Duwamish River Cleanup Coalition

Community Coalition for Environmental Justice • Duwamish Tribe• Georgetown Community Council • Green–Duwamish Watershed Alliance • ECOSS • People for Puget Sound • Puget Soundkeeper Alliance • Washington Toxics Coalition • Waste Action Project

August 14, 2002

Ms. Allison Hiltner
U.S. Environmental Protection Agency
1200 Sixth Avenue, ECL–111
Seattle, WA 98101

Dear Ms. Hiltner:

The Duwamish River Cleanup Coalition (DRCC) represents the Duwamish River Valley and greater Seattle community as EPA's Community Advisory Group for the Lower Duwamish Waterway (LDW) Superfund Site. DRCC also holds a Department of Ecology contract under the Washington State Public Participation Grant program to conduct community education, outreach and involvement programs related to the site cleanup. Our directing members are representatives of nine community, environmental, tribal and small business organizations affected by contamination and cleanup of the lower Duwamish River.

Coalition organizational member Waste Action Project holds a Technical Advisor Grant from EPA and provides technical expertise to the Coalition. Experts in ecological risk analysis, human health risk analysis, remedial action design and Duwamish River ecology and toxicology have assisted us with our review of technical documents related to the Lower Duwamish Waterway Phase I Superfund Cleanup.

DRCC and our members have previously submitted comments on the LDW National Priorities List site designation, Statement of Work, Public Participation Plans, Duwamish/Diagonal Way Combined Sewer Overflow Cleanup plan, and early drafts of the Remedial Investigation/Risk Assessment Problem Formulation, Effects and Exposure Assessment, Site Prioritization and Selection methodology, and associated technical memos. We are also currently reviewing LDWG's proposed Early Action Candidate Site Identification document, and the Draft Public Health Assessment for the LDW being prepared by the Washington State Department of Health and ATSDR in response to the Superfund listing.

The following are our comments on the Lower Duwamish Waterway Group's (LDWG) Draft Phase I Remedial Investigation (RI), Ecological Risk Assessment (ERA), and Human Health Risk Assessment (HHRA). We respectfully request that EPA and Ecology take these comments under consideration when reviewing and guiding development of the final RI, ERA and HHRA for the Lower Duwamish Waterway Superfund Site.

GENERAL COMMENTS

Arsenic must be driver for cleanup

The HHRA identifies arsenic as posing the highest human health risk for cancer, but the RI suggests that Duwamish River sediments are not significantly higher than "background" – implying that there may be no point in cleaning up the site. This is not only misleading and incorrect, but a dangerous suggestion that, if accepted by EPA, may result in continued unacceptable and avoidable cancer risks to the affected communities.

Central Puget Sound arsenic levels represent ubiquitous contamination (much of it largely from Asarco fallout), not "natural" background levels. Background arsenic levels for the State of Washington have been determined by the Washington State Department of Ecology and USGS to be about 7 ppm. This finding was recently upheld by the Washington State Pollution Control Hearings Board. Not only is this much lower than levels found throughout the Duwamish River, but the current standard applied in the RI/RAs of 20 ppm is likely not sufficiently conservative or protective and should be re-evaluated. Studies under development by the Department of Ecology suggest that arsenic levels of less than 7 ppm are necessary to protect human health.

A brief review of arsenic contamination data presented in the RI indicates that arsenic poses significant risks at several locations throughout the Duwamish River and must be considered as a driver in selecting sites for early action and long term cleanup.

Top 15 cm sediment sampling is insufficient

The RI/RAs use the top 15 cm to characterize risks and proposed cleanup sites in Phase I. This is insufficient for either purpose and has the potential to grossly underestimate risks to the environment and human health caused by chemical contamination in the Duwamish River.

The Report argues that the top15 cm is the biologically active zone. This assumes that this 15 cm zone is static, or only accumulating sediment, and that there are no organisms that burrow or are exposed to sediments below 15 cm, which is false (clams, for example). Current activities on the river, including boat prop wash, high volume storm water discharges, and

dredging routinely stir up sediments below 15 cm, both bringing them to the surface and burying them. These factors and the strong salt water tidal surge would cause surface sediments at any given point in time to actually be blended sediments, some portion of which may have been in the biologically active zone for some time and some of which will be recently disturbed deeper sediments which will now be part of the surface layer until the next time they are buried by new sediments or transported out of the site by disturbance. It is also impossible to define early action cleanup sites based on the top 15 cm of sediments, as contaminated sediments typically extend 6–9 feet deep or more throughout the Duwamish River. Proposed cleanup sites can be expected to expand in size and depth far beyond what the top 15 cm of data suggest.

PCB contamination in salmon need more study

The RI/RAs retains juvenile salmon for further study, but dismisses the need to further evaluate adult salmon for additional study on the effects of PCBs. Given the complete lack of data on the effects of early PCB exposure on adult salmon, this is completely unacceptable. There is also no data presented on specific PCBs (congeners) in juvenile and adult salmon, which could provide much information on both the source and effects of PCBs found in salmon from the Duwamish River. The larger question of whether Duwamish River salmon are safe to eat is being addressed elsewhere, but must also be discussed here, as it has potentially serious implications for human health and cumulative impacts on affected communities. This is especially important in light of recent EPA reports that salmon with similar or lower PCB levels in the Columbia River are not safe for tribal consumption.

Insufficient protections for children's health

The RI/RAs do not adequately evaluate the potential human health effects on children resulting from contact with and consumption of Duwamish River sediments. Many of the standards used were not developed for children, exposure scenarios regarding beach play excluded contact with subtidal sediments, cumulative effects of exposure to chemicals other than lead (mercury, PCBs, dioxins, DDT) were not considered, and fetal exposures for pregnant women were not considered at all. Children are not just small adults, and many children in the area of the Duwamish River can be expected to carry significant body burdens for many chemicals that must be taken into account when determining the effects of exposure to contaminated sediments. Anything less fails to protect our children and is entirely unacceptable.

