0.010	30	mg/kg dw	71/152	5.0 - 45	45	0.252/1.79	0.63 nc	yes	asl
7440-31-5	Tin	2.0	466	mg/kg dw	17/50	1.0 – 9.0	466	not eval.	4700 nc	no	bsl
108-88-3	Toluene	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	520,000 sat	no	bsl
	Total HPAH (calc'd)	3.0	68,900	µg/kg dw	198/203	53 – 78	68,900	not eval.	na	no	ntx
	Total LPAH (calc'd)	8.4	10,750	µg/kg dw	178/203	20 – 130	10,750	not eval.	na	no	ntx
8001-35-2	Toxaphene	nd	nd	µg/kg dw	0/24	10 - 3700	3700	not eval.	440 ca	no	ifd [d]
	TPH	23	23,000	mg/kg dw	49/55	20 – 20	23,000	not eval.	na	no	ntx
	TPH - Diesel Range	105.5	164	mg/kg dw	2/2	na	164	not eval.	na	no	ntx
	TPH - Gasoline Range	nd	nd	mg/kg dw	0/2	20 – 20	20	not eval.	na	no	ntx
	TPH - Heavy Fuel Oil Range	250	370	mg/kg dw	2/2	na	370	not eval.	na	no	ntx
156-60-5	trans-1,2-Dichloroethene	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	6300 nc	no	bsl
10061-02-6	trans-1,3-Dichloropropene	nd	nd	µg/kg dw	0/14	1.4 - 6.2	6.2	not eval.	na	no	ntx
110-57-6	trans-1,4-Dichloro-2-butene	nd	nd	µg/kg dw	0/10	11.5 – 18.4	18.4	not eval.	na	no	ntx
688-73-3	Tributyltin as ion	1.0	216	µg/kg dw	18/18	na	216	not eval.	900 nc [k]	no	bsl

CAS Number	Chemical	Min. Detected Conc.	Max. detected Conc.	Units	Detection frequency (location)	Range of detection limits	Conc. Used for Screening	Background conc.[a]	RBC[b]	COPC Flag	Rationale for Selection or Exclusion
79-01-6	Trichloroethene	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	2800 ca**	no	bsl
75-69-4	Trichlorofluoromethane	nd	nd	µg/kg dw	0/14	2.3 - 73.8	73.8	not eval.	39,000 nc	no	bsl
7440-62-2	Vanadium	15	150	mg/kg dw	129/129	na	150	36.0/59.6	55 nc	yes	asl
108-05-4	Vinyl acetate	nd	nd	µg/kg dw	0/3	7.0 – 12	12	not eval.	43,000 nc	no	bsl
75-01-4	Vinyl chloride	nd	nd	µg/kg dw	0/14	2.3 - 18.4	18.4	not eval.	22 ca	no	bsl
108-38-3/106-42-3	Xylene (meta & para)	nd	nd	µg/kg dw	0/14	1.4 - 7.4	7.4	not eval.	210,000 sat [l]	no	bsl
95-47-6	Xylene (ortho)	nd	nd	µg/kg dw	0/14	1.4 - 3.7	3.7	not eval.	210,000 sat [l]	no	bsl
7440-66-6	Zinc	16	6,400	mg/kg dw	210/211	128 - 128	6,400	52.6/98.5	2300 nc	yes	asl

[a] Background concentrations obtained from joint Ecology/PSAMP 1998 study entitled "Sediment Quality in Puget Sound. Year 2- Central Puget Sound" (Ecology 2000). Reported concentrations are mean and maximum from 52 samples collected from the following strata: South Port Townsend, Port Townsend, North Admiralty Inlet, South Admiralty Inlet, Possession Sound, Central Basin, Port Madison, West Point, East Passage, Liberty Bay, Keyport, Northwest Bainbridge Island, Southwest Bainbridge Island, Rich Passage, Port Orchard, and Port Washington Narrows

[b] Risk-based concentrations (RBCs) are derived from EPA Region 9 Preliminary Remediation Goals (PRGs) for residential soil (last updated October 1999). PRGs associated with a non-cancer endpoint (abbreviated "nc") were divided by 10 for this screening, reflecting the different target hazard quotients used in Region 9 (HQ = 1) and Region 10 (HQ = 0.1). All other PRGs were not modified for this screening. Abbreviations: ca = cancer endpoint, nc = non-cancer endpoint, sat = soil saturation, m = ceiling limit, * = nc < 100X ca, ** = nc < 10X ca

[c] 167 of 168 detection limits (and both detections) were less than RBC

[d] 23 of 24 detection limits were less than RBC

[e] RBC is for chlordane

[f] RBC is for endosulfan

[g] RBC is for benzo(a)pyrene

[h] RBC is for 4,4'-DDT

[i] RBC is for methylmercury

[j] RBC is for Aroclor 1254

[k] RBC for tributyltin oxide multiplied by 0.49 to account for differences in molecular weight

[l] RBC is for total xylenes

Other abbreviations: nd = not detected, n/a = not applicable; HPAH = high-molecular-weight polycyclic aromatic hydrocarbon; LPAH = low-molecular-weight polycyclic aromatic hydrocarbon

Rationale codes Selection reason: above screening level (asl)
Exclusion reason: infrequent detection (ifd)

no toxicity information (ntx)
below screening level (bsl)
chemical included in sum and is not evaluated separately (sum)
chemical included in TEQ calculation and is not evaluated separately (teq)

Table 3. Occurrence, distribution, and selection of chemicals of potential concern for tissue in the seafood consumption exposure scenario

CAS Number	Chemical	Min. Detected Conc.	Max. detected Conc.	Units	Detection frequency	Range of detection limits	Conc. Used for Screening	Background conc. [a]	RBC[b]	COPC Flag	Rationale for Selection or Exclusion
120-82-1	1,2,4-Trichlorobenzene	nd	nd	µg/kg ww	0/30	3.6 - 16	16	not eval.	980 nc	no	bsl
95-50-1	1,2-Dichlorobenzene	nd	nd	µg/kg ww	0/30	10.7 - 16	16	not eval.	8400 nc	no	bsl
122-66-7	1,2-Diphenylhydrazine	nd	nd	µg/kg ww	0/30	3.6 - 53	53	not eval.	1.5 ca	yes	asl
541-73-1	1,3-Dichlorobenzene	nd	nd	µg/kg ww	0/30	10.7 - 16	16	not eval.	2900 nc	no	bsl
106-46-7	1,4-Dichlorobenzene	nd	nd	µg/kg ww	0/30	10.7 - 16	16	not eval.	49 ca	no	bsl
95-95-4	2,4,5-Trichlorophenol	nd	nd	µg/kg ww	0/30	18 - 110	110	not eval.	9800 nc	no	bsl
88-06-2	2,4,6-Trichlorophenol	nd	nd	µg/kg ww	0/30	18 - 110	110	not eval.	110 ca	no	ifd [c]
120-83-2	2,4-Dichlorophenol	nd	nd	µg/kg ww	0/30	3.6 - 27	27	not eval.	290 nc	no	bsl
105-67-9	2,4-Dimethylphenol	nd	nd	µg/kg ww	0/30	3.6 - 27	27	not eval.	1900 nc	no	bsl
51-28-5	2,4-Dinitrophenol	nd	nd	µg/kg ww	0/30	53 - 72	72	not eval.	190 nc	no	bsl
121-14-2	2,4-Dinitrotoluene	nd	nd	µg/kg ww	0/30	11 - 18	18	not eval.	190 nc	no	bsl
606-20-2	2,6-Dinitrotoluene	nd	nd	µg/kg ww	0/30	11 - 67	67	not eval.	95.8 nc	no	bsl
91-58-7	2-Chloronaphthalene	nd	nd	µg/kg ww	0/30	10.7 - 97	97	not eval.	7700 nc	no	bsl
95-57-8	2-Chlorophenol	nd	nd	µg/kg ww	0/30	3.6 - 53	53	not eval.	480 nc	no	bsl
91-57-6	2-Methylnaphthalene	nd	nd	µg/kg ww	0/30	3.6 - 43	43	not eval.	1900 nc	no	bsl
95-48-7	2-Methylphenol	28	93.7	µg/kg ww	18/30	3.6 - 27	93.7	not eval.	4800 nc	no	bsl
88-74-4	2-Nitroaniline	nd	nd	µg/kg ww	0/30	7.1 - 110	110	not eval.	n/a	no	ntx
88-75-5	2-Nitrophenol	nd	nd	µg/kg ww	0/30	3.6 - 27	27	not eval.	n/a	no	ntx
91-94-1	3,3'-Dichlorobenzidine	nd	nd	µg/kg ww	0/27	27 - 27	27	not eval.	2.7 ca	yes	asl
99-09-2	3-Nitroaniline	nd	nd	µg/kg ww	0/30	3.6 - 110	110	not eval.	n/a	no	ntx
72-54-8	4,4'-DDD	1.1	4.96	µg/kg ww	6/20	1.3 - 1.3	4.96	not eval.	4.9 ca	no	sum
72-55-9	4,4'-DDE	1.1	5.94	µg/kg ww	7/20	1.0 - 1.3	5.94	not eval.	3.5 ca	no	sum
50-29-3	4,4'-DDT	nd	nd	µg/kg ww	0/20	1.3 – 2.0	2.0	not eval.	3.5 ca	no	sum
534-52-1	4,6-Dinitro-o-cresol	nd	nd	µg/kg ww	0/30	53 – 53	53	not eval.	95 nc	no	bsl

CAS Number	Chemical	Min. Detected Conc.	Max. detected Conc.	Units	Detection frequency	Range of detection limits	Conc. Used for Screening	Background conc. [a]	RBC[b]	COPC Flag	Rationale for Selection or Exclusion
101-55-3	4-Bromophenyl phenyl ether	nd	nd	µg/kg ww	0/28	11 - 70	70	not eval.	n/a	no	ntx
59-50-7	4-Chloro-3-methylphenol	nd	nd	µg/kg ww	0/30	3.6 - 53	53	not eval.	n/a	no	ntx
106-47-8	4-Chloroaniline	nd	nd	µg/kg ww	0/30	36 - 53	53	not eval.	380 nc	no	bsl
7005-72-3	4-Chlorophenyl phenyl ether	nd	nd	µg/kg ww	0/30	3.6 - 16	16	not eval.	n/a	no	ntx
106-44-5	4-Methylphenol	nd	nd	µg/kg ww	0/30	3.6 - 27	27	not eval.	480 nc	no	bsl
100-01-6	4-Nitroaniline	nd	nd	µg/kg ww	0/30	18 - 110	110	not eval.	n/a	no	ntx
100-02-7	4-Nitrophenol	nd	nd	µg/kg ww	0/30	36 - 53	53	not eval.	770 nc	no	bsl
83-32-9	Acenaphthene	nd	nd	µg/kg ww	0/30	3.6 - 11	11	not eval.	5700 nc	no	bsl
208-96-8	Acenaphthylene	nd	nd	µg/kg ww	0/30	3.6 - 16	16	not eval.	n/a	no	ntx
309-00-2	Aldrin	nd	nd	µg/kg ww	0/20	0.50 - 1.3	1.3	not eval.	0.072 ca	yes	asl
319-84-6	alpha-BHC	nd	nd	µg/kg ww	0/20	0.50 - 1.3	1.3	not eval.	0.19	yes	asl
5103-71-9	alpha-Chlordane	1.6	2.0	µg/kg ww	3/9	0.50 - 0.50	2.0	not eval.	3.4 ca [d]	no	bsl
959-98-8	alpha-Endosulfan	nd	nd	µg/kg ww	0/20	0.50 - 1.3	1.3	not eval.	570 nc [e]	no	bsl
62-53-3	Aniline	nd	nd	µg/kg ww	0/30	53 - 53.3	53.3	not eval.	210 ca	no	bsl
120-12-7	Anthracene	nd	nd	µg/kg ww	0/30	3.6 – 16	16	not eval.	29,000 nc	no	bsl
7440-36-0	Antimony	nd	nd	mg/kg ww	0/27	0.010 - 0.13	0.13	not eval.	0.038 nc	no	ifd [l]
12674-11-2	Aroclor-1016	nd	nd	µg/kg ww	0/33	5.3 – 20	20	not eval.	17 ca	no	sum
11104-28-2	Aroclor-1221	nd	nd	µg/kg ww	0/33	5.3 – 20	20	not eval.	0.61 ca	no	sum
11141-16-5	Aroclor-1232	nd	nd	µg/kg ww	0/33	5.3 – 20	20	not eval.	0.61 ca	no	sum
53469-21-9	Aroclor-1242	nd	nd	µg/kg ww	0/33	5.3 – 13	13	not eval.	0.61 ca	no	sum
12672-29-6	Aroclor-1248	9.0	26.1	µg/kg ww	7/45	0.21 – 13	26.1	not eval.	0.61 ca	no	sum
11097-69-1	Aroclor-1254	16	300	µg/kg ww	41/45	13 – 13	300	not eval.	0.61 ca	no	sum
11096-82-5	Aroclor-1260	26.5	210	µg/kg ww	23/45	13 – 13	210	not eval.	0.61 ca	no	sum
7440-38-2	Arsenic	0.34	15.1	mg/kg ww	33/33	n/a	15.1	7.7	0.00080 ca	yes	asl
92-87-5	Benzidine	nd	nd	µg/kg ww	0/27	640 – 640	640	not eval.	0.0053 ca	yes	asl
56-55-3	Benzo(a)anthracene	17	32.2	µg/kg ww	11/30	10.7 – 43	43	not eval.	1.6 ca	no	teq
50-32-8	Benzo(a)pyrene	nd	nd	µg/kg ww	0/30	3.6 – 43	43	not eval.	0.16 ca	no	teq

CAS Number	Chemical	Min. Detected Conc.	Max. detected Conc.	Units	Detection frequency	Range of detection limits	Conc. Used for Screening	Background conc. [a]	RBC[b]	COPC Flag	Rationale for Selection or Exclusion
205-99-2	Benzo(b)fluoranthene	43	43	µg/kg ww	1/30	10.7 – 43	43	not eval.	1.6 ca	no	teq
191-24-2	Benzo(g,h,i)perylene	nd	nd	µg/kg ww	0/30	10.7 – 170	170	not eval.	n/a	no	ntx
207-08-9	Benzo(k)fluoranthene	nd	nd	µg/kg ww	0/30	7.1 – 43	43	not eval.	16 ca	no	teq
65-85-0	Benzoic acid	793	4,000	µg/kg ww	22/30	36 - 110	4000	65	380,000 nc	no	bsl
100-51-6	Benzyl alcohol	28	28	µg/kg ww	1/30	3.6 - 27	28	14	29,000 nc	no	bsl
319-85-7	beta-BHC	nd	nd	µg/kg ww	0/20	0.50 - 1.3	1.3	not eval.	0.68	yes	asl
33213-65-9	beta-Endosulfan	nd	nd	µg/kg ww	0/20	1.0 - 1.3	1.3	not eval.	570 nc [e]	no	bsl
111-91-1	bis(2-chloroethoxy)methane	nd	nd	µg/kg ww	0/30	3.6 - 27	27	not eval.	n/a	no	ntx
111-44-4	bis(2-chloroethyl)ether	nd	nd	µg/kg ww	0/30	3.6 - 16	16	not eval.	1.1 ca	yes	asl
117-81-7	bis(2-ethylhexyl)phthalate	27.6	187	µg/kg ww	3/30	3.6 - 100	187	140	87 ca	yes	asl
108-60-1	bis-chloroisopropyl ether	nd	nd	µg/kg ww	0/30	10.7 - 53	53	not eval.	17 ca	yes	asl
85-68-7	Butyl benzyl phthalate	nd	nd	µg/kg ww	0/30	10.7 - 100	100	not eval.	19,000 nc	no	bsl
7440-43-9	Cadmium	0.012	0.84	mg/kg ww	24/27	0.0079 - 0.050	0.84	not eval.	0.098 nc	yes	asl
58-08-2	Caffeine	nd	nd	µg/kg ww	0/27	5.3 – 34	34	not eval.	n/a	no	ntx
86-74-8	Carbazole	nd	nd	µg/kg ww	0/30	3.6 - 27	27	not eval.	61	no	bsl
	cPAHs	45	48	µg/kg ww	13/30	10 - 53	53	not eval.	0.61 ca [m]	yes	asl
57-74-9	Chlordane	nd	nd	µg/kg ww	0/11	6.7 - 6.7	6.7	not eval.	3.4 ca	yes	asl
7440-47-3	Chromium	0.054	0.346	mg/kg ww	25/27	0.050 - 0.32	0.346	not eval.	0.29 nc [f]	yes	asl
218-01-9	Chrysene	19	45.8	µg/kg ww	11/30	3.6 – 16	45.8	not eval.	160 ca	no	bsl
7440-48-4	Cobalt	0.030	0.070	mg/kg ww	11/11	n/a	0.070	not eval.	1.9 nc	no	bsl
7440-50-8	Copper	0.175	15.8	mg/kg ww	33/33	n/a	15.8	0.31	3.8 nc	yes	asl
360-68-9	Coprostanol	nd	nd	µg/kg ww	0/30	110 – 180	180	not eval.	n/a	no	ntx
	DDTs (total-calc'd)	1.1	10.9	µg/kg ww	7/20	1.3 – 2.0	10.9	0.77	3.5 ca [g]	yes	asl
319-86-8	delta-BHC	nd	nd	µg/kg ww	0/20	0.50 - 1.3	1.3	not eval.	n/a	no	ntx
53-70-3	Dibenzo(a,h)anthracene	nd	nd	µg/kg ww	0/30	10.7 - 43	43	not eval.	0.16 ca	no	teq
132-64-9	Dibenzofuran	nd	nd	µg/kg ww	0/30	10.7 - 27	27	not eval.	380 nc	no	bsl
1002-53-5	Dibutyltin as ion	4.0	11.4	µg/kg ww	11/11	n/a	11.4	not eval.	n/a	no	ntx

CAS Number	Chemical	Min. Detected Conc.	Max. detected Conc.	Units	Detection frequency	Range of detection limits	Conc. Used for Screening	Background conc. [a]	RBC[b]	COPC Flag	Rationale for Selection or Exclusion
60-57-1	Dieldrin	nd	nd	µg/kg ww	0/20	1.0 - 1.3	1.3	not eval.	0.076 ca	yes	asl
84-66-2	Diethyl phthalate	nd	nd	µg/kg ww	0/30	3.6 - 170	170	not eval.	77,000 nc	no	bsl
131-11-3	Dimethyl phthalate	nd	nd	µg/kg ww	0/30	3.6 – 11	11	not eval.	980,000 nc	no	bsl
84-74-2	Di-n-butyl phthalate	20	20	µg/kg ww	1/30	3.6 – 27	27	not eval.	9800 nc	no	bsl
117-84-0	Di-n-octyl phthalate	nd	nd	µg/kg ww	0/30	3.6 – 16	16	not eval.	1900 nc	no	bsl
1031-07-8	Endosulfan sulfate	nd	nd	µg/kg ww	0/20	1.0 - 1.3	1.3	not eval.	n/a	no	ntx
72-20-8	Endrin	nd	nd	µg/kg ww	0/20	1.0 - 1.3	1.3	not eval.	29 nc	no	bsl
7421-93-4	Endrin aldehyde	nd	nd	µg/kg ww	0/20	1.0 - 1.3	1.3	not eval.	n/a	no	ntx
206-44-0	Fluoranthene	17	58.3	µg/kg ww	21/30	3.6 – 16	58.3	not eval.	3800 nc	no	bsl
86-73-7	Fluorene	nd	nd	µg/kg ww	0/30	3.6 – 16	16	not eval.	3800 nc	no	bsl
58-89-9	gamma-BHC	nd	nd	µg/kg ww	0/20	0.50 - 1.3	1.3	not eval.	0.92 ca	yes	asl
5103-74-2	gamma-Chlordane	0.52	0.52	µg/kg ww	1/9	0.50 - 0.50	0.52	not eval.	3.4 ca [d]	no	bsl
76-44-8	Heptachlor	nd	nd	µg/kg ww	0/20	0.50 - 1.3	1.3	not eval.	0.27 ca	yes	asl
1024-57-3	Heptachlor epoxide	nd	nd	µg/kg ww	0/20	0.50 - 1.3	1.3	not eval.	0.13 ca	yes	asl
118-74-1	Hexachlorobenzene	nd	nd	µg/kg ww	0/30	16 – 18	18	not eval.	0.76 ca	yes	asl
87-68-3	Hexachlorobutadiene	nd	nd	µg/kg ww	0/30	10.7 - 27	27	not eval.	15 ca	yes	asl
77-47-4	Hexachlorocyclopentadiene	nd	nd	µg/kg ww	0/30	27 – 36	36	not eval.	570 nc	no	bsl
67-72-1	Hexachloroethane	nd	nd	µg/kg ww	0/29	10.7 - 27	27	not eval.	87 ca	no	bsl
193-39-5	Indeno(1,2,3-cd)pyrene	nd	nd	µg/kg ww	0/30	18 – 27	27	not eval.	1.6 ca	no	teq
78-59-1	Isophorone	nd	nd	µg/kg ww	0/30	3.6 – 27	27	not eval.	1300 ca	no	bsl
7439-92-1	Lead	0.133	0.723	mg/kg ww	24/33	0.020 - 0.030	0.723	not eval.	n/a	yes	alt
7439-97-6	Mercury	0.0088	0.111	mg/kg ww	43/44	20 – 20	0.111	0.051	0.0095 nc [h]	yes	asl
72-43-5	Methoxychlor	nd	nd	µg/kg ww	0/20	6.7 – 10	10	not eval.	480 nc	no	bsl
22967-92-6	Methylmercury	18	24.6	µg/kg ww	3/3	n/a	24.6	not eval.	9.8 nc	no	asl [k]
7439-98-7	Molybdenum	0.023	0.101	mg/kg ww	10/10	n/a	0.101	not eval.	0.48 nc	no	bsl
78763-54-9	Monobutyltin as ion	1.75	4.91	µg/kg ww	9/11	1.74 - 1.74	4.91	not eval.	n/a	no	ntx
91-20-3	Naphthalene	nd	nd	µg/kg ww	0/30	3.6 – 43	43	not eval.	1900 nc	no	bsl

CAS Number	Chemical	Min. Detected Conc.	Max. detected Conc.	Units	Detection frequency	Range of detection limits	Conc. Used for Screening	Background conc. [a]	RBC[b]	COPC Flag	Rationale for Selection or Exclusion
7440-02-0	Nickel	0.051	0.42	mg/kg ww	24/27	0.020 - 0.13	0.42	not eval.	1.9 nc	no	bsl
98-95-3	Nitrobenzene	nd	nd	µg/kg ww	0/30	10.7 - 27	27	not eval.	48 nc	no	bsl
62-75-9	N-Nitrosodimethylamine	nd	nd	µg/kg ww	0/30	3.6 - 110	110	not eval.	0.024 ca	yes	asl
621-64-7	N-Nitroso-di-n-propylamine	nd	nd	µg/kg ww	0/30	3.6 – 27	27	not eval.	0.17 ca	yes	asl
86-30-6	N-Nitrosodiphenylamine	nd	nd	µg/kg ww	0/30	3.6 – 27	27	not eval.	240 ca	no	bsl
	PCBs (total-calc'd)	16	526	µg/kg ww	41/45	13 – 13	526	18	0.60 ca [i]	yes	asl
87-86-5	Pentachlorophenol	nd	nd	µg/kg ww	0/30	27 – 36	36	not eval.	9.9 ca	yes	asl
85-01-8	Phenanthrene	nd	nd	µg/kg ww	0/30	3.6 – 16	16	not eval.	n/a	no	ntx
108-95-2	Phenol	nd	nd	µg/kg ww	0/30	3.6 - 700	700	not eval.	57,000 nc	no	bsl
129-00-0	Pyrene	17	39.6	µg/kg ww	13/30	3.6 – 16	39.6	not eval.	2900 nc	no	bsl
7440-22-4	Silver	0.114	0.187	mg/kg ww	2/27	0.010 - 0.076	0.187	not eval.	0.48 nc	no	bsl
8001-35-2	Toxaphene	nd	nd	µg/kg ww	0/20	10 – 13	13	not eval.	1.1 ca	yes	asl
688-73-3	Tributyltin as ion	2.0	81.9	µg/kg ww	30/39	0.74 – 2.0	81.9	not eval.	14 nc [j]	yes	asl
7440-62-2	Vanadium	0.058	0.257	mg/kg ww	8/8	n/a	0.257	not eval.	0.67 nc	no	bsl
7440-66-6	Zinc	3.82	44.1	mg/kg ww	27/27	n/a	44.1	not eval.	29 nc	yes	asl

[a] Background concentration is an average from up to 229 English sole filet samples collected from non-urban areas by the PSAMP (West et al. 2001) from 1989-1999

[b] Risk-based concentrations (RBCs) are derived from EPA Region 3 risk-based concentrations for fish tissue (last updated October 2001). Site specific modifications were made to the Region 3 RBCs to reflect differences in body weight (70 kg for Region 3, 79 kg for this site), seafood consumption rate (54 g/day for Region 3, 84 g/day for this site), and exposure frequency (350 d/yr for Region 3, 365 d/yr for this site), and exposure duration (30 yrs for Region 3, 55 yrs for this site). For chemicals with cancer endpoints, the Region 3 RBCs were multiplied by 0.38 to reflect the site-specific modifications; RBCs for chemicals with non-cancer endpoints were multiplied by 0.70 to reflect the site-specific modifications. RBCs associated with a non-cancer endpoint (abbreviated "nc") were divided by 10 for this screening, reflecting the different target hazard quotients used in Region 3 (HQ = 1) and Region 10 (HQ = 0.1). All other RBCs were not modified for this screening. Abbreviations: ca = cancer endpoint, nc = non-cancer endpoint

[c] 27 of 30 detection limits are equal to RBC, but none are greater

[d] RBC is for chlordane

[e] RBC is for endosulfan

[f] RBC is for hexavalent chromium

[g] RBC is for 4,4'-DDT

[h] RBC is for methylmercury

[i] RBC is for Aroclor 1254

[j] RBC for tributyltin oxide multiplied by 0.49 to account for differences in molecular weight

[k] Not selected as COPC; mercury is used as surrogate

[l] 26 of 27 detection limits less than RBC

[m] RBC is for benzo(a)pyrene

Other abbreviations: nd = not detected, n/a = not applicable

Rationale codes		
	Selection reason:	above screening level (asl)
		alternate toxicity evaluation method available (i.e., IEUBK model) (alt)
	Exclusion reason:	infrequent detection (ifd)
		no toxicity information (ntx)
		below screening level (bsl)
		chemical included in sum and is not evaluated separately (sum)
		chemical included in TEQ calculation and is not evaluated separately (teq)

Table 4. Chemicals analyzed in sediment, but not in tissue samples evaluated in HHRA

CAS Number	Chemical	Detected in Surface Sediment	Detection frequency (location)	Identified as COPC in Sediment	Important Bioaccumulative Compound [a]
630-20-6	1,1,1,2-Tetrachloroethane	no	0/44	no	no
71-55-6	1,1,1-Trichloroethane	no	0/49	no	no
79-34-5	1,1,2,2-Tetrachloroethane	no	0/49	no	no
79-00-5	1,1,2-Trichloroethane	no	0/49	no	no
76-13-1	1,1,2-Trichlorotrifluoroethane	no	0/47	no	no
513-88-2	1,1-Dichloroacetone	no	0/42	no	no
75-34-3	1,1-Dichloroethane	no	0/49	no	no
75-35-4	1,1-Dichloroethene	no	0/49	yes	no
563-58-6	1,1-Dichloropropene	no	0/44	no	no
35822-46-9	1,2,3,4,6,7,8-HpCDD	yes	27/29	no	yes
67562-39-4	1,2,3,4,6,7,8-HpCDF	yes	26/29	no	no
55673-89-7	1,2,3,4,7,8,9-HpCDF	yes	11/29	no	no
39227-28-6	1,2,3,4,7,8-HxCDD	yes	2/29	no	yes
70648-26-9	1,2,3,4,7,8-HxCDF	yes	14/29	no	yes
57653-85-7	1,2,3,6,7,8-HxCDD	yes	20/29	no	yes
57117-44-9	1,2,3,6,7,8-HxCDF	yes	2/29	no	no
19408-74-3	1,2,3,7,8,9-HxCDD	yes	15/29	no	no
72918-21-9	1,2,3,7,8,9-HxCDF	yes	1/29	no	no
40321-76-4	1,2,3,7,8-PeCDD	yes	2/29	no	no
57117-41-6	1,2,3,7,8-PeCDF	yes	1/29	no	yes
87-61-6	1,2,3-Trichlorobenzene	no	0/44	no	no
96-18-4	1,2,3-Trichloropropane	no	0/44	yes	no
95-63-6	1,2,4-Trimethylbenzene	yes	2/44	no	no
96-12-8	1,2-Dibromo-3-chloropropane	no	0/44	no	no
106-93-4	1,2-Dibromoethane	no	0/44	yes	no
107-06-2	1,2-Dichloroethane	no	0/49	no	no

CAS Number	Chemical	Detected in Surface Sediment	Detection frequency (location)	Identified as COPC in Sediment	Important Bioaccumulative Compound [a]
540-59-0	1,2-Dichloroethene (total)	no	0/2	no	no
78-87-5	1,2-Dichloropropane	no	0/49	no	no
108-67-8	1,3,5-Trimethylbenzene	yes	1/44	no	no
142-28-9	1,3-Dichloropropane	no	0/44	no	no
109-69-3	1-Chlorobutane	no	0/44	no	no
90-12-0	1-Methylnaphthalene	yes	3/3	no	no
832-69-9	1-Methylphenanthrene	yes	3/3	no	no
594-20-7	2,2-Dichloropropane	no	0/44	no	no
60851-34-5	2,3,4,6,7,8-HxCDF	yes	2/29	no	no
57117-31-4	2,3,4,7,8-PeCDF	yes	2/29	no	yes
2245-38-7	2,3,5-Trimethylnaphthalene	yes	3/3	no	no
1746-01-6	2,3,7,8-TCDD	yes	3/29	no	yes
	2,3,7,8-TCDD TEQ	yes	29/29	yes	yes
51207-31-9	2,3,7,8-TCDF	yes	19/29	no	yes
53-19-0	2,4'-DDD	no	0/3	no	no
3424-82-6	2,4'-DDE	no	0/3	no	no
789-02-6	2,4'-DDT	no	0/3	no	no
581-42-0	2,6-Dimethylnaphthalene	yes	3/3	no	no
110-75-8	2-Chloroethyl vinyl ether	no	0/3	no	no
95-49-8	2-Chlorotoluene	no	0/44	no	no
591-78-6	2-Hexanone	no	0/49	no	no
91-57-6	2-Methylnaphthalene	yes	87/557	no	no
2531-84-2	2-Methylphenanthrene	yes	3/3	no	no
	3-Methylphenol and 4-Methylphenol coelution	yes	15/276	no	no
99-09-2	3-Nitroaniline	no	0/517	no	no
106-43-4	4-Chlorotoluene	no	0/44	no	no
67-64-1	Acetone	yes	3/49	no	no
	Acid volatile sulfides	yes	46/56	no	no

CAS Number	Chemical	Detected in Surface Sediment	Detection frequency (location)	Identified as COPC in Sediment	Important Bioaccumulative Compound [a]
107-05-1	Allyl Chloride	no	0/44	no	no
7429-90-5	Aluminum	yes	450/450	yes	no
7664-41-7	Ammonia	yes	18/18	no	no
37324-23-5	Aroclor-1262	yes	2/2	no	no
11100-14-4	Aroclor-1268	yes	1/1	no	yes
7440-39-3	Barium	yes	430/430	no	no
71-43-2	Benzene	yes	1/49	no	no
192-97-2	Benzo(e)pyrene	yes	3/3	no	no
56832-73-6	Benzofluoranthenes (total-calc'd)	yes	511/550	no	no
7440-41-7	Beryllium	yes	449/459	no	no
92-52-4	Biphenyl	yes	2/2	no	no
39638-32-9	bis(2-chloroisopropyl)ether	no	0/352	no	no
108-86-1	Bromobenzene	no	0/44	no	no
74-97-5	Bromochloromethane	no	0/44	no	no
75-27-4	Bromodichloromethane	no	0/49	no	no
75-25-2	Bromoform	no	0/49	no	no
74-83-9	Bromomethane	no	0/49	no	no
	Butyltin (total)	yes	33/44	no	no
7440-70-2	Calcium	yes	429/429	no	no
75-15-0	Carbon disulfide	yes	16/49	no	no
56-23-5	Carbon tetrachloride	no	0/49	no	no
107-14-2	Chloroacetonitrile	no	0/2	no	no
108-90-7	Chlorobenzene	no	0/49	no	no
75-00-3	Chloroethane	no	0/49	no	no
67-66-3	Chloroform	no	0/49	no	no
74-87-3	Chloromethane	no	0/49	no	no
2921-88-2	Chlorpyriphos	no	0/3	no	yes
18540-29-9	Chromium VI	yes	1/8	no	yes

CAS Number	Chemical	Detected in Surface Sediment	Detection frequency (location)	Identified as COPC in Sediment	Important Bioaccumulative Compound [a]
218-01-9	Chrysene	yes	529/557	no	yes
156-59-2	cis-1,2-Dichloroethene	no	0/47	no	no
10061-01-5	cis-1,3-Dichloropropene	no	0/49	no	no
5103-73-1	cis-Nonachlor	no	0/3	no	no
57-12-5	Cyanide	no	0/4	no	no
99-87-6	Cymene	yes	3/44	no	no
132-65-0	Dibenzothiophene	yes	3/3	no	no
124-48-1	Dibromochloromethane	no	0/49	no	no
74-95-3	Dibromomethane	no	0/44	no	no
75-71-8	Dichlorodifluoromethane	no	0/8	no	no
75-09-2	Dichloromethane	yes	1/49	no	no
60-29-7	Diethyl ether	no	0/44	no	no
115-29-7	Endosulfan	no	0/45	no	no
53494-70-5	Endrin ketone	yes	1/56	no	no
97-63-2	Ethyl Methacrylate	no	0/44	no	no
100-41-4	Ethylbenzene	yes	1/49	no	no
8006-61-9	Gasoline	no	0/8	no	no
74-88-4	Iodomethane	no	0/44	no	no
7439-89-6	Iron	yes	448/448	yes	no
98-82-8	iso-Propylbenzene	no	0/44	no	no
	Lube Oils	no	0/8	no	no
7439-95-4	Magnesium	yes	439/439	no	no
7439-96-5	Manganese	yes	445/445	yes	no
126-98-7	Methacrylonitrile	no	0/44	no	no
96-33-3	Methyl Acrylate	no	0/44	no	no
78-93-3	Methyl ethyl ketone	yes	17/49	no	no
108-10-1	Methyl iso-butyl ketone	no	0/49	no	no
80-62-6	Methyl Methacrylate	no	0/44	no	no

CAS Number	Chemical	Detected in Surface Sediment	Detection frequency (location)	Identified as COPC in Sediment	Important Bioaccumulative Compound [a]
2385-85-5	Mirex	no	0/3	no	yes
104-51-8	n-Butylbenzene	no	0/44	no	no
2406-65-7	n-Butyltin	yes	54/80	no	no
103-65-1	n-Propylbenzene	no	0/44	no	no
3268-87-9	OCDD	yes	29/29	no	no
39001-02-0	OCDF	yes	28/29	no	no
27304138	Oxychlordane	no	0/3	no	no
37680-73-2	PCB-101	yes	524/581	no	yes
32598-14-4	PCB-105	yes	415/578	no	yes
38380-03-9	PCB-110	yes	269/304	no	no
74472-37-0	PCB-114	yes	6/276	no	no
31508-00-6	PCB-118	yes	479/582	no	yes
65510-44-3	PCB-123	no	0/276	no	no
57465-28-8	PCB-126	yes	11/582	no	yes
38380-07-3	PCB-128	yes	324/578	no	yes
35065-28-2	PCB-138	yes	514/583	no	yes
35065-27-1	PCB-153	yes	529/580	no	yes
38380-08-4	PCB-156	yes	231/580	no	yes
69782-90-7	PCB-157	yes	71/578	no	no
52663-72-6	PCB-167	yes	44/276	no	no
32774-16-6	PCB-169	no	0/580	no	yes
35065-30-6	PCB-170	yes	448/583	no	yes
37680-65-2	PCB-18	yes	85/264	no	yes
35065-29-3	PCB-180	yes	482/583	no	yes
52663-68-0	PCB-187	yes	235/279	no	yes
39635-31-9	PCB-189	yes	29/580	no	no
52663-78-2	PCB-195	yes	41/279	no	yes
40186-72-9	PCB-206	yes	52/279	no	yes

CAS Number	Chemical	Detected in Surface Sediment	Detection frequency (location)	Identified as COPC in Sediment	Important Bioaccumulative Compound [a]
2051-24-3	PCB-209	yes	15/279	no	yes
7012-37-5	PCB-28	yes	155/279	no	yes
41464-39-5	PCB-44	yes	190/279	no	yes
35693-99-3	PCB-52	yes	3/3	no	yes
35693-99-3	PCB-55	yes	204/276	no	no
32598-10-0	PCB-66	yes	188/279	no	yes
32598-13-3	PCB-77	yes	20/583	no	yes
34883-43-7	PCB-8	yes	1/3	no	yes
70362-50-4	PCB-81	no	0/276	no	yes
	PCBs + PCTs (total)	yes	301/304	no	no
	PCTs (total)	yes	265/306	no	no
76-01-7	Pentachloroethane	no	0/44	no	no
198-55-0	Perylene	yes	3/3	no	no
104-40-5	Phenol, 4-Nonyl-	no	0/3	no	no
7440-09-7	Potassium	yes	439/439	no	no
483-65-8	Retene	yes	3/11	no	no
135-98-8	sec-Butylbenzene	no	0/44	no	no
7782-49-2	Selenium	yes	269/454	no	no
7440-21-3	Silicon	yes	3/3	no	no
7440-23-5	Sodium	yes	431/431	no	no
100-42-5	Styrene	no	0/49	no	no
	Sulfides (total)	yes	42/76	no	no
1634-04-4	Tert-butyl methyl ether	no	0/44	no	no
98-06-6	tert-Butylbenzene	no	0/44	no	no
1461-25-2	Tetrabutyltin as ion	yes	7/92	no	no
127-18-4	Tetrachloroethene	yes	2/49	no	no
109-99-9	Tetrahydrofuran	no	0/2	no	no
7440-28-0	Thallium	yes	302/458	yes	no

CAS Number	Chemical	Detected in Surface Sediment	Detection frequency (location)	Identified as COPC in Sediment	Important Bioaccumulative Compound [a]
7440-31-5	Tin	yes	185/279	no	no
7440-32-6	Titanium	yes	3/3	no	no
108-88-3	Toluene	yes	5/49	no	no
	Total HPAH (calc'd)	yes	544/557	no	no
	Total LPAH (calc'd)	yes	522/557	no	no
68334-30-5	TPH - Diesel #2 Range	no	0/8	no	no
	TPH - Diesel Range	yes	2/2	no	no
	TPH - Gasoline Range	no	0/2	no	no
	TPH - Heavy Fuel Oil Range	yes	2/2	no	no
156-60-5	trans-1,2-Dichloroethene	no	0/47	no	no
10061-02-6	trans-1,3-Dichloropropene	no	0/49	no	no
110-57-6	trans-1,4-Dichloro-2-butene	no	0/42	no	no
39765-80-5	Trans-Nonachlor	no	0/3	no	no
79-01-6	Trichloroethene	no	0/49	no	no
75-69-4	Trichlorofluoromethane	no	0/47	no	no
108-05-4	Vinyl acetate	no	0/3	no	no
75-01-4	Vinyl chloride	no	0/49	no	no
108-38-3/106-42-3	Xylene (meta & para)	yes	1/47	no	no
95-47-6	Xylene (ortho)	yes	1/47	no	no
1330-20-7	Xylene (total)	no	0/2	no	no

[a] From: EPA. 2000a. Bioaccumulation testing and interpretation for the purpose of sediment quality assessment. Status and needs. EPA 823-R-00-001. US Environmental Protection Agency, Office of Water and Office of Solid Waste, Washington, DC.