Hazard Quotient not protective

The Duwamish River Cleanup Coalition remains concerned that the Hazard Quotients and indices applied at the screening and final determination stages are not sufficient to account

for all the data gaps, uncertainties and safety factors that need to be addressed. For the purposes of this Phase I RI/RA, we recommend a more protective Hazard Quotient of 0.1.

DRAFT PHASE I REMEDIAL INVESTIGATION REPORT

Section 1.0: Introduction

Page 1, paragraph 4: With regard to risks assessments in the Phase II RI, the RI states, "These baseline risk assessments and other factors will be used by EPA and Ecology to set sediment cleanup levels for the rest of the LDW beyond the early action sites."

Please clarify that collection of additional data may result in information indicating additional cleanup needs even at completed early action sites. This possibility cannot be ruled out based on a limited and incomplete data set. Alternatively – and preferably – each Early Action site will undergo thorough data gap analysis, collection and evaluation prior to remedial action.

Section 1.3.1: Site description

Page 3, paragraph 2: The RI states, "Intertidal habitats are dispersed in relatively small patches (i.e., generally less than one acre in size." The RI should recognize that more and larger intertidal habitat areas are likely to be restored to the river in the future. An example of this trend can be found in the recent Hamm Creek restoration project. While this project was only recently completed, species designated as ROCs, such as spotted sandpipers and osprey, have been observed nesting at Hamm Creek, and waterfowl concentrations may bring bald eagles and peregrine falcons to the area.

Section 1.3.2: Site history

Page 3, paragraph 6: The second sentence should read: "Historical ***and current*** commercial and industrial operations include cargo handling and storage, marine construction, boat manufacturing, marina operations, concrete manufacturing, paper and metals fabrication, food processing, and airplane parts manufacturing. In addition, the LDW was ***and is*** a receiving water body for many different types of industrial and municipal wastewater. Some of these waste streams have been rerouted or discontinued, but there are still numerous storm drains, combined sewer overflows ***and direct industrial dischargers*** that currently discharge to the LDW"

To suggest that these commercial uses and industrial discharges are not ongoing is misleading and incorrect. The current industrial dischargers are listed in the list of NPDES permitted dischargers in Table E-1 on page 256.

Section 2.0: Environmental Setting and Previous Investigations
Section 2.3.1: Sediment quality

The exclusion of much pre-1990 data and all sediment samples below 15 cm in the Phase I RI needs to be clearly stated up front, with the rationale for the exclusion of this data. With regard to the 15 cm limitation, it should be noted that the DRCC has vigorously opposed this exclusion from the beginning. Subsurface sediment data show much higher levels of chemical contamination at depth, resulting in an RI and risk assessments that may significantly underestimate risk and potentially leading the LDWG and regulatory agencies to overstate the anticipated effectiveness of planned Early Action Site remediation projects.

Page 11, paragraph 1: The RI states that some sediment chemistry data were excluded due to removal/dredging actions. Tables D-1 and D-2 list the sediment sampling locations that were excluded, but do not provide dates when the dredging took place. Without this information, these samples cannot be excluded.

Of particular concern is exclusion of data from locations where other research has shown significant impacts from sediment contamination on LDW organisms. For example, juvenile Chinook that forage at Slip 4 exhibit grossly elevated PCB levels, attributed to site-specific contamination (Meador 2002). Did the data used in this study pre- or post-date the dredging event and excluded chemistry data from this site?

In addition, sediment chemistry at these excluded sites needs to be recognized as a data gap in Phase II unless new data exists. The RI should clarify where new data has been used to replace excluded data, and where new data still needs to be collected.

Finally, it appears that some data from previously remediated or partially remediated sites was retained. This is inconsistent with the approach described for dredged sediments, and may also contribute to the appearance of greater risk reduction for sites identified for cleanup than is actually true. Specifically, the Slip 4 and Boeing Plant II sites seem to have been identified in part based on previously remediated sediments. This issue will be addressed further in our comments on the Early Action Site Identification document, but must be addressed here.

Section 2.3.5: Fish and shellfish tissue chemistry

Page 11–2, Table 2–5:Footnote k states that six composite samples of juvenile Chinook livers were also analyzed, but that the data was not used in the RI/RAs. No explanation is given in Section 4.2.7. A brief explanation should be given in the RI document as to why the data were not used. At a minimum, the exact location where this information can be found in Appendix A should be provided.

Recent studies by Meador et al (2002) are missing. Also, please clarify why data collected by NOAA prior to 1989–90 were excluded from the RI. The rationale used to exclude sediment chemistry data prior to 1990 is not applicable to tissue and other data types. Given the limited database, all topical data should be included. Specifically, we refer the authors to McBain et al 1990; juvenile Chinook salmon samples from 1986; multiple studies on flatfish studies from the 1980's on reproductive output; Casillas et al 1991; Johnson et al 1988; chemical concentrations in sediments and fish from various locations, Malins et al 1984; benthic invertebrates uptake and bioavilability work by Varanasi et al 1985.

We do note that data from before 1990 was not excluded from the review of benthic invertebrate data (Table 2-6, page 19). Were no samples of invertebrates collected and analyzed by NOAA from 1980 to the present? It appears that there is a systematic exclusion of much of the existing NOAA data.

Explanations in the RI (or specific location of information in Appendix A) of excluded data noted in footnotes e and f are also necessary.

Section 2.4.4.3: Site usage by mammals

Page 38, paragraph 7: The RI states that harbor seals and California sea lions have “rarely” been observed in the Duwamish River. In fact, these marine mammals are regularly seen in the Duwamish River, especially during salmon runs. At a minimum, harbor seals and sea lions are observed in the Duwamish River annually.

Section 2.5.2: Land use

Page 42, paragraph 3: The RI states, “The LDW is primarily an industrial waterway today.” Page 44, paragraph 1: The RI states, “Predominant human use within the LDW is for commercial and industrial purposes. Recreational and residential uses also occur, but on a more limited scale.”

These statements mischaracterize the facts. Table 2–16 (Current land cover/use) on page 43 shows that low, medium and high density residential land use (41%) is nearly equal to industrial/commercial and city center/industrial and mining (42.5%). It is not clear which of the conifer, deciduous, grass, mixed forest, scrub/shrub, shadow, and open water areas are parkland and other recreational areas, but when these uses are included, residential and recreational land use may even exceed commercial and industrial.