Subappendix B.3 Analysis of Exposure Frequency and Exposure Duration for Muckleshoot Tribe Net Fishers

This text was prepared by Lon Kissinger, EPA Region 10, in January 2002, in support of the Phase 1 HHRA.

INTRODUCTION

Risks to Muckleshoot Tribe gill net fishers via sediment exposure are to be assessed as part of the HHRA for the Lower Duwamish River. The exposure frequency, or number of days per year that gill net fishers use the Duwamish River as well as the exposure duration, or number of years individuals may fish on the Duwamish River, are required to compute the aforementioned risk. These values were derived from information provided by Mike Mahovlich, Muckleshoot Tribe Assistant Harvest Manager (TAHM). A transcript of the interview is available. According to EPA policy on RME, the exposure frequency and duration should be set at the 95th percentile if data are available, and a reasonable maximum estimate (often the 90th percentile) if data are not available. Since we are dealing with data derived directly from Muckleshoot Tribe members, this analysis will use the 95th percentile criterion.

EXPOSURE FREQUENCY

Methodology

The 95th percentile exposure frequency was derived on the basis of the number of fishers in the group with the maximum number of days on the river. Though it was impossible for the TAHM to identify the fishing frequency associated with each fisher, the TAHM confidently estimated that 5 to 10 fishers generally pursue all Duwamish River salmon runs full time. The total number of Muckleshoot tribe net fishers on the Duwamish is estimated to be between 50 and 60 fishers. This "all run" fishing group therefore constitutes at a minimum, 8.3% of all tribal fishers (5 all run fishers / 60 total fishers). At a maximum, the "all run" fishing group constitutes 20% of all tribal fishers (10 "all run" fishers / 50 total fishers). Since the "all run" fishers make up more than 5% of all tribal fishers, the fishing frequency of the "all run" group encompasses the 95th percentile of the tribal fishing distribution.

The following formula was used to derive season length:

$$\Sigma[((\text{Run end date}) - (\text{Run start date})) * (\text{days per week fished}) / 7]$$

Table 1 gives these parameters for the different Duwamish River salmon runs. The sum of fishing effort for all runs, 123 days, is the exposure frequency for "all run" fishers. This total is greater than the length of the fishing season reported by the Muckleshoot Tribe over the past three years. These years and season lengths were

2000/2001—119 days, 1999/2000—120 days, and 1998/1999—112 days. The reason for differences between recorded season lengths and the season length analysis primarily results from fluctuations in the length of the Winter Steelhead season (Glen St. Amant, personal communication).

Table 1: Salmon run data and fishing season lengths

Species	Start Date	End Date	Run Length	Fishing Effort in Days per Week	Exposure Frequency Based on Run Length Adjusted for Fishing Effort
Summer Steelhead	6/1/01	7/4/01	33	7	33
Chinook	8/7/01	8/8/01	1	7	1
Chinook	8/15/01	8/16/01	1	7	1
Coho (test fishery)	9/5/01	9/20/01	15	1	2
Coho	9/7/01	11/9/01	63	5	45
Chum	11/11/01	12/2/01	21	3	9
Winter Steelhead	12/2/01	12/25/01	23	5	16
	12/26/01	2/01/02	37	3	16
				Sum:	123

Recommendation

It is recommended that the most recent recorded season length (2001) be used to characterize exposure frequency. This value is 119 days.

EXPOSURE DURATION

Methodology

Tribal members engaging in traditional gill net fishing may begin in their late teens and continue on into their sixties (Table 2).

Table 2. Estimated demographics of Muckleshoot net fishers

Approximate # of Full Time Net Fishers	Age Range	Approximate Duration of Angling to Date in Years
5 to 6	50 to 65	30
5 to 6	40 to 50	20 to 25
10	30 to 40	15 to 20
5	20 to 30	5 to 10

The oldest individual currently fishing commercially is 63 years of age. The TAHM identified five to six anglers that were in the 50 to 65 age range. Using the rationale described in section 2.1, the 50 to 65 year-old age group constitutes greater than 5% of the total population. Consequently, the exposure duration for this group encompasses the 95th percentile of the exposure duration distribution. It was not possible to

ascertain when these individuals started fishing. If these individuals started fishing at age 16, the exposure duration associated with this group ranges between 34 to 47 years. The TAHM noted that a number of individuals in this group of older fishers were physically fit and intended to continue to pursue fishing activity.

Recommendation

A plausible value approximating a 95th percentile exposure duration is 44 years. This is based on a tribal fisher that begins fishing at age 16 and ends fishing at age 60. The recommended value was used in the Phase 1 HHRA.

Subappendix B.4 Analysis of Muckleshoot Tribe Demographics Data

This text was prepared by Lon Kissinger, EPA Region 10, in January 2002, in support of the Phase 1 HHRA.

INTRODUCTION

Rarely is actual exposure duration data available for an exposed population. Consequently, exposure duration in Superfund risk assessments is often represented using a surrogate measure of exposure duration, such as an upper percentile estimate of the length of time a household spends in a particular residence (EPA, 1989). The basis for use of residential occupancy period as an estimate of exposure duration is that it is assumed that relocation of a household ends exposure. Residential occupancy period may not be an appropriate measure of exposure duration for tribes consuming contaminated fisheries resources. For cultural reasons, it has been suggested that tribal members maintain residence in close proximity to reservation lands. Individuals relocating over limited distances will continue to use Usual and Accustomed (U&A) fishing areas. Hence, use of residential occupancy period will potentially underestimate exposure to contaminants via consumption of locally caught fish. A more appropriate surrogate for exposure duration would therefore be the length of time individuals have lived on or near reservation lands. Unfortunately, few data sets from tribes have been available to evaluate reservation lands residence time.

Glen St. Amant, sediment specialist for the Muckleshoot Tribe, provided demographic data on years lived in tribal community (YLC) for the Muckleshoot Tribe. The package included a cover letter describing the data set and issues in its interpretation as well as a hard copy printout from the demographer. The data set included the birth date of the tribal member as well as the self-reported number of years that the individual lived on or near reservation lands.

The purpose of this work is to develop an exposure duration value for a residential seafood consumption exposure scenario using Muckleshoot Tribe YLC data. This data set was analyzed with the assistance of SAIC statistician Dennis Beal.

METHOD FOR DERIVING A RESIDENCE-TIME-BASED EXPOSURE DURATION

The Exposure Factors Handbook (1997) bases residence time on the length of time that a household spends in a particular residence. Similarly, this analysis will attempt to identify a residence time associated with the length of time that a household resides on or near reservation lands. The YLC data set did not identify respondents who were heads of household. A “heads of household” data set was estimated by removing from consideration all records for individuals having ages unlikely to be associated with head of household status (i.e., individuals less than 20, 25 or 30 years of age).

There is some question as to what percentile of the YLC distribution should be used to characterize exposure duration. Some guidance on this point is provided by Risk Assessment Guidance for Superfund, Part A, page 6-22, which states:

> Exposure frequency and duration are used to estimate the total time of exposure. These terms are determined on a site-specific basis. If statistical data are available, use the 95th percentile value for exposure time. In the absence of statistical data (which is usually the case), use reasonable maximum estimates of exposure time. National statistics are available on the upper-bound (90th percentile) and average (50th percentile) number of years spent by individuals at one residence (EPA 1989d). Because of the data on which they are based, these values may underestimate the actual time that someone might live in one residence. Nevertheless, the upper-bound value of 30 years can be used for exposure duration when calculating reasonable maximum residential exposures. In some cases, however, lifetime exposure (70 years by convention) may be a more appropriate exposure duration for residential exposures. The exposure frequency and duration selected must be appropriate for the contact rate selected. If a long-term average contact rate (e.g., daily fish ingestion rate averaged over a year) is used then a daily exposure frequency (i.e., 365 days / year should be assumed).

A question remains as to how well the YLC data indicate exposure duration for consumption of contaminated fish and shellfish. Ideally, exposure duration would be based on surveys of the duration of time Muckleshoot tribal members consume fish. The YLC data are a surrogate for the results of such a survey. Nonetheless, seafood consumption is an integral part of tribal culture. Seafood consumption begins at a young age and YLC may be an appropriate indicator of exposure via seafood consumption. These arguments can support choice of the 90th or 95th percentiles of the YLC data.

An additional factor to consider is the quality of the data and potential uncertainty. These points will be addressed in the next section.

The exposure duration for the residential seafood consumption scenario will be based on a 95 percent upper confidence limit on the 90th percentile of the Muckleshoot YLC data. This approach takes into account the fact that a surrogate measure of exposure duration is being used and that a data set with some data quality issues is being employed. The exact methodology used to compute this value will be discussed in the results section.

HISTOGRAMS

Figures 1 and 2 show histograms for YLC.

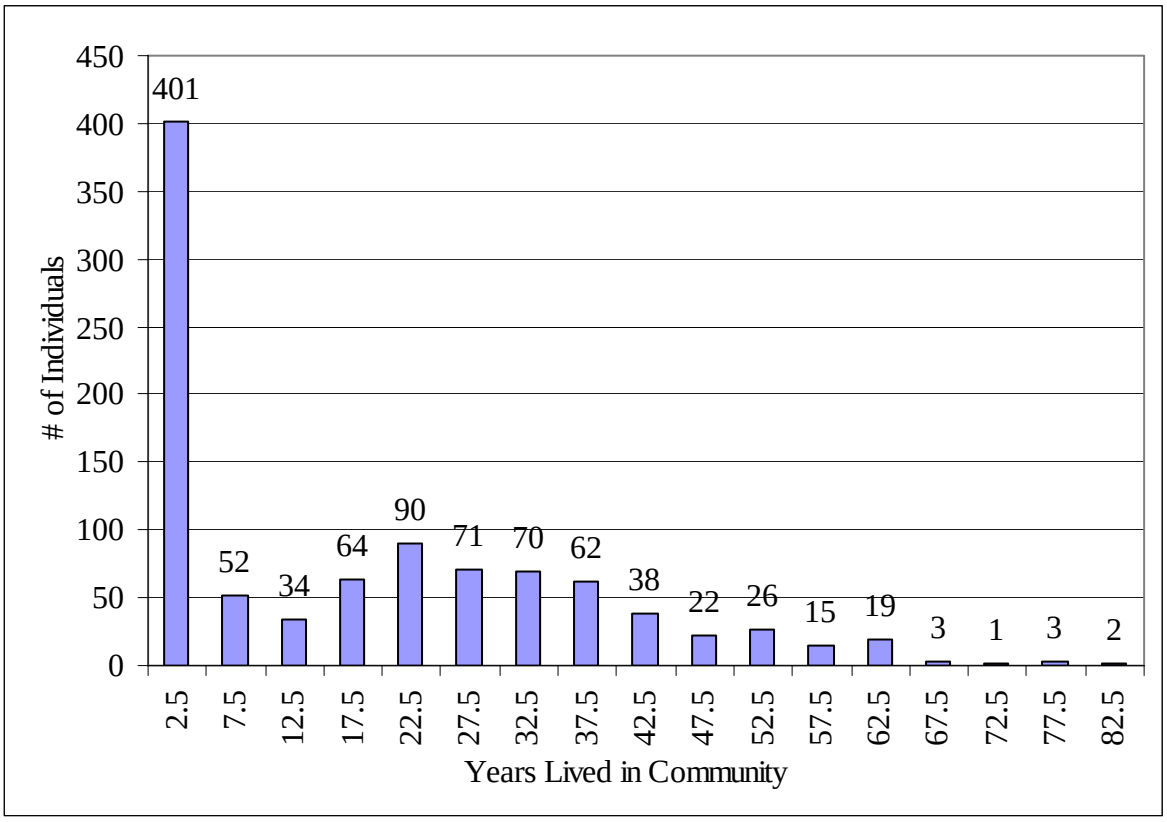

Figure 1. Years lived in community including individuals that reported 0 for YLC

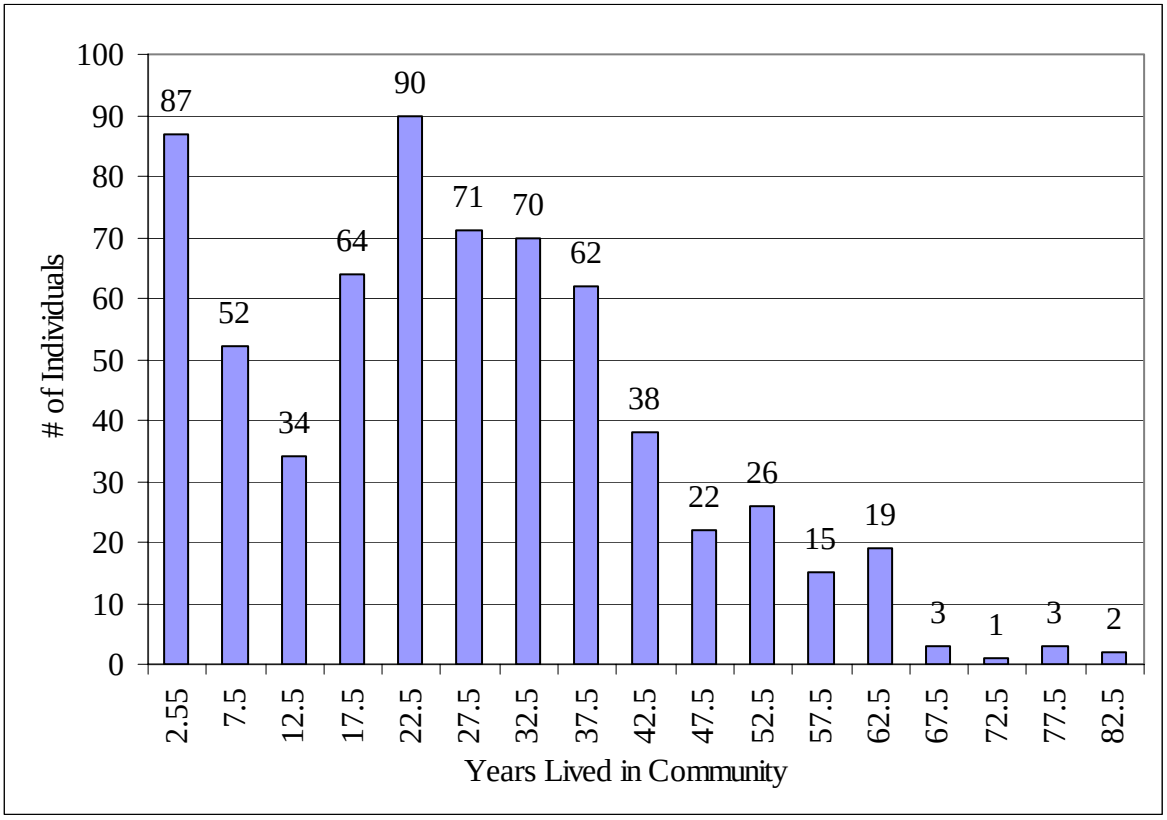

Figure 2. Years lived in community excluding individuals reporting 0 for YLC

IDENTIFICATION, RESOLUTION, AND DISCUSSION OF DATA INTERPRETATION ISSUES

There are a number of considerations that should be taken into account in interpreting the data.

Issue #1: A number of individuals did not answer the question: "How many years have you lived in the tribal community?" The value assigned to YLC in these instances was zero.

Approach to dealing with issue: It is believed that these records do not accurately reflect true YLC values for individuals, and these 314 records have been discarded. This leaves 659 records where individuals reported a non-zero value for YLC. These data are displayed in Figure 2.

Issue #2: Some individuals reported YLC values that were greater than their ages. There are two plausible reasons for this: 1) The individual reported the YLC value for the head of household rather than their own YLC; 2) There was an error in the age or YLC value reported by the individual. A large number of records were associated with this anomaly (184 or 28%). However, only 8 individuals had a difference between YLC

and age of greater than 3 years. This indicates that most of these anomalies were due to misreporting.

Approach to dealing with issue: Individuals reporting YLC values greater than their chronological age were assigned an YLC equal to their chronological age. Rank order nonparametric percentiles were computed for both the raw YLC data and data adjusted such that YLC was set equal to chronological age.[32] The percentiles derived from the raw and adjusted data were compared.

Results and interpretation: It was determined that setting YLC equal to age causes a 2 year decrease in the 90th and 95th percentile YLC values (SEE Table 1). Removal of individuals where YLC values were greater than age caused a 3-year decrease in the 90th and 95th percentile YLC values (SEE Table 2). For purposes of data analysis, it was decided to retain all records with non-zero YLC values and to set YLC equal to age for individuals reporting YLC values greater than age.

Table 1. Effect of setting YLC equal to age on the 90th and 95th percentiles of the YLC distribution

	PERCENTILE	
	90TH	95TH
Unadjusted YLC	50	58
YLC set equal to age for individuals reporting YLC > age	48	56

Table 2. Effect of excluding data where YLC > age on the 90th and 95th percentiles of the YLC distribution

	PERCENTILE	
	90TH	95TH
YLC adjusted by removal of individuals reporting YLC > age	45	53
YLC set equal to age for individuals reporting YLC > age	48	56

Issue #3: There has been an increase in individuals formally enrolling with the tribe as a result of increased tribal economic prosperity. Individuals may have considered themselves to be living in community prior to formally enrolling with the tribe. However, when answering the question: "How many years have you lived in

[32]In addition to derivation of non-parametric percentiles, distributions were fit to the data sets and 90th and 95th percentiles were derived using Monte Carlo simulations. There was close agreement between 90th and 95th percentiles computed using non-parametric and Monte Carlo methods. Results are available upon request.

community?," individuals may be providing the years they have been formally enrolled rather than YLC. The data suggest that this may be occurring. A visual inspection of Figure 2 suggests that this may be true. The frequencies associated with YLC values less than 10 years seem to be higher than the frequencies suggested by a distribution "visually fit" to YLC values greater than 10. If recorded YLC values for late enrollees are lower than actual YLC values, it is possible that the 90th and 95th percentiles of the YLC data are being underestimated.

Approach to dealing with issue: Rank order nonparametric 90th and 95th percentile YLC values were derived from data sets including and excluding YLC data < 10.[33] This permitted comparison of the effect of including and excluding these data.

Results and interpretation: Excluding YLC values less than 10 caused a two-year increase in 90th and 95th percentile YLC values (SEE Table 3). The effect of late enrollment is likely to be overestimated by excluding YLC < 10 records, as there will be some individuals that should be legitimately assigned YLC values less than 10. It was decided to retain records where YLC < 10.

Table 3. Effect of excluding YLC < 10 on the on the 90th and 95th percentiles of the YLC distribution [a]

	PERCENTILE	
	90TH	95TH
YLC < 10 included	48	56
YLC < 10 removed	50	58

[a] YLC values where YLC > age set at age

Issue #4: In the Exposure Factors Handbook (EPA 1997), residence time is estimated based on the duration of time that a household has lived in a residence. It is expected that inclusion of younger individuals will result in 90th and 95th percentile YLC values that are lower than corresponding values obtained using heads of household.

Approach to dealing with issue: The ideal approach would have been to identify heads of household in the Muckleshoot survey. An alternative approach is to remove records for individuals with ages such that they are not likely to be heads of household. Several "head of household" data sets were created by removing records of individuals with ages less than particular age cutoffs (i.e. 20, 25 and 30 years). Non parametric 90th and 95th YLC percentiles were then determined.[3]

[33] In addition to derivation of non-parametric percentiles, distributions were fit to the data sets and 90th and 95th percentiles were derived using Monte Carlo simulations. There was close agreement between 90th and 95th percentiles computed using non-parametric and Monte Carlo methods. Results are available upon request.

Results and interpretation: 90th and 95th percentile YLC values increase as the age cutoff is increased (Table 4). The difference between 90th percentile YLC values and values obtained using a 30 year age cutoff is 5 years.

Table 4. Effect of truncating the YLC data set at different age cutoffs to identify heads of household [a]

Percentile	YLC	YLC where age truncated at		
		20 years	25 years	30 years
90th	48	49	51	53
95th	56	58	58	60

[a] YLC values where YLC > age set at age. YLC < 10 years included

Development of a 95% UCL on the 90TH Percentile Exposure Duration

As noted in the methodology section, the intended statistic to represent exposure duration is the 95% upper confidence limit on the 90th percentile of the YLC distribution. When possible, it is preferable to compute a confidence limit using statistical methods associated with a distribution (e.g. the normal distribution, log normal distribution, etc.). An effort to determine a distribution fitting the existing data was done using quantile-quantile (QQ) plots. A QQ plot correlates the cumulative percentiles of the data distribution (y-axis values) with cumulative percentiles of the parametric distribution for which a fit is being evaluated (e.g. the normal distribution, plotted on the x-axis). If the data distribution fits the test distribution, the points (x = cumulative percentile of the test distribution, y = cumulative percentile of the data distribution) will fall on a straight line. Efforts to find parametric distributions that fit the existing data did not yield satisfactory results. Distributions evaluated included normal, lognormal, beta, and gamma distributions. An example of a QQ plot evaluating a gamma distribution is shown in Figure 3. Since a distribution describing the data cannot be identified, a 95% upper confidence limit on the 90th percentile must be derived using a distribution free or non-parametric method.

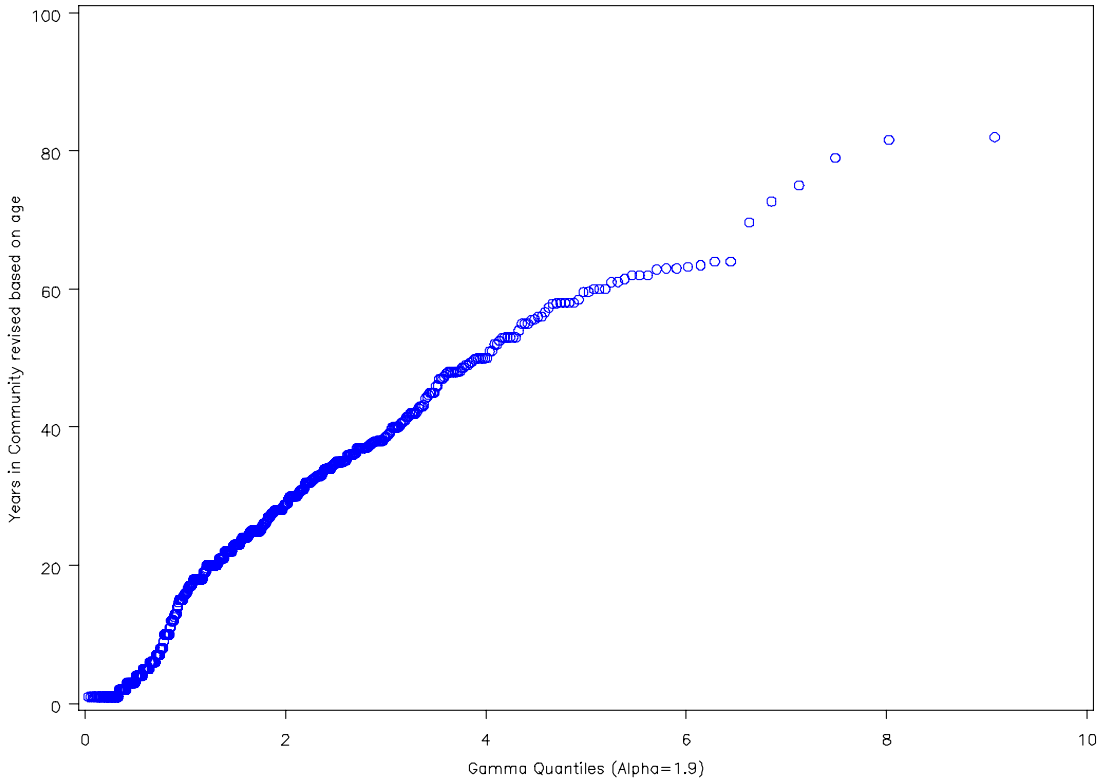

Figure 3. Quantile-Quantile Plot of years in community for Gamma distribution with $\alpha = 1.9$

Hahn and Meeker (1991) provide a good discussion on the calculation and interpretation of these confidence bounds. These distribution free confidence bounds are dependent on sample size *n*. Table A.16 (p. 325) in Hahn and Meeker tabulates these bounds for specific values of *n*. A SAS® program was written which reproduces the results of Table A.16 for any *n*. Using this program produces the exact distribution-free 95% upper confidence bound on the 90th or 95th percentiles. Upper confidence bounds for various data sets and percentiles are given in Table 5.

Table 5. Nonparametric 95% UCLs on percentiles of YLC

Percentile	YLC (n = 659)	YLC where age truncated at:		
		20 (n = 604)	25 (n = 499)	30 (n = 410)
90th	50	53	55	58
95th	60	61	62	63

RECOMMENDED EXPOSURE DURATION

EPA recommends a value of 55 years be used for exposure duration to assess seafood consumption risks for Muckleshoot Tribe members living on reservation lands. This value is based on the 95% distribution-free UCL on the 90th percentile for individuals 25 years or older. The recommended value was used in the Phase 1 HHRA.

REFERENCES

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EPA. 1997. Exposure factors handbook. EPA/600/P-95/002Fa. http://www.epa.gov/ncea/exposfac.htm. Office of Research and Development, US Environmental Protection Agency, Washington, DC.

EPA. 1989. Risk assessment guidance for Superfund: Volume 1: Human health evaluation manual, Part a. EPA/540/1-89/002. Office of Emergency and Remedial Response, US Environmental Protection Agency, Washington, DC.

Subappendix B.5 Toxicological Profiles for Chemicals of Potential Concern

The following sections provide toxicological information for each of the chemicals evaluated in this HHRA. The toxicity values used in this risk assessment (i.e., RfD or SF) are in bold type. Toxicity information was obtained primarily from EPA's Integrated Risk Information System (IRIS; http://www.epa.gov/iris/), EPA's 1997 Health Effects Summary Tables (HEAST), toxicological profiles presented in EPA (2000), EPA's Office of Ground Water and Drinking Water (OGWDW; http://www.epa.gov/OGWDW/hfacts.html), the Agency for Toxic Substance and Disease Registry (ATSDR) ToxFAQs (http://www.atsdr.cdc.gov/toxfaq.html), and the Hazardous Substance Database (HSDB; http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB). Online versions of IRIS, HSDB, ToxFAQs are cited by acronym only in the sections below. These databases were accessed in March and April 2002. Other citations are presented in standard form.

1,2-DIPHENYLHYDRAZINE

1,2-Diphenylhydrazine is a white solid that is only slightly soluble. It adheres to soil and can be carried into the air along with windblown dust. Once in water or exposed to air it is transformed into other chemicals within minutes, including azobenzene and benzidine. 1,2-Diphenylhydrazine is used to make fabric dyes in other countries, and to make certain medicines. There are no other major anthropogenic or natural sources of 1,2-diphenylhydrazine (ATSDR 1990a).

Pharmacokinetics

Pharmacokinetic studies of 1,2-diphenylhydrazine have not been conducted with humans. Soil particles contaminated by this compound may be inhaled or ingested, but it is likely that most of the chemical would be excreted via urine (ATSDR 1990a).

Acute toxicity

The acute health effects of 1,2-diphenylhydrazine in humans have not been studied. Animals die if they swallow large amounts of 1,2-diphenylhydrazine (ATSDR 1990a).

Chronic toxicity

Animals develop liver disease if they eat small amounts of 1,2-diphenylhydrazine for more than a year (ATSDR 1990a). Chronic toxicity data for humans are not available.

Carcinogenicity

EPA has determined that 1,2-diphenylhydrazine is a probable carcinogen (B2) because it causes cancer in rats and mice that have eaten it in food for most of their lifetime. EPA has established an **oral cancer slope factor of 0.8 per mg/kg-d**.

1,2,3-TRICHLOROPROPANE

This compound may be released to the environment in emissions and wastewater as a result of its manufacture, transport, storage, and use as a solvent for oils, fats, waxes, chlorinated rubber, and resins and in the synthesis of some thiokol polysulfide elastomers. It is also found as an impurity in nematicides and soil fumigants and will be released when these products are used. Exposure to 1,2,3-trichloropropane will be primarily occupational via inhalation of vapors and dermal contact. The general population may be exposed to 1,2,3-trichloropropane in drinking water (HSDB).

Pharmacokinetics

This compound appears to metabolized efficiently in mammals. Elimination half-lives of 20 and 120 min for kidney and fat, respectively, have been measured in rats following intravenous administration. A subsequent slower elimination phase for metabolites, with half-lives of 87 to 182 hr, was required, suggesting slower excretion of metabolites as compared with the parent compound (HSDB).

Acute toxicity

Occupational exposure studies have demonstrated that vapors of 1,2,3-trichloropropane were objectionable to all subjects exposed at a concentration of 100 ppm because of eye and throat irritation and unpleasant odor (HSDB).

Chronic toxicity

The effects of chronic human exposure to 1,2,3-trichloropropane are not well documented. EPA has developed a **RfD of 0.006 mg/kg-day** based on chronic studies with both mice and rats. Treatment-related deaths occurred at the 250 mg-kg / day dose level in both mice and rats during the early phase of the study. Other chemical-related findings common to both mice and rats were increased liver and kidney weights accompanied by histopathological changes in the organs. Additionally, rats showed decreased body weight gains, alterations in serum enzymes associated with hepatic and renal toxicity, and decreased red cell mass. An uncertainty factor of 1,000 was applied, which includes a factor of 10 for intraspecies, another factor of 10 for interspecies extrapolation, and another factor of 10 for extrapolating subchronic to chronic exposures.

Carcinogenicity

There is inadequate evidence in humans for the carcinogenicity of 1,2,3-trichloropropane. However, there is sufficient evidence in experimental animals for the carcinogenicity of 1,2,3-trichloropropane. EPA has not conducted a complete evaluation and determination of the carcinogenicity of this compound (IRIS). However, the World Health Organization (IARC 1995) has concluded that this compound is probably carcinogenic to humans (Group 2A). In making the overall evaluation, the WHO working group took into account the following evidence: 1) 1,2,3-trichloropropane causes tumors at multiple sites and at high incidence in mice and rats, 2) the metabolism of 1,2,3-trichloropropane is qualitatively similar in human and rodent microsomes, and 3) 1,2,3-trichloropropane is mutagenic to bacteria and to cultured mammalian cells and binds to the DNA of animals treated in vivo.

2,3,7,8-TCDD (DIOXIN)

TCDD is produced as an unwanted contaminant during the manufacture of chlorobenzenes, chlorophenols and their derivatives. TCDD is the most toxic of 210 polychlorinated dioxin and furan congeners. It is released to the environment primarily through emissions from the incineration of municipal and chemical wastes, in exhaust from automobiles using leaded gasoline, and from the improper disposal of certain chlorinated chemical wastes. The major route of exposure to the general population results from incineration processes and exhausts from leaded gasoline engines. These emissions accumulate in virtually all food products, where they are ingested by humans (HSDB).

Pharmacokinetics

Dioxins are absorbed through the gastrointestinal tract, respiratory tract, and skin and distributed throughout the body. Absorption is congener-specific, with decreased absorption of hepta- and octa-congeners compared with dioxins with fewer chlorines. Because of their lipophilic nature, dioxins tend to accumulate in fat and the liver. Dioxins are slowly metabolized by oxidation or reductive dechlorination and conjugation, and the major routes of excretion are the bile and feces. Reported half lives in the body range from 5 to 15 years. Small amounts may be eliminated in the urine (EPA 2000b).

Acute toxicity

The most commonly reported symptom related to TCDD exposure in humans is chloracne. The lesions of the skin may develop a few weeks after the exposure and may persist for over a year following the cessation of exposure. Other skin problems which have been reported include hyperpigmentation, hirsutism, increased skin fragility, and vesicular eruptions on exposed areas of the skin. Other less consistently reported non-carcinogenic effects from dioxin exposure in humans include asthenia,

headaches, and pain in the extremities, peripheral neuropathy, ulcers, altered liver function, enzyme induction, altered lipid metabolism, and abnormal urinary porphyrin patterns. Immune system dysfunction and altered T-cell subsets have been reported by some investigators but have not been found by others (HSDB).

Chronic toxicity

In animal studies, numerous effects have been documented, including hepatic, gastrointestinal, hematological, dermal, body weight changes, endocrine, immunological, neurological, reproductive, and developmental effects. Most of the studies have involved oral exposure. Despite the variety of adverse effects observed in animals exposed to dioxins, adverse health effects in humans have generally been limited to highly exposed populations in industrial factories or following chemical accidents and contamination episodes. The adverse human health effect most commonly associated with high-level exposure to dioxin-like agents is the skin disease chloracne, a particularly severe and prolonged acne-like skin disorder. Adverse human health effects were also noted following consumption of heated rice oil contaminated with PCBs and CDFs. Conclusive evidence of other adverse human health effects at lower dioxin exposure levels is generally lacking because of incomplete exposure data, concomitant exposure to other compounds, and/or small numbers of study participants. Some epidemiological studies have suggested that dioxins may cause immunosuppression, respiratory effects, cardiovascular effects, and liver effects in humans (EPA 2000b).

Carcinogenicity

There is limited evidence in humans for the carcinogenicity of TCDD, although there is sufficient evidence in experimental animals for the carcinogenicity of TCDD. The World Health Organization (IARC 1995) has concluded that TCDD is carcinogenic to humans (Group 1). In making the overall evaluation, the Working Group took into consideration the following supporting evidence: 1) TCDD is a multi-site carcinogen in experimental animals that has been shown by several lines of evidence to act through a mechanism involving the Ah receptor, 2) this receptor is highly conserved in an evolutionary sense and functions the same way in humans as in experimental animals, and 3) tissue concentrations are similar in both heavily exposed human populations in which an increased overall cancer risk was observed and in rats exposed to carcinogenic dosage regimens in bioassays. EPA has established TCDD as a probable carcinogen (category B2) and has established a **oral cancer slope factor of 150,000 per mg/kg-day**.

3,3'-DICHLOROBENZIDINE

3,3'-Dichlorobenzidine is a gray-to-purple colored crystalline solid. It changes from a solid to a gas very slowly. The salt of this compound is the major form in actual use. Neither 3,3'-dichlorobenzidine nor its salt are found naturally in the environment.

They are manufactured for pigments for printing inks, textiles, plastics and enamels, paint, leather, and rubber. Human exposure may occur in industrial settings via inhalation or direct contact, or in residential settings through contact with contaminated dirt or water.

Pharmacokinetics

When 3,3'-dichlorobenzidine enters the body, very little of it leaves the body unchanged. Over 90% of the parent compound is transformed to metabolites which leave the body, mainly in urine and to a lesser extent in feces, within 72 hours after exposure (ToxFAQs).

Acute toxicity

The salt form of 3,3'-dichlorobenzidine may have caused sore throat, respiratory infections, stomach upset, headache, dizziness, caustic burns, and dermatitis in workers exposed to the chemical. Death has occurred in laboratory animals that ate very high levels of 3,3'-dichlorobenzidine mixed in their food for short periods of time (ToxFAQs).

Chronic toxicity

IRIS does not provide a discussion of chronic effects of exposure to 3,3'-dichlorobenzidine or an RfD. Laboratory animals exposed to moderate levels of 3,3'-dichlorobenzidine mixed with food for a long time suffered mild injury to the liver (ToxFAQs).

Carcinogenicity

Studies show that 3,3'-dichlorobenzidine caused cancer of the liver, skin, breast, bladder, and tissues that form blood and other organs in laboratory animals that ate it in their food (ToxFAQs). There is no evidence that 3,3'-dichlorobenzidine has caused cancer in people who worked with it or who were exposed to it unknowingly or by accident for a short or long time. However, because of the many types of cancer that 3,3'-dichlorobenzidine has caused in different tissues of many types of laboratory animals, 3,3'-dichlorobenzidine has been classified as a probable human carcinogen (B2) with a **oral cancer slope factor of 0.45 per mg/kg-d**.

ALDRIN

Aldrin is the common name for a popular insecticide that was used extensively until 1970, at which time the US Department of Agriculture cancelled all uses. In 1972, however, EPA approved aldrin for killing termites. Use of aldrin to control termites continued until 1987, at which time the manufacturer voluntarily canceled the registration for use in controlling termites. Pure aldrin is a white powder, but technical-grade aldrin (>85% aldrin) is a tan powder. Aldrin slowly evaporates in the air.

Pharmacokinetics

Exposure of the general population to aldrin most likely occurs through eating contaminated food. Exposure of some infants occurs by drinking mother's milk containing aldrin. Studies in animals show that aldrin enters the body quickly after exposure. Once inside the body, aldrin quickly breaks down to dieldrin, where it is stored in lipid reserves.

Acute toxicity

Exposure to very high levels of aldrin for a short time causes convulsions or kidney damage. One very young child died from drinking a solution containing a very high level of dieldrin. Another very young child died after eating food contaminated with aldrin (ToxFAQs). Animal studies have shown that exposure to moderate levels of aldrin for a short time causes decreased ability to fight infections.

Chronic toxicity

Exposure to moderate levels of aldrin for a long time causes headaches, dizziness, irritability, vomiting, or uncontrollable muscle movements. Some sensitive people develop a condition in which aldrin or dieldrin causes the body to destroy its own blood cells (ToxFAQs).

EPA established an **RfD of 0.00003 mg/kg-day** based on observed liver toxicity in a chronic rat feeding study. A composite uncertainty factor of 1,000 encompasses the uncertainty of extrapolation from animals to humans, the uncertainty in the range of human sensitivities, and an additional uncertainty because the RfD is based on a lowest observed adverse effect level (LOAEL) rather than a no observed adverse effect level (NOAEL) (IRIS).

Carcinogenicity

EPA has classified aldrin as a probable human carcinogen (B2) and has established a **oral cancer slope factor of 17 per mg/kg-d** based on observations of significant increases in tumor responses in three different strains of mice in both males and females following aldrin exposure.

ALUMINUM

Aluminum is the most abundant metal and the third most abundant element, after oxygen and silicon, in the earth's crust. It is widely distributed and constitutes approximately 8 percent of the earth's surface layer. However, aluminum is a very reactive element and is never found as free metal in nature. It is found combined with other elements, most commonly with oxygen, silicon, and fluorine. High concentrations in the environment can be caused by the mining and processing of its ores and by the production of aluminum metal, alloys, and compounds. Small

amounts of aluminum are released into the environment from coal-fired power plants and incinerators (ATSDR 1999a).

Pharmacokinetics

Since little aluminum is absorbed, it is excreted in the feces, much of it in the form of aluminum phosphate. There is no generally no increase in the amount of aluminum in tissues, except in bone, as demonstrated in animal experiments. Some aluminum may be absorbed by patients undergoing dialysis; the kidney is responsible for removing the majority of absorbed aluminum (HSDB). Aluminum in lung tissue appears to be derived from inhaled particulates rather than any affinity of lung tissue for aluminum.

Acute toxicity

Low-level exposure to aluminum from food, air, water, or contact with skin is not thought to harm your health (ATSDR 1999a). Aluminum, however, is not a necessary substance for our bodies and too much may be harmful. People who are exposed to high levels of aluminum in air may have respiratory problems including coughing and asthma from breathing dust.