Table 2–17 (Designated land use) shows similar parity. Commercial and industrial uses account for 44% designated land uses in the LDW, while residential and parkland/open space accounts for 43%. If the mixed use (including residential) are added to the residential use, residential/recreational use exceeds commercial/industrial.

This statement needs to be corrected to recognize that residential/recreational and commercial/industrial uses are essentially equal in the LDW.

Section 2.5.3.2: Recreational site use

Page 44, paragraph 4: The RI states that, “few, if any, people engage in water activities such as swimming, SCUBA diving and windsurfing in the LDW.” While it may be possible to dismiss windsurfing as a frequent activity in the LDW, these other uses do exist frequently enough to merit serious consideration. In addition, some commercial SCUBA diving occurs in the LDW, and kayaking is a frequent recreational use that involves substantial contact with both water and sediments.

Page 45, paragraph 2: The RI states that recreational fishers fish at three sites in the LDW. The RI should also recognize that some of these fishers are more appropriately characterized as subsistence fishers, and that these three sites listed are not the only three locations where fishing occurs. In addition to the sites listed, at least five other sites have been identified as commonly used by recreational/subsistence fishers on the river, including the Spokane Street Bridge, the Railway Bridge, the Kenco Marine Dock, 1st Avenue Bridge, and Terminal 105 (M. Frame, pers. comm.). In addition, recreational fishers also fish by boat along the entire length of the river.

Section 2.5.3.3: Residential/commercial site use

Page 45, paragraph 4: The RI again states that, “most of the land use and zoning in the LDW corridor is industrial.” This statement should be corrected per our comments in Section 2.5.2 above.

Section 4.0: Summary of Nature and Extent of Contamination
Section 4.1.1.2: Tissue

Page 62, paragraph 2: The RI states, "Juveniles accumulate chemicals from the Duwamish from their food during their downstream migration." Juveniles may also be exposed to sediments in the shallow intertidal areas where they rest and forage. Several NOAA studies, rejected because they were laboratory studies, demonstrated that juvenile salmon also are exposed to significant amounts of chemicals from contact with sediments suspended in the water column. These studies should not be ignored, considering the likelihood of such resuspension to occur in the LDW.

In the third sentence, the RI states, "Salmon reside in the LDW for only brief periods during their migration to and from their spawning grounds." While this is likely true relative to their entire life span, it is unknown just what juvenile salmon's residency time is in the Duwamish River on their outmigration to Puget Sound and the ocean. Some salmon reside in the Duwamish longer than others, and may show high site fidelity within the estuary, sometimes residing and foraging almost exclusively in highly contaminated areas. This will be discussed further in our comments on the ERA.

The fourth sentence of paragraph 2 should read: "Other chemical sources ***may be*** hatchery feed and transfers from the mother to the eggs." Any positive statement should be accompanied by supporting data.

The final sentence of paragraph 2 states, "Therefore, only a very small percentage of their total chemical load comes from the LDW. This is certainly not true of juveniles, and any statement to this effect for adults should be qualified by the information in our comments above.

Section 4.2.1: Data selection and reduction

Page 66, paragraph 2: The RI states, "Chemistry for surface water and groundwater are not included in the LDWG database because analysis and mapping of these data was not needed due to the sediment and tissue focus of this project." Exclusion of this data is inappropriate, especially given that certain surface water chemistry is likely largely or exclusively due to sediment contamination, and some surface and groundwater contamination may be sufficient to contribute to contamination of LDW sediments. For example, PCBs in surface water could likely only come from re-suspension of contaminated sediments. NOAA and King County (1999) determined that PCB suspension in the water column was sufficient to cause

significant exposure and effects in juvenile salmon. In addition, groundwater needs to be examined for similar interrelationships.

In at least one instance in the 1990s, groundwater infiltration into Birmingham Steel's outfall pipe was identified as the cause of PCB contamination in the mill's effluent (Dept. of Ecology, draft NPDES permit for Birmingham Steel). If there are enough PCBs in upland soil and groundwater to be captured in Birmingham Steel's outfall pipe, it is virtually certain that PCBs would be caught within the LDW's zone of influence and transported to the river.

In the case of the Boeing Issacson Steel site, groundwater has been identified as being contaminated with arsenic. Some portion of the property's arsenic contamination has remained capped on site. Groundwater data for the site also shows that elevated levels of arsenic in proximity to the LDW sediments, though the RI does not provide the maximum contaminant levels for soil, or groundwater, or the distance from those contaminant sources to the LDW sediments. Given that LDW sediments and tissue samples are contaminated with elevated levels of arsenic, neglecting to look at upland concentrated arsenic sources is unacceptable.

The final sentence of paragraph 2 states, "Summaries of the nature and extent of water contamination will be taken from other documents." With reference to the King County 1999 CSO Water Quality Assessment this is inappropriate. The data and methodology from this study should be transparent in the Remedial Investigation Report. This is especially important given King County's responsibility and potential liability for remedial actions. It is standard in RIs to have independent verification of all data (which is rarely provided in data used for this RI). Accepting the results of a study using unseen data and unreviewed methodology is yet another step removed from proper data verification and accountability.

Page 66, paragraph 3: With regards to exclusion of sediment data, the same questions apply as in our comments on section 2.3.1, above. Please also provide information regarding where the dredged sediment was removed *to*. These receiving areas should be considered for inclusion in the definition of the "site."

Section 4.2.2: Surface sediments

Page 71, Table 4–3: The notes at the bottom of the table indicate that COCs and CPOCs were identified using a HQ of 1.0. Is this correct? DRCC understood that COCs/CPOCs were screened using a HQ of 0.1. Please correct or clarify.

The specific chemical sections that follow note exceedances of SQS and CSL levels but the RI does not provide a listing of these values. Please include a reference table of SQS/CSLvalues for ready comparison with the values of chemicals detected.