Chronic toxicity

Some studies show that people with Alzheimer's disease have more aluminum than usual in their brains. Data are inconclusive on whether aluminum causes the disease or whether the buildup of aluminum happens to people who already have the disease. Infants and adults who received large doses of aluminum as a treatment for another problem developed bone diseases, which suggests that aluminum may cause skeletal problems. Some sensitive people develop skin rashes from using aluminum chlorohydrate deodorants (ATSDR 1999a).

ATSDR (1999a) has developed a **minimum risk level (MRL), which is equivalent to an RfD, for aluminum of 2 mg/kg-day**. This MRL is based on a neurological endpoint and includes an uncertainty factor of 30.

Carcinogenicity

EPA has not conducted a complete evaluation and determination of the carcinogenicity of aluminum (IRIS). Available data suggest that this element is not carcinogenic (ATSDR 1999b).

ANTIMONY

Antimony is naturally present in the earth's crust. The release of antimony into the environment occurs primarily through anthropogenic sources like non-ferrous metal mining, smelting, refining, and production, the use and disposal of antimony alloys and compounds, coal combustion, and refuse and sludge combustion. Antimony

exposure occurs through inhalation, ingestion of food containing antimony, and through dermal contact (IRIS).

Pharmacokinetics

Antimony is absorbed by erythrocytes and distributed to other tissues such as liver, adrenals, spleen, and thyroid. Much of the absorbed antimony is excreted via urine and feces. Of the antimony that is not excreted, the longest biological half-life is believed to occur in the lungs. The highest concentrations of antimony after acute or chronic oral or parenteral exposure have been found in the thyroid, adrenals, liver, and kidney (HSDB).

Acute toxicity

Violent vomiting, diarrhea, lowered respiratory rate, myocardial edema, hyperemia, and capillary engorgement are major results of acute exposure to antimony. Seventy people became acutely ill after ingesting lemonade containing 0.013% antimony. Fifty-six of the victims were treated for burning stomach pains, colic, nausea, and vomiting. Most recovered after approximately three hours, while some required hospitalization for a few days (IRIS).

Chronic toxicity

Dyspnea, weight and hair loss, popular eruptions on the skin, jaundice, albuminaria, damage to the heart and liver, and spleen, glomerular nephritis, abnormal increase in erythrocytes, and a decrease in leukocytes are reported from long-term exposure to antimony. Chronic inhalation results in interstitial pneumonitis, intra-alveolar lipoid deposits, and liver and cardiac damage (HSDB). EPA developed an **RfD for antimony of 0.0004 mg/kg-day** based on a study in which rats were exposed to potassium antimony tartrate (IRIS).

Carcinogenicity

EPA has not conducted a complete evaluation and determination of the carcinogenicity of antimony (IRIS).

ARSENIC

Arsenic is a naturally occurring element in the earth's crust that is usually found combined with other elements. Arsenic combined with elements such as oxygen, chlorine, and sulfur is referred to as inorganic arsenic; arsenic combined with carbon and hydrogen is referred to as organic arsenic. Arsenic in seafood is more commonly in the organic form (EPA 1997b). Most of the common organic forms, such as arsenobetaine and arsenocholine, are non-toxic, but other forms that may also occur to some extent, such as dimethylated and monomethylated arsenic acids, are more toxic (EPA 1997b). Some seafood may also contain arseno-sugars, which may be metabolized to dimethyl arsenic (Chew 1996).

Pharmacokinetics

Pharmacokinetic studies show that water-soluble arsenic compounds are well absorbed across the gastrointestinal tract. They appear to be transported throughout the body; analysis of tissues taken at autopsy from people who were exposed to arsenic found arsenic present in all tissues of the body. The arsenic levels in hair and nails were the highest, with somewhat lower levels in internal organs (ATSDR 2000a).

The metabolism of arsenic consists mainly of a reduction reaction, which converts pentavalent arsenic to trivalent arsenic, and methylation reactions, which convert arsenite to monomethylarsonic acid and dimethylarsenic acid (EPA 2000b). Recent research suggests that the methylation pathway may increase arsenic toxicity (Petrick et al. 2000, 2001; Thomas et al. 2001). The primary excretion route for arsenic and metabolites is in the urine, with human studies showing that 45 to 85 percent is excreted in the urine within 1 to 3 days. Very little is excreted in the feces (ATSDR 2000a).

Acute toxicity

Arsenicals have been recognized as a human poison since ancient times, and large doses, approximately 600 µg/kg-d or higher, taken orally have resulted in death (EPA 2000b). Oral exposure to lower levels of arsenic has resulted in effects on the gastrointestinal system (nausea, vomiting); central nervous system (headaches, weakness, delirium); cardiovascular system (hypotension, shock); and the liver, kidney, and blood (anemia, leucopoenia). Because significant information is available on the acute effects of arsenic poisoning in humans, few animal studies have been carried out. The limited available data have shown arsenic to have low to moderate acute toxicity to animals, based on LD50s between 50 and 5,000 mg/kg (ATSDR 2000a).

Chronic toxicity

The primary effects noted in humans from chronic exposure to arsenic are effects on the skin. Oral exposure has resulted in a pattern of skin changes that include the formations of warts or corns on the palms and soles, along with areas of darkened skin on the face, neck, and back (EPA 2000b). Blackfoot disease, a disease characterized by a progressive loss of circulation in the hands and feet, leading ultimately to necrosis and gangrene, is associated with arsenic (ATSDR 2000a). Other effects noted from chronic oral exposure include peripheral neuropathy, cardiovascular disorders, and liver and kidney disorders.

EPA's IRIS database provides an **RfD for inorganic arsenic of 0.0003 mg/kg-d**, based on a no observed adverse effects level (NOAEL) (adjusted to include arsenic exposure from food) of 0.0008 mg/kg-d and an uncertainty factor of 3. The RfD was based on two studies that showed that the prevalence of blackfoot disease increased with both age and dose for individuals exposed to high levels of arsenic in drinking water. An

uncertainty factor of 3 was used to account for both the lack of data to preclude reproductive toxicity as a critical effect and for uncertainty as to whether the NOAEL of the critical studies accounts for all sensitive individuals (EPA 2000b). EPA has medium confidence in the studies on which the RfD was based and in the RfD. The key studies were extensive epidemiologic reports that examined effects in a large number of people. However, doses were not well-characterized, other contaminants were present, and potential exposure from food and other sources were not fully characterized.

Carcinogenicity

There is clear evidence that chronic exposure of humans to inorganic arsenic increases the risk of cancer. Ingestion of arsenic has been associated with an increased risk of nonmelanoma skin cancer, and bladder, liver, and lung cancer. In addition, studies have reported that inhalation of arsenic results in an increased risk of lung cancer (EPA 2000b). Dimethyl arsenic may be a promoter of various forms of cancer in rats and mice (Kenyon and Hughes 2001). EPA has classified inorganic arsenic in Group A – Known Human Carcinogen, based on the increased incidence in humans of lung cancer through inhalation exposure and the increased risk of skin, bladder, liver, and lung cancer through drinking water exposure.

The **oral cancer slope factor for arsenic is 1.5 per kg-d/mg** (EPA 2000b). EPA used data from Taiwan concerning skin cancer incidence, age, and level of exposure via drinking water. In 37 villages that had obtained drinking water for 45 years from artesian wells with various elevated levels of arsenic, 40,421 individuals were examined for hyperpigmentation, keratosis, skin cancer, and blackfoot disease. The local well waters were analyzed for arsenic, and the age-specific cancer prevalence rates were correlated with both local arsenic concentrations and duration of exposure.

BARIUM

Barium metal does not occur in nature. The most common barium ores are sulfate, barite, carbonate, and witherite. The largest use of barium is in the removal of traces of gases from vacuum and television picture tubes. Barium is released into the environment through the disposal of drilling waste, copper smelting, manufacture of motor vehicle parts, combustion of coal and oil, and the mining, refining, and production of barium and barium-based chemicals (OGWDW).

Pharmacokinetics

The human body contains approximately 22 mg of barium, 66% of which is in the bones. Common routes of exposure are ingestion, inhalation of dust or fumes, and skin or eye contact (HSDB).

Acute toxicity

Exposure to large quantities of barium can cause gastrointestinal disturbances and muscular weakness. No Health Advisories have been established for short-term exposure to barium (OGWDW).

Chronic toxicity

Chronic exposure to barium can cause hypertension (OGWDW). Populations with pulmonary diseases are especially at risk. Barium is not considered an industrial health hazard (HSDB). EPA has established an **oral RfD for elemental barium of 0.07 mg/kg-day**.

Carcinogenicity

No suitable bioassays or epidemiological studies are available to assess the carcinogenicity of barium (IRIS). EPA has placed barium in weight-of-evidence group D, not classifiable as to human carcinogenicity.

BENZIDINE

Benzidine may be released as emissions and in wastewater during its production and use as an intermediate in the manufacture of direct azo dyes. Large-scale manufacturing of benzidine in the US has been suspended since 1976. It is now produced in the US for domestic consumption only with strict regulations that it be maintained in isolated or closed systems that would limit its release (HSDB). Exposure to benzidine is primarily occupational via dermal adsorption, inhalation, and ingestion in workers connected with its production and conversion into direct azo dyes. The respiratory route is of major importance under some manufacturing conditions.

Pharmacokinetics

Absorbed doses of benzidine are rapidly transferred to the excretory organs, liver, GI tract, kidney, and bladder. Half-lives determined experimentally range from 65 h in rat to 88 h in dogs (HSDB).

Acute toxicity

Ingestion of benzidine may produce nausea, vomiting, liver, and kidney damage (HSDB).

Chronic toxicity

Long-term exposure to benzidine has been shown to produce a spectrum of lesions of the epithelium of the urinary bladder, which may precede appearance of malignancy. Presence of visible or occult of blood in urine or the development of pain or difficulty in urinating may signal appearance of such lesions (HSDB). EPA has established an

RfD of 0.003 mg/kg-day based on a chronic oral mouse bioassay (IRIS). An uncertainty factor of 1000 was applied to account for uncertainty in the extrapolation of dose levels from laboratory animals to humans (10X), uncertainty in the threshold for sensitive humans (10X), and uncertainty in the estimation of a NOAEL from a lowest observed adverse effects level (LOAEL) (10X).

Carcinogenicity

EPA has classified benzidine as a known (Class A) carcinogen based on observations of increased incidence of bladder cancer and bladder cancer-related deaths in exposed workers. EPA has established a **oral cancer slope factor of 230 per mg/kg-day**.

BIS(2-CHLOROETHYL)ETHER

Bis(2-chloroethyl) ether (BCEE) is a colorless nonflammable liquid with a strong, unpleasant odor. It does not occur naturally, but is manufactured for use in the production of pesticides and other chemicals. Limited amounts of BCEE will dissolve in water, and it also will slowly evaporate into air. In the environment, BCEE is broken down by bacteria in soil and water and by chemical reactions in the air, so it does not tend to persist for long periods (ToxFAQs).

Pharmacokinetics

BCEE enters the body easily after being swallowed in food or water, or after being inhaled in air. It may also enter by crossing the skin when dermal contact occurs. Once inside the body, BCEE is broken down to a number of different chemicals, and these are eliminated in the urine or the breath. Most BCEE that enters the body is removed in this way within two to three days, so BCEE does not tend to bioaccumulate (ToxFAQs).

Acute toxicity

People exposed to BCEE vapors report that it is highly irritating to the eyes and the nose. Animal studies show that BCEE vapors can cause severe injury to the lungs, and may lead to death (ToxFAQs).

Chronic toxicity

The chronic effects of BCEE on other organs (besides the lung) and body functions have not been well studied. It is not known if BCEE impairs reproduction or the development of fetuses (ToxFAQs). EPA has not established an RfD for BCEE.

Carcinogenicity

Mice given repeated doses of BCEE through the mouth developed liver tumors. This suggests that BCEE might cause cancer in humans, although no cases of cancer due to BCEE have been reported in people and BCEE was also not found to induce excess cancer after feeding to rats. EPA has classified BCEE as a probable human carcinogen

(B2) and has established a **oral cancer slope factor of 1.1 per mg/kg-d** based on positive carcinogenicity results in two strains of mice and evidence of mutagenicity (IRIS).

BIS(2-CHLOROISOPROPYL) ETHER

Bis(2-chloroisopropyl) ether is used primarily as a solvent in the manufacture of fats, waxes, and greases; as an extractant; in paint and varnish removers; in spotting and cleaning solutions; and in textile processing (HSDB). There is no evidence of commercial production of this compound within the US.

If released to water or moist soil, bis(2-chloroisopropyl) ether will hydrolyze rapidly based on an estimated hydrolysis half-life of <38.4 sec in water. Therefore, biodegradation, bioconcentration in aquatic organisms and adsorption to soil and sediment are not expected to be significant fate processes (HSDB).

Pharmacokinetics

After single oral doses, bis-chloroisopropyl ether appeared to be readily absorbed by both female rats and monkeys (HSDB). With respect to the percentage of the radiolabeled administered dose recovered in the tissues and excreta, higher amounts of radioactivity were found in the fat (1.98%), urine (63.36%), feces (5.87%), and expired air (15.96%) of the rat compared to the monkey. The corresponding figures in the monkey were 0.78%, 28.61%, 1.19%, and 0%.

Acute toxicity

The acute toxicity of bis(2-chloroisopropyl) ether is not well-studied. Studies with rats exposed to an atmosphere saturated with bis(2-chloroisopropyl) ether exhibited signs of immediate eye irritation and incoordination; the maximum exposure time causing no death was 1 hr. When rats were exposed to 700 ppm, deaths occurred after 6 hr of exposure. Autopsy revealed slight lung irritation and moderate to severe liver damage (HSDB).

Chronic toxicity

EPA has established an **RfD of 0.04 mg/kg-d** based a chronic oral study with mice that documented a decrease in hemoglobin and possible erythrocyte destruction (IRIS). A 100-fold uncertainty factor was applied to account for both interspecies and interhuman variability in the toxicity of this chemical in lieu of specific data. An additional uncertainty factor of 10 was applied to account for data gaps in the studies on bis-chloroisopropyl ether.

Carcinogenicity

There is limited evidence in experimental animals for the carcinogenicity of bis(2-chloroisopropyl) ether. The IARC (1995) indicated the carcinogenicity of this chemical

to humans is not classifiable (Category 3). IRIS does not address the carcinogenicity of bis-chloroisopropyl ether.

BIS(2-ETHYLHEXYL)PHTHALATE

Bis(2-ethylhexyl)phthalate (BEHP) is a man-made chemical that is commonly added to plastics to make them flexible. This compound is present in plastic products such as rainwear, footwear, upholstery materials, imitation leather, waterproof gloves, tablecloths, shower curtains, food packaging materials, floor tiles, and children's toys. It can be an ingredient in paints, flexible tubing, plastic bags, containers for blood, printing inks, pesticides, cosmetics, and vacuum pump oil and can be used for testing air filtration systems.

Pharmacokinetics

Small amounts of BEHP may enter your body by skin contact with plastics, but most evidence indicates that very little enters this way (ToxFAQs). Most BEHP that enters the body in food, water, or air is taken up into the blood from the intestines and lungs. After BEHP is absorbed into your body, most of it is rapidly broken down to mono(ethylhexyl)phthalate (MEHP) and 2-ethylhexanol. The toxicities of MEHP and 2-ethylhexanol are similar to the toxicity of BEHP. These compounds travel through the bloodstream to the liver, kidneys, and testes, and small amounts will become stored in fat or secreted in breast milk. Most of the BEHP, MEHP, and 2-ethylhexanol leaves your body within 24 hours in the urine and feces.

Acute toxicity

BEHP appears to affect rats and mice more than it affects humans and some other animals. Short-term exposures to high levels of BEHP interfered with sperm formation in mice and rats. These effects were reversible, but sexual maturity was delayed when the animals were exposed before puberty. Short-term exposures appeared to have no effect on male fertility (ToxFAQs).

Chronic toxicity

Long-term exposure of rats to BEHP resulted in structural and functional changes in the kidney. The structural kidney changes seen in rats are similar to those in the kidneys of long-term dialysis patients (ToxFAQs).

EPA has established an **RfD of 0.02 mg/kg-d** based on a subchronic to chronic bioassay with guinea pig that documented increased relative liver weight (IRIS). Uncertainty factors of 10 each were used for interspecies variation and for protection of sensitive human subpopulations. An additional factor of 10 was used since the guinea pig exposure was longer than subchronic but less than lifetime, and because, while the RfD was set on a LOAEL, the effect observed was considered to be minimally adverse.

Carcinogenicity

EPA has classified BEHP as a probable human carcinogen (B2) and has established a **oral cancer slope factor of 0.014 per mg/kg-d** based on observations of significant dose-related increases in liver tumor responses in rats and mice of both sexes (IRIS).

CADMIUM

Cadmium is a heavy metal that is released through a wide variety of industrial and agricultural activities. The accumulation of cadmium in human and other biological tissue has been evaluated in both epidemiological and toxicological studies. ATSDR (1993a) has determined that exposure conditions of most concern are long-term exposure to elevated levels in the diet.

Pharmacokinetics

Cadmium is not readily absorbed when exposure occurs via ingestion. Absorption may be much higher in iron-deficient individuals. Evaluations of the impact of cadmium complexation indicate that cadmium absorption from food is not dependent upon chemical complexation. Some populations with high dietary cadmium intakes have elevated blood cadmium levels, which could be due to the particular forms of cadmium in their food (ATSDR 1993a).

Cadmium is not directly metabolized, but absorption appears to involve sequestering by metallothionein, and plasma cadmium is found primarily bound to this protein. This type of binding appears to protect the kidney. It is thought that kidney damage by cadmium occurs primarily due to unbound cadmium (ATSDR 1993a). Once cadmium is absorbed, it is eliminated slowly; the biological half-life has been estimated at 10 to 30 years (FDA 1993a).

Acute toxicity

Effects of acute oral exposure to cadmium include GI irritation, nausea, vomiting, abdominal pain, cramps, salivation, and diarrhea. Lethal doses in humans caused massive fluid loss, edema, and widespread organ destruction. The ingested doses were 25 and 1,500 mg/kg (ATSDR 1993a).

Chronic toxicity

Kidney toxicity is the main concern with cadmium exposure, with the critical effect being significant proteinuria (an indicator of kidney toxicity). The **RfD for cadmium in food was calculated to be 0.001 mg/kg-d**. The RfD was calculated using a toxicokinetic model to determine the highest level of cadmium in the human renal cortex not associated with significant proteinuria (EPA 2000b).

Cadmium causes many other types of toxic effects in addition to nephrotoxicity, such as reducing the gastrointestinal uptake of iron, bone disorders, and increased calcium excretion. Some human studies have shown cardiovascular toxicity and elevated

blood pressure, but the results are conflicting (ATSDR 1993a). In addition, animal studies indicate that cadmium causes a wide variety of alterations in the function of the immune system.

Carcinogenicity

No animal or human oral exposure studies suggest that cadmium is carcinogenic via the oral exposure route, although cadmium is classified as a probable human carcinogen (B1) by EPA based on inhalation studies in humans (EPA 2000b). ATSDR has concluded that there is minimal evidence of an association between cadmium exposure and increased cancer risk in humans but that the statistical power of the studies examined to detect an effect was not high. They determined that neither the human nor the animal studies provided enough evidence to agree on the carcinogenic status of cadmium (ATSDR 1993a).

CARCINOGENIC POLYCYCLIC AROMATIC HYDROCARBONS (PAHS)

PAHs are a group of organic chemicals that have a fused ring structure of two or more benzene rings, and are formed during the incomplete combustion of organic materials. Industrial activities which produce PAHs include: coal coking, production of carbon blacks, creosote, coal tar, petroleum refining, synfuel production from coal, and the use of Soderberg electrodes in aluminum smelters and ferrosilicum and iron works (EPA 2000b). Domestic activities which produce PAHs include: cigarette smoke, burning of wood and fossil fuels, waste incineration, broiling and smoking foods, and the use of combustion engines. Benzo(a)pyrene is the PAH with the most available health effects data.

Pharmacokinetics

PAHs can be absorbed through the lungs, the stomach, or the skin. Oral absorption increases with more lipophilic PAHs or in the presence of oil in the GI tract. Upon inhalation, oral or dermal exposure of animals, the highest levels of PAHs were found in highly perfused tissues, such as the lung, liver, GI tract and kidneys. It has been demonstrated that PAHs metabolize to reactive intermediates by enzyme systems, which then covalently bind to cellular macromolecules leading to mutation and tumor development (EPA 2000b).

Acute toxicity

There are little data describing acute toxicity of PAHs after inhalation, oral, or dermal exposure in humans or animals. However, benzo(a)pyrene is fatal to mice following ingestion, and the liver and the skin have been identified as target organs in animals after oral or dermal exposure, respectively (ATSDR 1995). The intraperitoneal LD50 values in mice for pyrene, anthracene, and benzo(a)pyrene are 514, >430, and 232 mg/kg, respectively.

Chronic toxicity

PAHs have a high chronic exposure toxicity characterized by chronic dermatitis and hyperkeratosis (ATSDR 1995). Chronic studies in animals exposed to PAHs ingestion, intratracheal installation, or skin-painting have as yet not identified adverse health effects other than cancer. RfDs have not been developed for any of the PAHs being evaluated in this Phase 1 HHRA.

Carcinogenicity

Occupational studies of workers exposed to mixtures containing PAHs have shown that mixtures of PAHs are carcinogenic to humans. Cancer associated with exposure to PAH containing mixtures in humans occurs mainly in the lung and skin following inhalation and dermal exposure.

The EPA and others have developed a relative potency estimate approach for PAHs, based on their potency relative to benzo(a)pyrene (EPA 1993). The oral cancer slope factor developed by EPA for carcinogenicity of benzo(a)pyrene is **7.3 per mg/kg-d**. This cancer potency factor was applied to the sum of cPAHs, using the TEFs described in Section B.2.3.

CHLORDANE

Chlordane is an organochlorine insecticide comprised of the sum of cis- and trans-chlordane and trans-nonachlor and oxychlordane for purposes of health advisory development. First introduced in 1947, it was used extensively on agricultural crops, livestock, lawns, and for termite control. Because of concern over cancer risk, human exposure, and effects on wildlife, most uses were banned in 1978, and all uses were banned by 1988. Due to its long half-life and ability to concentrate in biological materials, it is still widely distributed in fish in the United States (EPA 2000b).

Pharmacokinetics

Chlordane is extremely lipid soluble, and lipid partitioning of chlordane and its metabolites has been documented in both humans and animals. Chlordane is metabolized via oxidation, which results in a number of metabolites, including oxychlordane, that are very persistent in body fat. Human studies have found chlordane in pesticide applicators, residents of homes treated for termites, and those with no known exposures other than background (EPA 2000b).

Acute toxicity

Chlordane is moderately to highly toxic with an estimated lethal dose to humans of 6 to 60 g (IRIS). Effects reported in humans after acute exposure include headaches, irritability, excitability, confusion, incoordination, seizures, and convulsions. There is also some evidence that acute exposures to chlordane may be associated with immunologic dysregulation and aplastic anemia in humans (EPA 2000b).

Chronic toxicity

IRIS provides an **RfD of 0.0005 mg/kg-d** based on a NOAEL of 0.15 mg/kg-d for hepatic necrosis in a 2-yr feeding study in mice (IRIS). The LOAEL in the principal study was 0.75 mg/kg-d. An uncertainty factor of 300 was applied to the NOAEL, 10 each for inter- and intraspecies variability and 3 for lack of any reproductive studies. The confidence in the principal study is rated medium, as is the confidence in the database.

Carcinogenicity

Chlordane is classified as a probable human carcinogen (B2) by EPA based on oral studies in animals. An increased incidence of hepatocellular carcinoma was observed in both sexes in mice in two separate studies using different strains. Hepatocellular carcinomas were also observed in another study in male mice using a third strain. The **oral cancer slope factor of 0.35 per mg/kg-d** is the geometric mean of the cancer potencies calculated from five data sets (IRIS).

CHROMIUM

Trivalent chromium is a naturally occurring chemical with low toxicity. Hexavalent chromium, however, is released into the environment through industrial emissions and is highly toxic due to its strong oxidation characteristics and membrane permeability. Hexavalent chromium is used in chromate manufacturing, ferrochromium industries, and in metal alloys (HSDB).

Pharmacokinetics

Trivalent chromium is an essential ion required for lipid, protein, and fat metabolism and to maintain normal glucose metabolism. The most common routes of exposure to toxic levels of chromium are through inhalation and ingestion (ToxFAQs).

Acute toxicity

The acute toxic effects of hexavalent chromium were studied in 1965 when 155 people were exposed to 20 mg/L hexavalent chromium in their drinking water. The victims suffered from mouth sores, diarrhea, stomachaches, indigestion, vomiting, increased white blood cell counts, and a higher per capita cancer rate. Acute exposure to hexavalent chromium may also affect fetal development. Dermal exposure to hexavalent chromium can cause skin irritation and allergic contact dermatitis (IRIS).

Chronic toxicity

Chronic exposure to chromium can cause damage to the liver, kidney, and circulatory system, as well as cause nerve tissue damage and dermatitis (OGWDW). **EPA has developed RfDs of 1.5 and 0.003 mg/kg-day for trivalent and hexavalent chromium, respectively**. The RfD for hexavalent chromium will be applied to all chromium data

in this HHRA since the proportion of trivalent chromium in the total chromium measurements is not known.

Carcinogenicity

EPA has classified trivalent chromium as Group D, not classifiable as to human carcinogenicity. Hexavalent chromium is a Group A known human carcinogen via the inhalation pathway (IRIS). EPA has not developed an oral cancer potency factor for hexavalent chromium.

COPPER

Copper occurs naturally in elemental form and as a component of many minerals. Because of its high electrical and thermal conductivity, it is widely used in the manufacture of electrical equipment. Common copper salts, such as the sulfate, carbonate, cyanide, oxide, and sulfide are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (Faust 1992). Copper can be absorbed by the oral, inhalation, and dermal routes of exposure.

Pharmacokinetics

Copper is an essential nutrient that is normally present in a wide variety of human tissues (Faust 1992). Copper is incorporated into more than a dozen specific copper proteins, such as cytochrome oxidase, tyrosinase, and erythrocyte superoxide dismutase. Copper is essential for hemoglobin formation, carbohydrate metabolism, catecholamine biosynthesis, and cross-linking of collagen, elastin, and hair keratin (EPA 1987).

Acute toxicity

In humans, ingestion of gram quantities of copper salts may cause gastrointestinal, hepatic, and renal effects with symptoms such as severe abdominal pain, vomiting, diarrhea, hemolysis, hepatic necrosis, hematuria, proteinuria, hypotension, tachycardia, convulsions, coma, and death (Faust 1992). Acute inhalation exposure to copper dust or fumes at concentrations of 0.075-0.12 mg Cu / m^3 may cause metal fume fever with symptoms such as cough, chills, and muscle ache (Faust 1992). Among the reported effects in workers exposed to copper dust are gastrointestinal disturbances, headache, vertigo, drowsiness, and hepatomegaly.

Chronic toxicity

Gastrointestinal disturbances and liver toxicity have resulted from long-term exposure to drinking water containing 2.2-7.8 mg Cu / L (Faust 1992). The chronic toxicity of copper has been characterized in patients with Wilson's disease, a genetic disorder causing copper accumulation in tissues. Vineyard workers chronically exposed to Bordeaux mixture (copper sulfate and lime) exhibit degenerative changes of the lungs

and liver. Dermal exposure to copper may cause contact dermatitis in some individuals (ATSDR 1990b).

EPA has not developed an oral RfD for elemental copper. EPA's HEAST proposed a provisional value of **0.04 mg/kg-day**. Provisional RfDs have greater uncertainty than RfDs certified by EPA.

Carcinogenicity

No suitable bioassays or epidemiological studies are available to assess the carcinogenicity of copper (Faust 1992). EPA has placed copper in weight-of-evidence group D, not classifiable as to human carcinogenicity.

DDT AND METABOLITES

DDT is an organochlorine pesticide that has not been marketed in the United States since 1972 but is ubiquitous due to its widespread use in previous decades and its relatively long half-life. DDT's close structural analogs, DDE and DDD, are metabolites of DDT and have also been formulated as pesticides in the past (EPA 2000b). DDT is very widely distributed; it has been found in wildlife all over the world and in many human samples as well.

Although some use of DDT continues throughout the tropics, it remains of human health concern in the United States primarily due to its presence in water, soil, and food. Because individuals are typically exposed to a mixture of DDE, DDT, and DDD and their degradation and metabolic products, the sum of the 4,4'- and 2,4'- isomers of DDT, DDE, and DDD will be evaluated together in this HHRA.

Pharmacokinetics

DDT and its analogs are stored in fat, liver, kidney, and brain tissue; trace amounts can be found in all tissues (EPA 2000b). DDE is stored more readily than DDT. DDT is eliminated through first-order reduction to DDD and, to a lesser extent, to DDE. The DDD is converted to more water-soluble *bis*(p-chlorophenyl)acetic acid, with a biological half-life of 1 year. DDE is eliminated much more slowly, with a biological half-life of 8 years. Because elimination occurs slowly, ongoing exposure may lead to an increase in the body burden over time.

Acute toxicity

The low effect dose for severe effects (acute pulmonary edema) in infants has been reported to be 150 mg/kg. In adults, behavioral effects were noted at 5 to 6 mg/kg and seizures at 16 mg/kg (HSDB). Evidence from acute exposure studies of dogs indicates that DDT may sensitize the myocardium to epinephrine. This was observed for both injected epinephrine and epinephrine released by the adrenal glands during a seizure and resulted in ventricular fibrillation. DDT may concurrently act on the CNS, in a manner similar to that of other halogenated hydrocarbons, to increase the likelihood of

fibrillation. Chronic exposure to 10 mg/kg-d did not produce increased incidence of arrhythmias in rats or rabbits (EPA 2000b).

DDD is considered less toxic than DDT in animals. Symptoms develop more slowly and have a longer duration with DDD than with DDT exposure. Lethargy is more significant and convulsions are less common than with DDT exposure (HSDB).

Chronic toxicity

Extensive research has been conducted on chronic and subchronic exposure effects of DDT in animals and in humans working with DDT. These studies have primarily focused on carcinogenic effects, which are discussed in the following section. Studies have also identified liver damage, and there is limited evidence that DDT may cause leukocytosis and decreased hemoglobin level (EPA 2000b). Immunological effects have been associated with exposure to DDT.

IRIS lists an **oral RfD of 0.0005 mg/kg-d** for DDT based on liver effects with a NOAEL of 0.05 mg/kg-d from a 27-wk rat feeding study conducted in 1950. Uncertainty factors of 10 each for inter- and intraspecies variability were used; however, the usual factor of 10 for a less-than-lifetime study was not applied "because of the corroborating chronic study in the data base" (IRIS).

Carcinogenicity

DDE, DDT, and DDD are all considered probable human carcinogens (category B2) based on animal studies, with **oral cancer slope factors of 0.24, 0.34, and 0.34 per mg/kg-d**, respectively (IRIS). Liver tumors were associated with each chemical. The occupational studies of workers exposed to DDT are of insufficient duration to assess carcinogenicity (IRIS). Elevated leukemia incidence, particularly chronic lymphocytic leukemia, was noted in two studies of workers. Lung cancer has also been implicated in one study. Bone marrow cells in experimental animals have also been affected by exposure, including an increase in chromosomal fragments in the cells (HSDB). The oral cancer slope factor for DDT (0.34) will be used for total DDTs in this HHRA, in accordance with EPA (2000) recommendations.

DIELDRIN

Dieldrin is an organochlorine pesticide that was phased out between 1974 and 1987. It continues to be detected nationwide due to its relatively long half-life. Dieldrin is also a product of aldrin metabolism (ATSDR 1991).

Pharmacokinetics

Dieldrin is absorbed from the GI tract and transported through the hepatic portal vein and the lymphatic system. Soon after ingestion, it is found in the liver, blood, stomach, and duodenum. Dieldrin is lipophilic and ultimately stored primarily in fat and tissues with lipid components. A correlation between exposure and dieldrin levels in

human breast milk has been established, and placental transfer of dieldrin has been observed in women (ATSDR 1991).

Acute toxicity

The following symptoms are commonly associated with exposure to organochlorines: behavioral changes, sensory and equilibrium disturbances, involuntary muscle activity, depression of vital centers, myocardial irritability, convulsion, and unconsciousness (EPA 2000b). Additional effects of dieldrin exposure include: possible hematological effects in humans (pancytopenia and thrombocytopenia, immunohemolytic anemia)(ATSDR 1991). The estimated human lethal dose is 65 mg/kg (EPA 2000b).

Chronic toxicity

Liver toxicity has been observed in multiple animal studies and in human acute exposure episodes. Neurotoxicity has been observed in humans with chronic inhalation and dermal exposures (ATSDR 1991). Chronic exposures of pesticide applicators to dieldrin led to idiopathic epilepsy, which ceased when exposure was terminated (EPA 2000b).

IRIS provides an **RfD of 0.00005mg/kg-d** based on a NOEL of 0.005 mg/kg-d from a 1969 2-year rat feeding study that found liver lesions. Uncertainty factors of 10 each for inter- and intraspecies variability were applied (EPA 2000b). It appears the IRIS RfD provides adequate protection against neurological effects and reproductive toxicity, using standard assumptions and uncertainty factors for calculating an estimate exposure limit.

Carcinogenicity

Dieldrin is classified as a probable human carcinogen (Group B2) by EPA based on oral studies in animals. EPA has developed a **cancer potency factor of 16 per mg/kg-d**. ATSDR has concluded, based on studies that have been reviewed, that dieldrin is probably a tumor promoter. Varieties of tumor types have been observed in animal studies including pulmonary, lymphoid, thyroid, and adrenal (ATSDR 1991). In addition, dieldrin has recently been observed to have estrogenic effects on human breast cancer estrogen-sensitive cells and it may cause disruption of the endocrine system due to its estrogenic activity (Soto et al. 1994).

GAMMA-BHC (LINDANE) AND METABOLITES (ALPHA-BHC AND BETA-BHC)

Lindane is an organochlorine pesticide that is comprised of isomers of hexachlorocyclohexane (BHC), with the gamma isomer constituting the major (>99 percent) component. There appears to be some difference in toxicity of the various hexachlorocyclohexane isomers (EPA 2000b). Lindane is used primarily for controlling

wood-inhabiting beetles and as a seed treatment. Lindane is also used as a prescription pharmaceutical to control head lice and mites (scabies) in humans.

Pharmacokinetics

Lindane is readily absorbed by the GI tract following oral exposure. Distribution is primarily to the adipose tissue but also to the brain, kidney, muscle, spleen, adrenal glands, heart, lungs, blood, and other organs. It is excreted primarily through urine as chlorophenols. The epoxide metabolite may be responsible for carcinogenic and mutagenic effects (EPA 2000b). Male exposure to lindane through the environment results in accumulation in testes and semen in addition to the tissues listed above (ATSDR 1994).

Acute toxicity

The estimated human lethal dose is 125 mg/kg (HSDB). Occupational and accidental exposures in humans have resulted in headaches, vertigo, abnormal EEG patterns, seizures, and convulsions. Death has occurred primarily in children.

Chronic toxicity

IRIS provides an **RfD of 0.0003 mg/kg-d** based on a NOAEL of 0.33 mg/kg-d from a subchronic rat study that found liver and kidney toxicity at higher doses. Uncertainty factors of 10 each for inter- and intraspecies variability and the use of a less-than-lifetime study were applied (IRIS). The confidence in the principal study, database, and RfD are rated as medium. Liver damage has been observed in many animal studies and appears to be the most sensitive effect (EPA 2000b). Immune system effects have been observed in humans exposed via inhalation and in orally dosed animals. A 5-week study in rabbits found immunosuppression at 1 mg/kg-d (ATSDR 1994).

Most observed effects in humans exposed accidentally to lindane are neurological. Behavioral effects have also been noted in many studies on experimental animals, and at relatively high levels seizures were reported. More subtle behavioral effects were noted at an LOAEL of 2.5 mg/kg-d with 40 days of exposure in rats. No NOAEL was reported (ATSDR 1994).

Carcinogenicity

Lindane has been classified as Group B2 (probable human carcinogen) (EPA 2000b) and a **oral cancer slope factor of 1.3 per mg/kg-d** has been listed (HEAST). Lindane's related isomers, alpha and beta hexachlorocyclohexane, are classified as probable human carcinogens and have cancer potencies similar to that of lindane (**6.3 per mg/kg-d for alpha-BHC and 1.8 per mg/kg-d for beta-BHC**). In addition to tumors identified in experimental animals, human study data indicate that this chemical may cause aplastic anemia (EPA 2000b).

HEPTACHLOR

Heptachlor is a synthetic chemical that was used in the past for killing insects in homes, buildings, and on food crops. Heptachlor is both a breakdown product and a component of the pesticide chlordane (approximately 10% by weight). Pure heptachlor is a white powder. Technical-grade heptachlor is a tan powder. Heptachlor may be found in the soil or air of homes treated for termites, dissolved in surface water or groundwater, or in the air near hazardous waste sites. Heptachlor is still approved by EPA for killing fire ants in power transformers.

Pharmacokinetics

Approximately 20% of heptachlor is changed within hours into heptachlor epoxide in the environment and in your body. Heptachlor has been shown to bioaccumulate in fish and cattle. People store heptachlor epoxide in their fatty tissue. Some studies show that heptachlor epoxide can still be measured in fatty tissue 3 years after a person is exposed (ToxFAQs). Most of the heptachlor that is swallowed is absorbed into blood. Heptachlor can pass directly from a mother's blood to an unborn baby through the placenta.

Acute toxicity

Blood tests suggest that heptachlor may cause mild liver changes in humans. A few human cases show that breathing pesticide mixtures containing heptachlor may affect the nervous system causing dizziness, fainting, or convulsions (ToxFAQs). Studies of people who made or used pesticides that included heptachlor found no serious health effects. Acute toxicity studies with animals indicate that heptachlor can cause tremors, convulsions, and loss of kidney function at high doses.

Chronic toxicity

Subchronic dietary studies with mice resulted in liver and adrenal gland damage. Animals that ate food containing heptachlor before and/or during pregnancy had smaller litters (ToxFAQs). EPA has established an **RfD of 0.0005 mg/kg-day** for heptachlor based on a 2-yr rat feeding study that documented increased liver weight in males (IRIS). An uncertainty factor of 100 was applied to account for inter- and intraspecies differences. An additional factor of 3 was considered appropriate because of the lack of chronic toxicity data in a second species.

Carcinogenicity

Animals fed heptachlor throughout their lifetime had more liver tumors than animals that ate food without heptachlor. EPA has classified heptachlor as a probable human carcinogen (B2) and established a **oral cancer slope factor of 4.5 per mg/kg-day**.

HEPTACHLOR EPOXIDE

Heptachlor epoxide is a breakdown product of the organochlorine pesticides heptachlor and chlordane and is a contaminant of both products. It is more toxic than either parent compound (ATSDR 1993b). Although most uses of heptachlor were suspended in 1978 and chlordane was removed from the market in 1988 (EPA 2000b), heptachlor epoxide continues to be a widespread contaminant due to its relatively long half-life.

Pharmacokinetics

Based upon animal and limited human data, heptachlor epoxide is absorbed through the GI tract and is found primarily in the liver, bone marrow, brain, and fat, although it is distributed widely to other tissues as well. It is stored primarily in fat. Fetal blood levels were approximately four times those measured in women.

Heptachlor epoxide has a very long half-life, particularly in adipose tissue. Human tissue levels have correlated well to age, with 97 percent of North Texas residents tested (ages 41 to 60) having measurable levels. Based on the Texas study, heptachlor epoxide tissue levels have not decreased appreciably since the 1960s (EPA 2000b).