Section 4.2.5: Surface water

Page 81, Table 4–4: Modeled chemical concentrations in surface water derived from the King County Water Quality Assessment HHRA are shown in Table 4–4. The PCB data shows that modeled PCB concentrations in surface water exceed chronic water quality criteria, and both arsenic and PCBs exceed Federal Human Health WQC for Aquatic Organisms. The only known source for PCBs in surface water is suspension of sediments. This finding confirms and emphasizes the need to include surface water chemistry as a potential significant exposure route in the data evaluated for the RI/RAs, per our comments on section 4.2.1.

Page 82, paragraph 1: The RI states that the modeled arsenic concentrations shown in Table 4–4 are similar to regional background concentrations of arsenic in the Green/Duwamish watershed. The authors need to recognize the ubiquitous nature of arsenic contamination throughout the watershed and central Puget Sound. Similar levels to this "background" likely reflect many other contamination sources more than any "natural" background.

All references to "background" in regard to these elevated arsenic concentrations in the LDW should be removed from the document, except where the detections referred to are consistent with natural background concentrations as determined by Ecology and the USGS for our area (7 ppm; USGS). The draft RI is a document written for the public, as well as the agencies. To the general public, the term "background" is taken to mean that the source is natural rather than human-caused. EPA, Ecology, and the LDWG are aware that this is not the case for elevated levels of arsenic in the LDW.

Any prediction that arsenic levels in LDW sediments are not likely to be reduced by remediation is unsubstantiated. In fact, the potential to reduce arsenic levels through cleanup of the LDW is quite high, given the number of concentrated arsenic depositions on the river, such as multiple cement kiln dust (CKD) sites and the Boeing Issacson Steel facility, to name a few.

Section 4.2.6: Groundwater

See our comments on Section 4.2.1, Section 4.3.2.4, and others.

Section 4.2.7.1: Adult Chinook salmon fillet samples
Section 4.2.7.1: Adult coho salmon fillet samples

Page 84, paragraph 3: The RI states, “These data were not used in the risk assessments because the chemical concentrations in these fish are largely unrelated to sediment contamination in the LDW.” This conclusion is based on a single paper by O’Neill (O’Neill et al 1998). More supporting evidence is needed.

The O’Neill paper is inappropriate for use in reaching this conclusion without any other supporting data. The study was designed to address different questions from the one for which it is being applied; the juvenile and adult PCB data used for comparison were not comparable – juvenile data (from Varanasi) is based on whole body concentrations, while O’Neill analyzes PCB concentrations in fillets; the study did not consider age differences and differences between ocean and river type salmon; and the data was not lipid-normalized, among other problems. It is not known whether a properly designed study to examine the ratio of juvenile to adult PCB concentrations would result in higher or low percentages than estimated by O’Neill.

PSAMP data exists that can be more appropriately used to analyze the juvenile to adult PCB ratio using carefully designed and approved methodologies. Much of this data is already cited in the RI/RAs. For example, when corrected for weight, as the O’Neill study did, how does the 160 ug/kg ww PCBs in adult Chinook compare to the 260 ug/kg ww PCBs in juvenile Chinook reported in sections 4.2.7.1 and 4.2.7.2, respectively?

This issue will be discussed in more detail in our comments on the Risk Assessments.

The RI also provides no discussion of consumption of yearling or "jack" Chinook salmon, that spend a year in the estuary prior to heading out to the ocean. Juvenile salmon over 6” may be consumed by recreational or subsistence fishers prior to their outmigration from the LDW. There is also no discussion in the document about the presence of resident salmonids, which if present would not be exposed to PCBs in Puget Sound or the ocean.

Section 4.2.7.4: English sole whole body samples

Page 85, paragraph 4: With regard to chemicals in composite samples of whole body English sole, the RI states, “These data were used in the Phase I ERA.” Why were these data not also used in the HHRA? Some Duwamish River fishers consume whole fried fish, as well as some organs.

Section 4.3: Sources, Pathways and Source Control

Page 89, Figure 4–1: The Conceptual Model of chemical sources and pathways to the LDW fails to show a direct pathway between *Industrial and municipal discharges (permitted and non-permitted)* to *Surface water*. This is incorrect, per our comments on section 1.3.2. Please provide a corrected conceptual model.

Section 4.3.1: Potential sources

Page 90, paragraph 1: This section repeats the error of the conceptual model in Figure 4–1. With regard to industrial or municipal discharges, the RI states that, "chemicals from these sources may contribute to elevated chemical concentrations in various *upland* environmental media, including soils, groundwater, surface water, and impervious surfaces that can then act as *secondary* sources to the LDW" (emphasis added). There are in fact direct industrial and municipal discharges to the LDW.

Similarly, spills or leaks, atmospheric deposition and waste disposal (not to landfills, but illegal dumping in the river) may also be primary sources to the LDW. The conceptual model in Figure 4–1 captures pathways of these other sources adequately (except for illegal waste dumping), but the text suggests that their route is only secondary. Please correct.

Overall, we are disturbed at the lack of consideration of significant known historic sources of contaminants to the LDW site. Concern over this issue was brought into sharp focus in the recent public meeting on the draft RI. When exclusion of arsenic was questioned as it applies to selection of early action sites, the agency/consultant panel initially characterized arsenic as ubiquitous, and implied that even though the risks from sediment levels were high enough to act as a site selection driver, that the majority of related risk was from natural, or background arsenic levels. When this view was questioned, in light of the significant amount of data on wide spread contamination from the Tacoma ASARCO site plume, EPA was quick confirm that elevated levels of arsenic are in fact due to the plume, not high background. The consultant for LDWG, did not affirm this view however.

The Department of Ecology has in hand an assessment of state wide background metals levels, including arsenic. According to the study done for Ecology by the USGS the background concentration for arsenic in soils is 7 parts per million (Pete Kmet, Ecology 2001). Recent soil sampling done in King County as part of an Ecology data collection effort, has shown widespread elevated levels of arsenic and lead concentrations (KCSPH 2001). There are a number of known metals sources that may, or are known to, contribute to elevated concentrations of arsenic within the LDW site. A few of these sources include the

Tacoma ASARCO plume, the Western Processing Superfund site, and Cement Kiln Dust disposal areas in South Park, Georgetown, and West Seattle.