Acute toxicity

The LD50s for heptachlor epoxide range from 40 to 162 mg/kg in rodents (EPA 2000b).

Chronic toxicity

IRIS provides an **RfD of 1.3×10^{-5} mg/kg-d** based on an LOAEL of 0.0125 mg/kg-d from a 60-week dog feeding study reported in 1958. The critical effect was increased liver-to-body-weight ratios in both males and females at the lowest dose tested. Uncertainty factors of 10 each were applied for inter- and intraspecies variability and the use of an LOAEL rather than a NOAEL (IRIS). No additional uncertainty factors were applied for the use of a less-than-lifetime study. The principal study is of low quality and there is low confidence in the RfD (IRIS).

Animal studies have identified the following effects associated with heptachlor (and subsequently heptachlor epoxide via metabolism) or heptachlor epoxide directly: elevated bilirubin and white blood cell count, increased serum creatinine phosphokinase levels suggestive of muscle damage, muscle spasms secondary to CNS stimulation, adrenal gland pathology, and neurological disorders (EPA 2000b).

Carcinogenicity

Heptachlor epoxide is classified as a probable human carcinogen (category B2) by EPA based on oral studies in animals. The **oral cancer slope factor is 9.1 per mg/kg-d**. This value is based on the geometric mean of several studies that identified liver

carcinomas (IRIS). Five structurally related compounds have produced tumors in mice and rats: chlordane, aldrin, dieldrin, heptachlor, and chlorendic acid (IRIS).

Heptachlor (and consequently heptachlor epoxide) exposures have been associated with cerebral gliosarcoma in children exposed prenatally. Multiple chromosomal abnormalities were also identified in the tumor cells. It was not determined whether the effects were caused by environmental or familial factors (EPA 2000b).

HEXACHLOROBENZENE

Hexachlorobenzene is a byproduct of manufacturing and in the past it has been used as a fungicide seed protectant. At ambient temperatures, it exists as a solid, and in aquatic environments, it is found in higher quantities in sediment than water due to its low solubility (ATSDR 1990c).

Pharmacokinetics

Hexachlorobenzene is persistent in the body due to its lipophilic nature. It is found in human breast milk (ATSDR 1990c), which may be a significant route of exposure for young children.

Acute toxicity

The following symptoms are commonly associated with exposure to organochlorines: behavioral changes, sensory and equilibrium disturbances, involuntary muscle activity, depression of vital centers, myocardial irritability, convulsion, and unconsciousness (EPA 2000b). Acute exposure studies in animals have demonstrated a low acute toxicity for hexachlorobenzene with LD50s between 1,700 and 4,000 mg/kg. Based on animal studies, the following systems are negatively affected following acute exposure: liver, kidney, hematological, and dermal (ATSDR 1990s).

Chronic toxicity

A large number of people in Turkey were exposed from 1955 to 1959 to grain contaminated with hexachlorobenzene. Precise exposure estimates are not available, but it was estimated that exposure levels of 0.7 to 2.9 mg/kg-d for a 70 kg individual occurred (ATSDR 1990c). The following effects were associated with this exposure: shortening of the digits due to osteoporosis, painless arthritis, decreased uroporphyrin synthase levels, muscle weakness, rigidity and sensory shading, thyroid enlargement, and histopathological changes in the liver often accompanied by skin lesions (ATSDR 1990c). These effects have also been observed in numerous animal studies.

Based on animal studies, the hepatic system appears to be the most sensitive systemic endpoint for hexachlorobenzene exposure. The results from these studies have been converted by EPA to an **RfD of 0.0008 mg/kg-d** using uncertainty factors of 10 each for inter- and intraspecies variability (ATSDR 1990c).

Carcinogenicity

Carcinogenic assays of hexachlorobenzene in animals have identified an increased incidence of multiple tumor types including hepatomas, hemangioendotheliomas, liver, and thyroid tumors in numerous species. Hexachlorobenzene is classified as a possible human carcinogen (B2) based on the results of animal studies (EPA 2000b). EPA has established a **cancer potency factor of 1.6 per mg/kg-d**. Follow-up studies of the exposure of hexachlorobenzene to the victims in Turkey have not identified cancers in the 25- and 20- to 30-year exposure cohorts. However, ATSDR notes that the enlarged thyroids noted in members of these cohorts have not been adequately investigated (ATSDR 1990c).

HEXACHLOROBUTADIENE

Hexachlorobutadiene, also known as HCBD, is formed during the processing of other chemicals such as tetrachloroethylene, trichloroethylene, and carbon tetrachloride. Hexachlorobutadiene is an intermediate in the manufacture of rubber compounds and lubricants. It is used as a fluid for gyroscopes, a heat transfer liquid, or a hydraulic fluid. Outside of the United States it is used to kill soil pests (ToxFAQs).

Pharmacokinetics

In animal studies, most of the hexachlorobutadiene is metabolized into more toxic compounds. It is not known how rapidly hexachlorobutadiene and its breakdown products are removed from your body through your urine and feces. Some is expected to remain in your body fat for long periods (ToxFAQs).

Acute toxicity

Ingestion of hexachlorobutadiene damaged the kidneys of rats and mice and, to a lesser extent, the liver of rats. These effects occurred after both short- and long-term exposures at very low dose levels. Young rats were affected more than adult rats. The kidneys of female rats appeared to be affected more than those of males. On the other hand, the liver of male rats was affected, but the liver of female rats was not. It is not clear if the differences between the sexes might be seen in humans. Kidney, brain, and liver damage were also seen in rabbits after contact of their skin with the compound for a short period.

Chronic toxicity

Hexachlorobutadiene was shown to affect the function of the liver in one study of workers at a solvent production plant who breathed hexachlorobutadiene for long periods. Hexachlorobutadiene decreased fetal body weight in rats, but did not affect fetal development or impair their ability to produce offspring. The lungs, heart, brain, blood, muscles, and skeleton in rats or mice were not damaged after long-term exposure.

Carcinogenicity

Studies in rats indicate that hexachlorobutadiene may increase the risk of kidney cancer if exposures occur for long periods. EPA has classified hexachlorobutadiene as a possible human carcinogen (C) based on observations of renal neoplasms in male and female rats in one study. EPA established a **oral cancer slope factor of 0.078 per mg/kg-day**.

IRON

Iron is the second most abundant metal in the earth's curst. The most common iron ores include hematite, magnetite, limonite, and siderite (HSDB). Iron salts are used as fertilizer micronutrients, herbicides, electrolytes in dry cell batteries, animal feed additives, galvanizers, and as emulsion breakers. The major route of exposure to iron is through the mining and handling of iron ores (HSDB).

Pharmacokinetics

Iron is found naturally in the body as an important component of hemoglobin. In overdoses (>20 mg/kg-day), iron may be absorbed into the body may be extremely fast, where it is incorporated into structural proteins (Spanierman 2001). Excretion may be extremely slow in these cases.

Acute toxicity

Acute iron toxicity is the main cause of pediatric poisoning death in the United States. The hallmark feature of iron overdose is gastrointestinal bleeding. Iron is an extremely corrosive substance in the GI tract. The absorption of excessive quantities of ingested iron will result in systemic iron toxicity. Severe overdose causes impaired oxidative phosphorylation and mitochondrial dysfunction, which can result in cellular death. One of the most affected organs is the liver, but other organs, such as the heart, kidneys, lungs and the hematologic systems may be impaired (Spanierman 2001).

Chronic toxicity

Chronic exposure to iron oxide fume or dust can cause a pulmonary roentgenographic appearance called siderosis. This is considered a benign pneumoconiosis and does not ordinarily cause significant physiologic impairments (HSDB). Iron is also suspected to be a cardiovascular or blood toxicant, gastrointestinal or liver toxicant, neurotoxicant, and respiratory toxicant (HSDB). EPA's National Center for Exposure Assessment has developed a **provisional RfD for iron of 0.3 mg/kg-day**. Provisional RfDs have greater uncertainty than RfDs certified by EPA.

Carcinogenicity

EPA has placed iron in weight-of-evidence group D, not classifiable as to human carcinogenicity.

LEAD

Lead is a naturally occurring bluish-gray metal found in small amounts in the earth's crust. Lead's most important industrial use is in the production of some types of batteries. It is also used in the production of ammunition, in some kinds of metal products (such as sheet lead, solder, some brass and bronze products, and pipes), and in ceramic glazes. Human activities (such as the former use of "leaded" gasoline) have spread lead and substances that contain lead to all parts of the environment. Before the use of leaded gasoline was banned, most of the lead released into the US environment came from car exhaust. Other sources of lead released to the air include burning fuel, such as coal or oil, industrial processes, and burning solid waste.

Sources of lead in dust and soil include lead that falls to the ground from the air, and weathering and chipping of lead-based paint from buildings and other structures. Lead in dust may also come from windblown soil. Disposal of lead in municipal and hazardous waste dump sites may also add lead to soil. Mining wastes that have been used for sandlots, driveways, and roadbeds can also be sources of lead (ATSDR 1999c).

People living near hazardous waste sites may be exposed to lead and chemicals that contain lead by breathing air, drinking water, eating foods, or swallowing or touching dust or dirt that contains lead. For people who do not live near hazardous waste sites, exposure to lead may occur in several ways: 1) by eating foods or drinking water that contain lead, 2) by spending time in areas where leaded paints have been used and are deteriorating, 3) by working in jobs where lead is used, 4) by using health-care products or folk remedies that contain lead, and 5) by having hobbies in which lead may be used such as sculpturing (lead solder) and staining glass.

Pharmacokinetics

Absorbed lead is distributed in various tissue compartments.

Acute toxicity

Lead can affect almost every organ and system in your body. The most sensitive is the central nervous system, particularly in children. Lead also damages kidneys and the reproductive system. The toxic effects of lead are the same regardless of the route of entry into the body, and they are correlated with internal exposure as blood lead level.

Chronic toxicity

At high levels over long periods of time, lead may decrease reaction time, cause weakness in fingers, wrists, or ankles, and possibly affect the memory. Lead may cause anemia, a disorder of the blood. It can also damage the male reproductive system. The connection between these effects and exposure to low levels of lead is uncertain.

Since most of the toxicity data for lead is based on an internal dose, a reference dose, which is based on an external dose (i.e., mg/kg-day) has not been developed. Data on external exposure (i.e., mg/kg-day) are available from animal studies, but these data are generally not used to assess human health impacts because of the large database available using blood levels. Risks from lead exposure will be evaluated using the IEUBK model, as described in Section B.3.4.4. EPA and the Centers for Disease Control and Prevention have determined that childhood blood lead concentrations at or above 10 µg/dL present risks to children's health.

Carcinogenicity

The Department of Health and Human Services has determined that lead acetate and lead phosphate may reasonably be anticipated to be carcinogens based on studies in animals. There is inadequate evidence to clearly determine lead's carcinogenicity in people (ToxFAQs).

MANGANESE

Manganese is an element considered essential to human health. However, divalent manganese is about 2.5 to 3 times more toxic than trivalent manganese, and the anions of manganese salts influence the overall manganese toxicity. Industrial activities which use manganese include steel manufacturing, nonferrous alloys, purifying and scavenging agent in metal production, manufacturing of aluminum, ceramics, matches, glass, and welding rods (HSDB).

Pharmacokinetics

Humans ingest manganese from three main sources: diet, drinking water, and inhaled particles. Manganese that is inhaled is mostly brought up from the respiratory tract by ciliary action and swallowed, eventually being absorbed in the GI tract (Clayton and Clayton 1981). After oral exposure, absorbed manganese is quickly eliminated from blood and distributed mainly to the liver, kidneys, and endocrine glands. Minor amounts go to the brain and bone as shown in studies using mice, rats and monkeys.

Acute toxicity

Acute manganese poisoning has effects similar to other heavy metals if dust or fumes are inhaled in sufficient quantity. The minimum dose that produces effects on the central nervous system is not known and, with few exceptions, such effects have been observed only in occupationally exposed individuals. Sixteen cases of manganese poisoning have been described for a small Japanese community, three of which were fatal (including one suicide). The manganese content of the water was about 14 mg/l and concentrations of about 8 and 11 mg/l were found in two other wells. The subjects exhibited psychological and neurological disorders associated with manganese poisoning and high **manganese** and zinc levels were found in organs at autopsy (WHO 1981).

Chronic toxicity

The usual form of chronic manganese poisoning primarily involves the central nervous system. Early symptoms include, languor, sleepiness, and weakness in legs, emotional disturbances such as uncontrollable laughter and a tendency to fall while walking (ACGIH 1986). Experimental studies have suggested that populations at greatest risk of adverse effects due to manganese exposure are the very young and those with an iron deficiency, and workers exposed to manganese at or near the recommended threshold limit value. EPA has established an **RfD of 0.14 mg/kg-d** for a 70 kg adult.

Carcinogenicity

Manganese is not classified as a carcinogen to humans, although existing studies are inadequate to assess the carcinogenicity of manganese to humans and animals (ToxFAQs).

MERCURY

Mercury is widely distributed in the environment due to both natural and anthropogenic processes. It is released generally as elemental mercury (Hg^0) or divalent mercury (Hg^{2+}). It can be converted between these forms and may form mercury compounds by chemical processes in air, water, and soil. Biological processes in other media, primarily soil and sediment, can convert inorganic mercury into organic mercury, primarily methylmercury. In fish tissue, the majority of mercury is in the form of methylmercury (EPA 2000b).

Pharmacokinetics

Methylmercury is rapidly and nearly completely absorbed; estimates of absorption efficiency are 90 percent or greater (ATSDR 1999d, EPA 1997c, WHO 1990). Methylmercury is readily distributed to all tissues following absorption from the GI tract. Methylmercury in the body is considered to be relatively stable and is only slowly demethylated to form mercuric mercury. Estimates for the half-life of methylmercury range from 44 to 80 days (EPA 1997c).

Methylmercury binds readily to protein and can be found throughout fish tissue. A substantial portion of the mercury in fish can be found in trimmed filets, making it difficult to reduce exposure by trimming fat and skin prior to cooking (EPA 2000b).

Acute toxicity

Acute high-level exposures to methylmercury may result in kidney damage and failure, gastrointestinal damage, cardiovascular collapse, shock, and death. The estimated lethal dose is 10 to 60 :g/g-day (ATSDR 1999d).

Chronic toxicity

Neurotoxicity is the chronic effect of greatest concern, both to the developing embryo or fetus and to adults and children (EPA 2000b). Effects to humans from consumption of contaminated food have been documented in Japan and Iraq.

The current EPA **RfD for methylmercury of 1×10^{-4} mg/kg-day** was originally based on data on neurologic changes in 81 Iraqi children who had been exposed *in utero*. This value was subsequently updated using data from a population in the Faroe Islands who were exposed to methylmercury and PCBs through consumption of fish and pilot whale. In deriving the RfD, EPA used a benchmark dose (BMD) approach to quantify a dose-effect relationship between methylmercury in cord blood and a neurological endpoint. A BMD limit of 58 μg/L cord blood was estimated based on findings from the Boston Naming Test, a neuropsychological evaluation. A methylmercury intake level associated with a blood level of 58 μg/L was then calculated to be 1.0 μg/kg-day. A total uncertainty factor of 10 was then applied to account for variation in the methylmercury intake-to-blood ratio, lack of data on toxicodynamic variability, and limitations in the toxicological database (i.e., insufficient data on cardiovascular effects, lack of a two-generation reproductive study, and inadequate data to quantify long-term effects). The current RfD of 0.1 μg/kg-day (i.e., 0.0001 mg/kg-day) derived from the Faroe Islands data, is thus unchanged from the previous RfD derived from the Iraqi data. EPA's overall confidence in the RfD is rated as medium, based on medium confidence levels for both the primary study and the supporting database.

Carcinogenicity

Methylmercury is currently Class C, a possible carcinogen based on inadequate data in humans and limited evidence in animals. Dietary exposure of mice to methylmercury resulted in significant increases in the incidences of kidney tumors in males but not in females (EPA 1997c). Evidence points to a mode of action for methylmercury carcinogenicity that operates at high doses certain to produce other types of toxicity in humans. Given the relatively low levels of exposure, even among consumers of highly contaminated fish, methylmercury is not likely to present a carcinogenic risk to the US population (EPA 2000b). An oral slope factor is currently not available for methylmercury.

NICKEL

Nickel is used in a wide variety of industries. Occupational exposure is the predominant cause of harmful exposure to nickel.

Pharmacokinetics

Nickel is hepatotoxic in animals and is shown to affect renal function in humans. It binds to anionic glycosaminoglycan sites of the glomerular basement membrane. This causes ionic blocking and leads to loss of sensitivity in the filtration of albumin.

Divalent nickel ions can penetrate the skin at sweat duct and hair follicle ostia. The ions then bind with keratin and cause contact dermatitis. Nickel has a biological half-life of 20-34 hours in plasma and 17-39 hours in urine (HSDB).

Acute toxicity

Dermal contact with nickel causes contact dermatitis. Nickel poisoning occurred in 23 dialyzed patients when nickel leached in dialysate from a nickel-plated stainless steel water heater. The victims experienced nausea, vomiting, weakness, headache, and palpitation (HSDB).

Chronic toxicity

Nasal and lung cancer have resulted from chronic inhalation of nickel particles (IRIS). Damage to the nasal mucosa, asthma, pneumoconiosis, conjunctivitis, and epiphora have also been observed after long term exposure (HSDB). EPA (IRIS) has developed an **RfD of 0.02 mg/kg-day** based on decreased body and organ weights in a long-term rat feeding study. Uncertainty factors of 10 were used for both interspecies extrapolation and to protect sensitive populations. An additional uncertainty factor of 3 was used to account for inadequacies in the reproductive studies.

Carcinogenicity

EPA has classified nickel refinery dust as a known (Class A) carcinogen, but the soluble salts of nickel on which the oral RfD is based are not classified as carcinogenic. This classification was based on a study of sulfide nickel matte refinery workers who developed lung and nasal tumors after being exposed to nickel refinery dust, and also on data collected from nickel carcinogenicity studies with rats (IRIS).

N-NITROSODIMETHYLAMINE

N-nitrosodimethylamine is not currently used in industrial processes, except for research purposes where it may be released to the environment with laboratory waste. It was once used as an antioxidant, additive for lubricant, as a softener of copolymers, and in the production and use of rocket fuels (HSDB).

Pharmacokinetics

N-nitrosodimethylamine is absorbed from GI tract and lung; skin absorption is slow. When administered to rats, mice, and rabbits, it is distributed uniformly in tissue and has a half-life of approximately 4 hr. Although the liver is the main organ concerned with its metabolism and is a site of selective toxicity, N-nitrosodimethylamine does not concentrate there (HSDB).

Acute toxicity

Systemic effects are characterized by onset in a few hours of nausea and vomiting, abdominal cramps and diarrhea. Headache, fever, and weakness may also occur. Ultimately liver disease may result (HSDB).

Chronic toxicity

Chronic toxic effects other than liver disease and cancer have not been well-documented. EPA has not established an RfD for N- nitrosodimethylamine.

Carcinogenicity

EPA has classified N- nitrosodimethylamine as a probable human carcinogen (B2) based on the induction of tumors at multiple sites in both rodents and nonrodent mammals exposed by various routes (IRIS). EPA has established a **oral cancer slope factor of 51 per mg/kg-day** (IRIS).

N-NITROSO-DI-N-PROPYLAMINE

N-Nitroso-di-n-propylamine is produced primarily as a research chemical and not for commercial purposes. However it has been identified as a contaminant in the substituted dinitrotrifluralin herbicides, and thus may be released to the environment when these herbicides are used and from spills, as well as from some industrial effluents. The general population may be exposed to N-Nitroso-di-n-propylamine in spray drifts from fields where trifluralin is used. N-Nitroso-di-n-propylamine is rarely found in food. (HSDB).