The Western Processing Superfund site is located in Kent, WA. EPA sediment monitoring found significant contamination from the site as far downstream as sampling was conducted. While the cleanup of this site did address Mill Creek sediments, they did so only for a very limited portion of the known contaminated sediments in the creek that were immediately adjacent to, and a very limited distance downstream (north) of the site. The limits of sediment cleanup were based on negotiation with the principle responsible parties, primarily the Boeing Company, and were not based on risks posed by the sediments. Mill Creek in Kent is a significant source of sediments and water to the Black River, which enters the Green River at the point it becomes the Duwamish, just upstream of the LDW site. Bioassays on the Mill Creek sediments performed by EPA found that the sediments were extremely toxic to test organisms (Judi Schwarz, 1984). Indeed EPA staff expressed some alarm that the sediments were more toxic than indicated via risk analysis of the known concentration of contaminants. Coalition members have periodically monitored the confluence of the Black River and the Green River since 1984. Those observations have noted significant loading of sediments into the Duwamish River from the Black River, and that the Black River is virtually always more turbid than the Green River. Due to the concern about PCBs and arsenic in the LDW site area, and concern about the potential for recontamination of early action sites, additional investigation of the Black River sediments is needed.

ASARCO provided another route of arsenic input to the Duwamish beside the Tacoma smelter plume. ASARCO contracted with Industrial Mineral Products (now Reserve Silica), which in turn contracted with a major cement company (Holnam Cement) on the Duwamish River, within the LDW site. IMP/Reserve Silica provides silica for the manufacture of cement. The contract IMP had with ASARCO was for the exclusive marketing of products developed from ASARCO slag. Two of the primary products made by IMP were grit (from ASARCO slag) for composite roofing, and raw material feedstock for cement. The company supplied slag to Holnam Cement, which in turn provided waste Cement Kiln Dust (CKD) to IMP for on site disposal. Ecology analysis of the CKD showed, as expected, very high levels of arsenic. There are numerous sites containing this same source of CKD throughout the West Seattle, Georgetown, South Park area, including sites along the LDW, which are available to contribute arsenic to the LDW site. A major release from one such site, the Malarkey property was recorded two years ago. The King County Department of Development and Environmental Services and Dept. of Ecology files contain documentation of the discharge, including relevant analysis and photographs. Analysis of the discharge (which was monitoring pH, not metals) showed pH of above 11 significantly downstream of

the property. A particular problem with this source of arsenic is that due to the way it was processed, the arsenic is in the form of very small particulate in a high pH substrate. With the modification of the pH, such as when the CKD is discharged into water, the availability of the arsenic and other metals is modified as well.

An additional source of metals that has long been problematic, but never adequately researched or remediated, is the abandoned mining operations in the Green River Gorge. While the majority of known mining in the region was related to coal, there was also significant metals mining in the Green River Gorge area, including for mercury. It is unknown at this point what the potential metals loading to sediments would be from these sources, but it should be noted as a data gap for further consideration.

Section 4.3.1.1: Industrial and municipal discharges

Page 91, paragraph 1: The RI states, "Chemicals may originate from specific industrial and municipal sites before discharging to the LDW via stormwater, combined sewer overflows (CSOs), or groundwater." In fact, both industrial and municipal sources discharge directly to the LDW. Please correct.

Pages 92–96: The RI lists locations of concern, include several sites with known or suspected deposits of cement kiln dust potentially contaminated with arsenic and other metals from ASARCO. Because there has not been a thorough assessment of the historic dumping of cement kiln dust with Asarco slag source material, considerable uncertainty exists as to the locations of this material, which should be recognized here.

The RI states that there has not been any substantive leaching of arsenic and other heavy metals at the outfall of Puget Creek at T-105. This may be because the outfall has only been in place since about 1990. What is also not considered is that the basic nature of the cement kiln dust may result in atypical mobilization and sedimentation due to different chemical forms of the metals. Has this been investigated?

Page 95, paragraph 3: The RI states that Phillip Services plans to close by the end of 2003. This plan has been amended based on plans by Phillip Services to close by the end of 2002. Additional well monitoring at Phillip is underway, due to the likelihood that the contaminated plume from the facility has intersected the river. There is currently no plan for containing or remediating the plume to prevent further releases to the LDW.

Please provide the most recent monitoring data from Phillip Services, as well as an assessment of the rate and recontamination potential of the plume.

Section 4.3.2: Pathways to LDW

Page 99, paragraph 4: The RI states, “Chemicals released to upland environmental media such as soil, groundwater, surface water or impervious surfaces may migrate to the LDW through various pathways.” Please amend to recognize direct industrial and municipal discharges to the LDW.

Section 4.3.2.4: Groundwater transport

Page 106, paragraph 5: The RI states, “there are currently no identified linkages between chemicals in groundwater at specific sites and resulting in sediment chemical concentrations in the LDW” This statement is incorrect. LDWG did not examine existing data regarding sources of contaminants migrating from groundwater to LDW sediments. There are also likely many data gaps that need to be filled to adequately assess this issue riverwide.

As stated above, there have been documented PCB discharges from the Birmingham Steel NPDES permitted outfall, as well as arsenic in groundwater at the Boeing Issacson Steel site. There are additional examples of where groundwater or product seeps may have contaminated sediments, including at the Boeing Plant II facility and the Malarkey Asphalt site. The document should be amended to state, "LDWG has not examined the potential for groundwater to transport contaminants to LDW sediments. There are, however, a number of sites where groundwater is known to contain or to have discharged COCs (including PCBs and arsenic) to the LDW. As such, this area is noted as a significant data gap for further consideration and data collection.”

Section 4.4.2: Sediment transport

Page 116, Table 4–14.The Table indicates a minimum sediment deposition rate of 20 cm/yr from approximately the Boeing Plant II site to the Turning Basin, near the end of the Superfund site. The implication is that surface data (from the top 15 cm) collected in 1998 is now at a depth of 80–95 cm. Surface data collected earlier, for example from 1990, would increase the current depth of sampled sediments to 240–255 cm, and applying a median (60 cm) rather than minimum deposition rate would further increase the depth of 1990 samples to 720–735 cm.

Has any effort been made to verify this model by sampling the same location today and determining the depth of the chemical concentrations recorded in the year sampling

occurred? LDWG would also have to take into account any instances of flood waters exceeding 7,000 cfs. Has there been a net burial of contaminated sediments? What is the actual rate of burial since 1990 as measured in the field? What is the source of surface contaminated sediments where high concentrations of PCBs exist if sediments are buried at a rate of 20–110 cm/year. Before any such modeling can be accepted, the model needs to be field tested by determining the model's predictability in the field and answering the questions above. In several areas of the river, data likely already exists to conduct this exercise; in others data gaps may need to be filled to test the model presented. The actual observed deposition rate likely varies by location in the river, and is critical to understanding sediment behavior at specific proposed cleanup sites. Failing to do this could result in fatal flaws in assumptions applied during Phase I cleanup design and execution.

Section 4.5: Key Processes Identified from Detailed Site-Specific Studies

Page 121, paragraph 3: The RI states, "Initial capping specifications have been developed to provide protection from potential vessel propeller scour." Please provide references for the capping depth required to protect from vessel propeller scour. What is the minimum depth?

DRAFT PHASE I ECOLOGICAL RISK ASSESSMENT

GENERAL COMMENTS

Top 15 cm Sediment Depth Insufficient

LDWG has used the top 15 cm to characterize risks and proposed cleanup sites in Phase I. This is insufficient for either purpose. Natural and human caused disturbance in the river disturbs sediments to a depth >15 cm. This issue has been brought up several times before. Some LDW organisms burrow and/or are in contact w/sediments below top 15 cm (e.g., clams). When considering Early Actions in locations where data on contaminated sediments exist at depth, how will this data be used to address cleanup guidelines? How will this information be connected into the Thiessen polygons? It is impossible to define Early Action Site boundaries based on the top 15 cm of sediment data. How will this inconsistency be addressed?

Need to Consider Toxicity of Duwamish River PCBs in Context with other Chemicals

Studies at NOAA over the past 20 years have demonstrated that there is simultaneous uptake of a multitude of chemicals from sediments from the LDW. A major effort at NOAA has been to

determine which compounds are responsible for many of the effects observed. These studies were not designed as toxicological studies, but were more concerned with issues like uptake, metabolism, and mechanisms for the correlations between a variety of population-wide effects observed since the mid 1970s. Meador et al (2002) states, "It is toxicologically valid to suggest that the results of these field studies may be due to the additive or synergistic relationship among all bioaccumulated contaminants; however, we lack the data necessary to assess such interactions. *This is a feature that should be incorporated into future studies and ecological risk assessments"* (emphasis added).

Total PCBs vs. Aroclors vs. Congeners

No ROCs should be dropped for further analysis until a more complete PCB analysis is performed based upon congener data. DRCC applauds the inclusion of herons following review of PCB data, and has included additional data from a bald eagle study in Hood Canal that indicates eagles received high levels of PCBs in the region in the early 1990s. Eagles may also be receiving high PCB loading in the LDW where higher PCB concentrations occur in prey items. Data from otters collected from Sinclair Inlet also show high levels of PCBs (Grove et al 2001).

1. Great Blue Heron Eggs from the West Seattle Colony

There are some problems with the presentation of the new data on the great blue herons eggs collected in West Seattle in 1998 (Krausmann 2002). Problems with the concentrations of PCBs in the heron eggs as well as the HQ calculation are discussed further in our comments on Sections A.5 and A.7. Such problems may also plague some of the assumptions made by LDWG in other prey item concentrations, food habits and Exposure assessment methods. Alternate methods need to be considered.

2. Hazard Quotients/Screening Levels

The DRCC has made requests to EPA and Ecology concerning issues relating to the exclusion of various COPCs in the Superfund process. Recently, EPA stated that the Hazard Quotients (HQs) used in the screening process were set at 0.1. It is somewhat unclear in some cases whether a safety factor is an *uncertainty*, or even a *data gap*. In attempting to understand the HQs at the screening level, we have reviewed some previous documents to determine whether certain safety factors were used to address cross-species differences in sensitivities, unknown extrapolations and other uncertainties. There appear to also have been omissions of many effects studies from the KC WQA after the initial assessments, without any further discussion about the 0.1 HQ, or any consistency across the safety factor issue. While these issues are considered by LDWG as resolved, it is certainly within the Superfund process at this early stage to reconsider ROCs and COPCs as new information becomes available. In fact, the Data

Gaps document appears to have that purpose in mind. What has not been resolved are inconsistencies in studies used in the KC WQA with lower effects levels in earlier studies that have been dropped, and a more detailed discussion of the relationship of the uncertainties discussed for certain ROCs and how these discussions could alter the previously deleted COCPs. This is particularly troublesome since several of these COPCs were discussed in comments by EPA concerning issues of incompleteness and lack of updated literature searches.

As an example of this process, data were submitted from heron eggs collected in the LDW (Krausmann 2002), and the risks estimated from the TEQs found in those eggs appear to warrant the retention of herons and now also eagles for PCBs, which were initially eliminated in the Exposure and Effects Assessment. Comments below will focus upon possible problems with the screening HQs for mercury and TBT in otters, which are also supported by empirical data from otters analyzed for these compounds by USGS (Grove et al 2001). Several COPCs being eliminated for certain fish species appear to be inconsistent with the same process. These more detailed discussions include:
Section A.5: Scaling and Safety Factors in HQ TRVs.
Section A.4.1.1**:** Changes from the King County CSO-WQA for Mercury Effects in Otters
Section A.4.1.1: Changes from the King County CSO-WQA for TBT Effects in Otters
Section A.5.2.1.3: Changes from the King County CSO-WQA for Lead Effects in Birds
Section A.5.2.1.4: Changes from the King County CSO-WQA for Mercury Effects in Birds
Section A.5.2.3.2: Changes from the King County CSO-WQA for Arsenic Effects in Otters
Section A.5.2.3.3: Changes from the King County CSO-WQA for Lead Effects in Otters

Our detailed comments on each of the above can be found in the corresponding "Specific Comments" sections, below.