Pharmacokinetics

N-Nitroso-di-n-propylamine is distributed evenly throughout the body. When administered to pregnant animals, the compound crosses the placental barrier and can be found in fetal tissue. It has been measured in milk and blood one hour after oral administration (HSDB).

Acute toxicity

Acute toxic effects from N-Nitroso-di-n-propylamine are not well-documented.

Chronic toxicity

Chronic toxic effects other than teratogenicity and carcinogenicity have not been well-documented. EPA has not established an RfD for N-Nitroso-di-n-propylamine (IRIS).

Carcinogenicity

EPA has classified N-Nitroso-di-n-propylamine as a probable human carcinogen (B2) based on the increased tumor incidence at multiple sites in two rodent species and in

monkeys administered the compound by various routes. EPA has established a **oral cancer slope factor of 7 per mg/kg-day** (IRIS).

POLYCHLORINATED BIPHENYLS (PCBS)

Although the production and use of PCBs were banned in this country in 1979, this chemical group is extremely persistent in the environment and bioaccumulates through the food chain (EPA 2000b). There is evidence that some dioxin-like PCB congeners, which are assumed to be the most toxic, preferentially accumulate in organisms higher on the food chain, including humans. As a result, the composition of PCB mixtures in fish tissue may differ significantly from the environmental PCB source. Often the mixtures of interest are not those that have been used in studies of laboratory animals to determine toxicity (EPA 2000b).

Pharmacokinetics

PCBs are absorbed through the gastrointestinal tract and distributed throughout the body, although the highest accumulation is typically in lipid-rich tissues. Human milk may contain relatively elevated PCB concentrations due to its high fat content (ATSDR 2000b).

The retention of PCBs in fatty tissues is linked to the degree of chlorination and also to the position of the chlorine atoms in the biphenyl ring. In general, more chlorinated congeners persist for longer periods of time. In occupationally exposed individuals, less chlorinated congeners had half-lives between 1 and 6 years, while more chlorinated congeners had half-lives ranging from 8 to 24 years (ATSDR 2000b). In subjects who consumed PCB-contaminated rice in Taiwan, the half-lives of several PCBs ranged from 3 to 24 months (EPA 2000b).

Acute toxicity

Studies in animals have shown that exposure to very high doses of PCBs can cause death. However, doses of such magnitude are unlikely in environmental exposures and current industrial settings. There have been no reports of deaths in humans after exposure to PCBs even where exposures were much higher than those typically identified with environmental exposures (ATSDR 2000b).

Chronic toxicity

Numerous effects have been documented in animal studies including hepatic, GI, hematological, dermal, body weight, endocrine, immunological, neurological, reproductive, developmental, and liver cancer (ATSDR 2000b). Evidence of chronic effects in humans is not nearly as definitive. Several studies in humans have suggested that PCB exposure, particularly via in utero exposure through maternal fish consumption, may cause adverse effects in children and in developing fetuses (ATSDR 2000b). Neurobehavioral effects in such children have been documented by Fein et al.

(1984), Jacobson and Jacobson (1996, 1997), and Schantz (1996). Over intermediate durations (i.e., less than 10% of an organism's lifetime), learning problems have been noted in monkeys fed PCB mixtures similar in composition to human breast milk (ATSDR 2000b).

EPA has derived an **RfD of 2×10^{-5} mg/kg-day** for Aroclor 1254. The RfD was based on a LOAEL of 0.005 mg/kg-day for ocular and immunological effects in monkeys. Uncertainty factors of 10 for sensitive individuals, 3 for extrapolation from monkeys to humans, 3 for extrapolation from a subchronic exposure to a chronic RfD, and 3 for use of a minimal LOAEL were applied by EPA, resulting in a total uncertainty factor of 300. EPA's overall confidence in the RfD is rated as medium, based on medium confidence levels for both the primary study and the supporting database.

Carcinogenicity

PCBs are classified by EPA as Class B2, probable human carcinogens. This designation is based on studies that have found liver tumors in rats exposed to Aroclors 1260, 1254, 1253, and 1016. Human epidemiological studies of PCBs have not yielded conclusive results (Silberhorn et al. 1990).

EPA has developed a range of slope factors for PCBs (EPA 1996b). Using information on environmental processes, they have provided guidance for choosing an appropriate slope factor based on the class of the mixture and the exposure pathway. Because bioaccumulated PCBs appear to be more toxic and more persistent in the body than commercial PCBs, the upper bound slope factor associated with high risk and persistence (**2.0 per mg/kg-d**) was used in this HHRA.

When assessing PCB mixtures, it is important to recognize that both dioxin-like and non-dioxin-like modes of action contribute to overall PCB toxicity. It is possible that concentrations of dioxin-like congeners are increased in an environmental mixture. When congener concentrations are available, the mixture-based approach based on Aroclor analyses can be supplemented by analysis of dioxin TEQs to evaluate dioxin-like toxicity. In that analysis, the dioxin slope factor (150,000 kg-day/mg) is used. In some cases, the magnitude of the dioxin slope factor results in PCB dioxin-like congeners contributing the majority of the risk.

PENTACHLOROPHENOL

Pentachlorophenol is a man-made substance that does not occur naturally in the environment. At one time, it was one of the most widely used biocides in the United States. Now the purchase and use of pentachlorophenol are restricted to certified applicators. It is no longer available to the general public. Application of pentachlorophenol in the home as an herbicide and pesticide accounted for only 3% of its consumption. Before use restrictions, pentachlorophenol was widely used as a

wood preservative. It is now used industrially as a wood preservative for power line poles, cross arms, and fence posts (ToxFAQs).

Pharmacokinetics

The most common exposure routes for pentachlorophenol are inhalation and dermal contact. Human studies have estimated half lives of less than 33 hours. Bioaccumulation appears to be minor; most absorbed pentachlorophenol does not break down, but instead leaves in urine. Much smaller amounts leave in feces (ToxFAQs).

Acute toxicity

Many, but not all, the harmful effects associated with exposure to pentachlorophenol may be due to impurities present in commercial pentachlorophenol. Short exposures to large amounts of pentachlorophenol in the workplace or through the misuse of products that contain it can cause harmful effects on the liver, kidneys, blood, lungs, nervous system, immune system, and gastrointestinal tract. Contact with pentachlorophenol (particularly in the form of a hot vapor) can irritate the skin, eyes, and mouth. If large enough amounts enter the body, heat is produced causing an increase in body temperature. The body temperature can increase to dangerous levels, causing injury to various organs and tissues and even death (ToxFAQs).

Chronic toxicity

Long-term exposure to low levels such as those that occur in the workplace can cause damage to the liver, kidneys, blood, and nervous system. The major organs or systems affected by long-term exposure to low levels in animals are the liver, kidney, nervous system, and immune system. All these effects get worse as the level of exposure increases (ToxFAQs).

EPA has established an **RfD for pentachlorophenol of 0.03 mg/kg-day** based on a rat chronic oral study that documented liver and kidney pathology (IRIS). A 100-fold uncertainty factor accounts for the expected intra- and inter- species variability to the toxicity of this chemical in lieu of specific data.

Carcinogenicity

EPA has classified pentachlorophenol as a probable human carcinogen (B2) and has established a **oral cancer slope factor of 0.12 per mg/kg-d** based on statistically significant increases in the incidences of multiple biologically significant tumor types (hepatocellular adenomas and carcinomas, adrenal medulla pheochromocytomas and malignant pheochromocytomas, and/or hemangiosarcomas and hemangiomas) in mice. In addition, a high incidence of two uncommon tumors (adrenal medulla pheochromocytomas and hemangiomas/hemangiosarcomas) was also observed.

SILVER

Silver is toxic by all routes of exposure. Exposure is predominantly occupational via industries associated with electroplating, photographic materials, brazing, welding, and the manufacturing of jewelry, mirrors, coinage, pigments, and antiseptics (HSDB).

Pharmacokinetics

Silver is retained in all body tissues. It is primarily deposited in the skin, adrenals, lung, muscle, pancreas, kidney, heart, and spleen. Excretion of silver from the body is mainly via the GI tract. Silver has a biological half-life of 1.7-2.5 days, as determined in studies using dogs, rats, monkeys, and mice (HSDB).

Acute toxicity

Acute exposure to silver can result in skin and eye irritation, mild bronchitis, metal fume fever and hepatic damage, stomach pain, and lung and throat irritation (HSDB).

Chronic toxicity

The most common result of chronic exposure to silver is generalized argyria, a blue-gray discoloration of the skin, mucous membranes, and eyes (HSDB). Workers involved in manufacturing precious metal powder experienced elevated urine and blood silver concentrations and respiratory irritation. EPA has calculated the **RfD for silver at 0.005 mg/kg-day** based on a long-term study of argyria in humans (IRIS). An uncertainty factor of 3 was applied to account for minimal effects in a subpopulation that has exhibited an increased propensity for the development of argyria.

Carcinogenicity

EPA has placed silver in Class D, not classifiable as to human carcinogenicity (IRIS).

THALLIUM

Thallium occurs in nature as a trace compound and is mainly associated with potassium and rubidium. Anthropogenic sources of thallium are gaseous emissions from cement factories, coal burning power plants, metal sewers, and leaching from ore processing operations (OGWDW).

Pharmacokinetics

Thallium has been shown to inhibit enzymatic action in the body. In acute thallium poisoning, human brain areas densely populated with neurons were found to accumulate thallium more than other areas. Thallium excretion is a very slow process mainly occurring via the kidney, gut, and salivary glands (HSDB).

Acute toxicity

Acute exposure to thallium can cause sever paroxysmal abdominal pain, vomiting, and diarrhea. Thallium poisoning often causes an increase in heart rate and blood pressure. Some victims have experienced a loss of vision in industrial exposures. In very severe cases, tremors, delirium, convulsions, hypotension, bradycardia, paralysis, coma, and death can occur (HSDB).

Chronic toxicity

Long-term exposure to thallium causes the relaxation of vascular smooth muscle, increased sympathetic tone, vagus nerve damage, fatty infiltration and necrosis of the liver, nephritis, gastroenteritis, pulmonary edema, degenerative changes in the adrenals, degeneration of peripheral and central nervous system, alopecia, and in some cases death (HSDB). IRIS cites an **RfD for thallium chloride of 0.00008 mg/kg-d**, which is based on a subchronic study with rats.

Carcinogenicity

EPA has placed thallium in Class D, not classifiable as to human carcinogenicity.

TOXAPHENE

Toxaphene is an organochlorine pesticide that is comprised of a mixture of at least 670 chlorinated camphenes. Toxaphene was probably the most heavily used pesticide in the United States during the 1970s after DDT was banned. It was banned for most uses in 1982; all uses were banned in 1990. However, due to its relatively long half-life, it persists in the environment. The soil half-life is approximately 1 to 14 years (HSDB).

Pharmacokinetics

The components of toxaphene are metabolized in mammals via dechlorination, dehydrodechlorination, and oxidation, primarily through the action of the mixed function oxidase system and other hepatic microsomal enzymes. Conjugation may occur but is not a major route of metabolism. Each component of toxaphene has its own rate of biotransformation, making the characterization of toxaphene pharmacokinetics complex. Some components of toxaphene are highly lipophilic and poorly metabolized; these components may accumulate in body fat (ATSDR 1996).

Acute toxicity

Acute high-level exposures to toxaphene and toxaphene-contaminated food have resulted in death in adults and children with an estimated minimum lethal dose of 2 to 7 g, which is equivalent to 29 to 100 mg/kg for an adult male. LD50 values in rats were 80 mg/kg for females and 90 mg/kg for males. Transient liver and kidney effects, and periods of memory loss have been observed in humans after single large oral

exposures. In animals, the most sensitive organ is the liver. Toxicity to the central nervous system, kidney, and adrenal glands have also been observed (ATSDR 1996).

Chronic toxicity

IRIS does not provide a discussion of chronic effects of exposure to toxaphene or an RfD. Chronic exposure to toxaphene may result in damage to the following organ systems: liver, kidney, adrenal, immunological, and neurological (ATSDR 1996). Chronic exposure to toxaphene may cause hormonal alterations. A study on chronic exposures found increased levels of hepatic metabolism of the hormones estradiol and estrone and a decrease in their uterotropic action. Some adverse effects of toxaphene that do not occur with a single exposure may result from repeated exposures. Exposures at 0.06 mg/kg-d over 5 weeks caused adrenal hormone reductions, whereas a single dose of 16 mg/kg did not cause effects.

Carcinogenicity

Toxaphene is classified as a probable human carcinogen (B2) by EPA based on oral studies in animals (IRIS). No conclusive human epidemiological studies are available for toxaphene (ATSDR 1996c). Oral administration of toxaphene resulted in an increased incidence of hepatocellular carcinomas and neoplastic nodules in mice, and thyroid tumors in rats (IRIS). The cancer potency is 1.1 per mg/kg-d, based on liver tumors in experimental animals (IRIS, 1999).

Toxaphene has recently been observed to have estrogenic effects on human breast cancer estrogen-sensitive cells (Soto et al. 1994). Xenoestrogens have been hypothesized to have a role in human breast cancer. In addition to potential carcinogenic effects, toxaphene may also cause disruption of the endocrine system due to its estrogenic activity (Soto et al. 1994).

TRIBUTYLTIN

TBT is one of several organotin compounds that have been used as biocides, disinfectants, and antifoulants. This overview focuses primarily on bis(tri-*n*-butyltin) oxide (TBTO) because this is the only TBT compound for which the EPA has established an RfD for assessing chronic toxicity to humans, and because more toxicological information is available for this compound than for other organotin compounds.

Pharmacokinetics

No studies are available regarding the distribution of tin in human tissues following oral exposure (ATSDR 1992). Laboratory studies with mammals have shown that organotin compounds are absorbed; studies with rats detected tin compounds in the GI tract, kidney, and liver. Rats that orally ingested tin compounds showed the highest concentrations in the liver and kidneys; concentrations in the brain and

adipose tissue were 10 to 20 percent of those found in the kidneys and liver (Krajnc et al. 1984). Studies involving trialkyltin compounds show that absorbed compounds are metabolized, with the data suggesting that the liver is the active site and dealkylation the principle metabolic pathway (ATSDR 1992).

Acute toxicity

There are no controlled studies on the effects of TBTO in humans. The available data demonstrate that TBT is toxic to animals, with LD50 values ranging from 122 to 194 mg/kg in rats.

Chronic toxicity

There are no studies on the effects of TBTO in humans. Animal studies have shown effects on the blood and liver, and immunological effects, including thymus atrophy and depletion of T-lymphocytes in the spleen and lymph nodes (ATSDR 1992).

EPA's IRIS database provides an **RfD for TBTO of 3×10^{-4} mg/kg-day**, based on a NOAEL of 0.025 mg/kg-day and an uncertainty factor of 100. This was based on a chronic feeding study of rats in which immunologic function analyses for specific and nonspecific resistance were performed after 4–6 or 15–17 months of exposure to test doses of TBTO ranging from 0.025 to 2.5 mg/kg-day (Vos et al. 1990). The uncertainty factor of 100 is the product of a factor of 10 for uncertainty associated with extrapolating from a laboratory animal species to humans, and a factor of 10 to protect sensitive humans. EPA's overall confidence in the RfD is rated as high, based on high confidence levels for both the primary study and the supporting database. The RfD for TBTO can be converted to TBT ion units by multiplying it by the ratio (0.49) of the molecular weights for the two substances. The resulting **RfD for the TBT ion is 0.00015 mg/kg-day**.

Carcinogenicity

TBTO is currently Class D, which is defined as a chemical not classifiable with respect to human carcinogenicity. There are no data documenting the development of cancer in humans following exposure to TBTO. A large number of studies show that TBTO is not genotoxic, and there are no structure-activity relationships suggesting that TBTO might be a carcinogen.

VANADIUM

Vanadium compounds are widely distributed in the earth's crust. Elemental vanadium does not occur in nature, but its compounds exist in over 50 different mineral ores and in association with fossil fuels (HSDB). The route of entry of vanadium compounds most commonly seen in industrial exposures is through the respiratory system. Exposures are usually limited to areas where vanadium pentoxide is produced, in

steel mills where vanadium pentoxide is used, and in cleaning boilers fired by oil containing vanadium (HSDB).

Pharmacokinetics

Vanadium compounds and metallic vanadium, when absorbed, are rapidly excreted and exhibit low degrees of toxicity, as indicated by minor irritation and lack of systemic effects. Absorbed vanadium is widely distributed in the body. In animals, the highest values are found in bone, kidney, liver, spleen and lung. Bone maintains essentially unchanged levels for several weeks. The lowest values are found in the brain, but in human autopsy material, brain concentrations of vanadium are more or less the same as those found in other organs (HSDB).

Acute toxicity

Vanadium and its compounds are principally eye and respiratory tract irritants that result in conjunctivitis, coughing, wheezing, difficulty in breathing, and industrial bronchitis. A metallic taste and throat irritation may occur. Greenish discoloration of the fingers, scrotum, and upper legs may also be present. A greenish black discoloration of the tongue indicates heavy exposure (HSDB).

Chronic toxicity

Some studies suggest exposure to vanadium may impair the lung resistance to respiratory infection, although the available data on chronic respiratory effects of vanadium are still inconclusive. HEAST provides an **RfD of 0.007 mg/kg-day** for vanadium.

Carcinogenicity

EPA has placed vanadium in Class D, not classifiable as to human carcinogenicity.

ZINC

Zinc is an essential trace element that plays a necessary role in enzymatic functions, protein synthesis, and carbohydrate metabolism. Small doses of zinc are necessary for normal growth and development in birds and mammals. Zinc also has many industrial uses. It is used as a galvanizing agent, component in brass, bronze alloys, light metal alloys, and in wet batteries (HSDB). The most common route of high-level exposure to zinc is through consumption of liquid contained in galvanized metal containers or by water contaminated with industrial zinc waste (ToxFAQs).

Pharmacokinetics

Absorption of zinc occurs in the intestine when ingested or through the lung when zinc dust or fumes are inhaled. Zinc is mainly stored in skeletal muscle, but significant

concentrations can also occur in the pancreas, prostate, liver, and retina. Zinc has a biological half-life of 162-500 days (HSDB).

Acute toxicity

In humans, ingestion of gram quantities of zinc may cause pancreatic derangement, light-headedness, and mild derangement of cerebellar function. Acute exposure to zinc can also cause dizziness, nausea, tightness in the throat, diarrhea, and vomiting. Metal fume fever has been observed after inhalation of zinc oxide fumes (HSDB).

Chronic toxicity

Prolonged exposure to drinking water that contained 40 mg/L of zinc triggered symptoms such as irritability, muscular stiffness and pain, loss of appetite, and nausea (HSDB). EPA has established an **RfD of 0.3 mg/kg-day for zinc** based on a human diet supplement study in which adult females experienced a 47% decline in erythrocyte superoxide dismutase (ESOD) after 10 weeks of exposure (IRIS). An uncertainty factor of 3 was applied, based on a minimal LOAEL from a moderate-duration study of the most sensitive humans and consideration of a substance that is an essential dietary nutrient.

Carcinogenicity

EPA has placed zinc in Class D, not classifiable as to human carcinogenicity (IRIS).

2-NITROANILINE

2-Nitroaniline is a chemical intermediate for the production of dyes and pigments. There are no known sources of this compound in the LDW. Occupational exposure via dermal contact is the most probable route of human exposure (HSDB).

Pharmacokinetics

No information available.

Acute toxicity

Acute exposure to 2-nitroaniline can cause methemoglobinemia (a blood disorder) and cyanosis (HSDB).

Chronic toxicity

Chronic exposure to 2-nitroaniline may cause liver damage (HSDB). EPA determined that this chemical does not reach levels in the workplace environment that are cause for concern for chronic, development, and reproductive effects (53 FR 31805). A **Rfd of 0.000057 mg/kg-day** was published in HEAST.

Carcinogenicity

EPA has not classified 2-nitroaniline for carcinogenicity, but it is likely to be carcinogenic given the lack of mutagenic effects (HSDB).