3. Adult salmon should be retained for further study

Significant controversy remains over the issue of whether juvenile salmon are sufficiently exposed to PCBs and other contaminants (especially PAHs) to affect their growth, survival, and reproduction. No studies have been performed to address the reproductive performance of adult salmon that were exposed to contaminants as juveniles in the LDW, or even more importantly, the survival and status of offspring of the reproductive effort of the adults exposed as juveniles (so called second-generation effects). In light of the lack of available data or appropriate studies, adult salmon should be retained as a data gap as well as a COPC for the Phase 2 RI/ERA.

There is also controversy over the percentage of PCBs in adult salmon that return to the LDW. The DRCC does not doubt that a majority of PCBs are accumulated in salmon when they are in Puget Sound or the Pacific ocean. What has clearly not been presented is a

specific PCB congener based data set that help answer questions of Duwamish vs. Puget Sound/ocean exposure to PCBs. Because of the importance of the exposure of people to PCBs from salmon consumption, adult salmon need to be retained in Phase 2, and the Data Gaps Report should direct attention to this issue. The RI/ERA contains many unsubstantiated assumptions regarding the source of and risks posed by PCBs in LDW adult salmon. The citations in O'Neill et al (1998) [1.1% of PCBs] and in App. A (Page 53, as a footnote, that an estimate of a 15kg Chinook with 50ug/kg PCBs is well below an estimate of 4ug total PCBs in a outmigrating smolt) avoid many basic toxicological principles.

a. The effects of early PCB exposure is a data gap for adult salmon. "Although exposure to contaminants is correlated with reduced growth rates and short-term survival, the effects on long term marine survival and abundance of salmon populations are *unknown*. " (O'Neill et al.1998). Meador et al (2002) provides an argument for a lipid based threshold (RET) for juvenile salmon of 2.4ug PCB g-1 lipid. This limit is exceeded by most Duwamish River juvenile salmon. Meador states, "*This tissue concentration may indicate the potential for adverse effects in adult salmon as well*." Meador et al (2002).

b. The type of PCB congener and its TEF value is totally ignored. Not only does the LDW contain congeners with higher TEF values than the PCBs from the open ocean, but studies performed by NOAA indicate that these chemicals are retained. As indicated elsewhere in our comments, the most logical methodology for addressing some of the issues would be to examine PCBs on a congener by congener basis. The methodology for calculating Aroclor concentrations is difficult and confounded by changes in selective uptake and metabolism of congeners through the food chain, causing substantial changes from the Aroclor composition pattern from which estimates of Aroclor concentration are made. Rather than argue over the methodology and concentration issues, basing the assessment on individual compounds is a sound way to proceed with the risk assessment. Estimates of specific congeners in Aroclor mixtures can be made to link effects to empirical data. TEQ risk estimates are increasingly being made by linking the effects of PCBs to dioxins and furans.

c. There is a great deal of variability in the exposure that juvenile salmon may receive. The size and age of various salmon used to make these estimates are not presented. Age, size, and lipid content will cause significant variability in PCB concentrations. The O'Neill (1998) and ESG (1999) studies do not mention the type of salmon (Ocean vs Stream Type) used in their analysis. This is critical because the type of salmon captured by fisherman in the LDW is not characterized to make a comparison to existing PCB data. There is also some evidence that juvenile salmon show high site fidelity, rather than foraging widely over the LDW, which would cause some selective uptake of contaminants of concern in certain locations.

d. No impact of the effects of PCBs on growth and smoltification are mentioned, as well as the impact of juvenile exposure upon subsequent smoltification. The LDWG disregards much of the NOAA research as if it does not provide any information. IP injection has been found to be a comparable method to ingestion of COPCs, but has a much better dose-response relationship. Studies are available from EPA on the impacts of chemicals upon smoltification (EPA-Corvallis Lab). It is not known if any of the COPCs were investigated. If there is no data, then this is a potential data gap.

e. The ERA needs to provide analysis that protect individual Chinook salmon under the ESA. The NOAA White Papers were written to address the protection of Chinook Salmon under ESA relating to exposures in the LDW. Under ESA, more stringent protective measures are granted protected species than provided for under Superfund. Impacts relating to individuals and not just populations need to be considered.

SPECIFIC COMMENTS

A.2.2.3.1 Anadromous salmonids

Page 23-24: The information provided in the RI text is a critical component of estimating exposure time for juvenile Chinook salmon. Are there differences in the amount of time that the two juvenile Chinook salmon fry types (upper Green River vs LDW) spend in the LDW? How are these fish distinguished? Have there been any mark-recapture studies to verify individual residence time or are the estimates based upon size class analysis? It was stated (Meador et al 2002) that only in recent years have coded wire tagged fish been monitored in the Green-Duwamish. The text states that juvenile Chinook are found from mid-February to September; naturally spawned fish enter in late April- early May; and major hatchery releases are in May. Details about the size of released fish vs wild runs, their residence time, an the applicability of the Clarke and Shelburne study to conditions in the LDW need to be much more clearly stated.

A.2.2.4.1: Birds

Page 29: DRCC appreciates the attempt to present what must be a snapshot of one year of bird observations in the LDW (Canning et al 1979) but more comprehensive residence information is available (Hunn 1982). The method of obtaining such numbers, the time frame over which they were made, and the habitat associations related to the observations, which are critical for making any type of exposure assessment, are trivialized, and belittle the extensive use of the LDW by birds. Detailed methodologies for monitoring birds, including mark-recapture studies, exist for many of the species of concern in the LDW, and could provide important food web information.

Considerably more dietary information on diving ducks is available in the literature, and more specifically from the Pacific Northwest, especially British Columbia (Butler and Vermeer 1994, and references therein). Several species of waterfowl that occur in the LDW are extensive molluscivores and benthivores, which places them in a position to be much more exposed than other waterfowl. Extensive study of contaminant loading in surf scoters has been performed in Commencement Bay demonstrating uptake of contaminants over the wintering period (Henny et al. 1990).

Loons and grebes are not considered waterfowl. Along with mergansers, they may be increasing their usage of the LDW (despite their overall decline in Puget Sound) as they focus their foraging patterns in space and time with salmon hatcheries. Studies in Commencement Bay with western grebes has also shown overwintering accumulation of metals and organochlorines (Henny et al. 1990).

A.2.2.4.3: Raptors

Page 31: The Buehler 2000 reference is missing. It is doubtful that shiner perch are a major prey item in the LDW for eagles as they are quite small. The July 1998 King County CSO/WQA and Appendix E presents no detailed information on bald eagle food habits that define shiner perch as a major prey item. Prey item studies may have been performed at the eagle nesting studies at Discovery Park relating to the expansion of the Westpoint Treatment Plant - those studies are available at the King County DNR library or on the web (King County 2002) and should be included here.

A.2.2.4.4: Shorebirds/waders

Page 32: It does not appear that the Matsuda et al 1969 study of fishes of the Green-Duwamish River discusses the importance of shiner perch to the survival of juvenile herons. We presume that the reference is to the presence of shiner perch in the LDW.

Section A.2.3.2.3: English Sole

Page 42: The RI states "English sole is one of the most abundant fish in the LDW and is closely related to starrry flounder...." One of the primary aspects of the inadequacy of the interspecies safety factors used in the RI can be demonstrated by the differences in the uptake and transformation of PAHs between these two species, as well as the rates of tumors (Varanasi et al 1987). For the many species of fish that are different in the LDW that are intended to be represented by English sole, such major differences between species requires a large set of safety factors for cross species sensitivities, in addition to the factors used for sensitive individuals within a population.

Section A.2.3.3.2: Bald Eagle

Page 44: Bird tissue data for nearby Commencement Bay are available. PCB concentrations in dunlin, a prey item of peregrine falcons, are also available for Puget Sound (Schick et al 1987).

Section A.2.5.2: Food Web

Page 101: The web model is lacking a line between piscivorous and omnivorous mammals and piscivorous/omnivorous birds. Eagles are known to feed on marine mammal carcasses and also potentially take mammal prey. The web model also lacks a connection between zooplankton and benthivorous/sediment probing birds, as some shorebirds may take larger epibenthic zooplankton. While not a major system in the LDW, some waterfowl (e.g. brant) consume significant amounts of macropytes, which with restoration could be a major pathway.

Section A.4: Exposure and Effects Assessment: Fish

Section A.4.1.1: Tissue Data

Changes from the King County CSO-WQA for TBT Effects in Otters

Page 142: TBT was also dropped for birds and mammals, and the levels found in an otter (Grove et al 2001) stimulated DRCC to investigate the issue again. DRCC has not performed a rigorous library search, as was suggested to the LDWG after the problem formulation (see comment #115 -Section 5.4.1.1 on lack of TBT studies in birds in the EPA and Ecology Comments on LDW Scoping Phase ERA Problem Formulation), as the only studies located were those recommended in the memo. In addition, the King County CSO-WQA used a value from IRIS 1997 for a rat, and scaled the NOAEL to 0.14 mg/kg/day for birds. Levels of TBT were listed in Table 3-4 as ranging from 0.02 to 0.2, with likely higher concentrations in larger salmon and rockfish. Crab was listed as 0.8 as a weighed average for the crab (Table 2-6). Once again, to reach an HQ of 1 the concentration would have to be 0.5ppm, or 0.05 for an HQ of 0.1. The uncertainties in the concentrations of TBT in larger fish, molluscs and crabs makes it likely that TBT should also be reconsidered as a COPC.

Changes from the King County CSO-WQA for Mercury Effects in Otters

Page 142 and Page 144, Table A-4-3: Mercury was not considered as a COPC in the RI. A review of the process was initiated by DRCC and finds several inconsistencies with the RI from the King County CSO-WQA. The first problem with the mercury data is that the TRV is based upon inorganic mercury. Otters would likely be consuming almost entirely methyl-mercury. The scaled inorganic NOAEL for mercury given (Page D-42) is 0.06 mg/kg/d. References given were Carmignani et al 1989, USEPA 1993, Wobeser et al 1976, and Dieter et al 1983. None of these references were given in the report. they are assumed to be:

- Wobeser et al. 1976. Mercury and mink II. Experimental methyl mercury intoxication. Can. J. Comp Med. 40: 34-45.
- EPA 1993. Biofactors handbook.

Using the calculated food consumption rate for otters (Equation 6 from the Exposures and Effects Report, 0.281 kg/day) and then multiplying by an average mercury concentration of 0.22 mg/kg wet weight in flesh eaten would given a HQ of 1. For the screening purpose, a concentration of 0.022 mg/kg would give a HQ of 0.1. Levels found in O'Neill et al 1998 and West et al 1998, averaged from 0.06 mg/kg in the flesh. Using the 95th UCL from Table 3-4 in the EEA (Page 44), these values ranged from 0.09 for perch to 0.08 for English sole (Page 144 of the RI), which are between 0.1 and 1.0 for a HQ. Crab was estimated from LDW samples to be almost 0.2mg/kg ww as an organ weighted sample, however it is likely that an otter would consume more of the hepatopancreas than the muscle tissue as it is more accessible and thus more likely a higher value.

Section A.4.3: Regional Field Studies

Pages 193-205: Disease and immunocompetence need to be included as endpoints in the ERA. There are connections between these endpoints in the literature. Endpoints relating to PCB effects upon thyroid function have been used to relate to survival of seals in the Wadden Sea (Reijenders 1989) and more recently shown to be similar to reproductive outcomes in otters (Trass et al 2001, Murk et al 1998, Kester et al 2000). The recent NOAA immune studies, do indicate that disease increases due to a compromised immune system may have a link to survival of juvenile salmon (Varanasi et al 1993, Stein et al 1995, Arkoosh et al 1998 and references in RI in Section A.4.3).

Section A.4.3.2.2: Growth

Page 203: It is not clear why the Kubin 1997 study was trivialized. The paper does not appear in the references. It is also not clear what reference the Johnson et al 1998 study refers to, as it is also not in the reference list. Considering that growth is a major endpoint for consideration of effects, and since several other fish species were excluded from further consideration of the same COPCs that were retained for English sole, these are important references to provide.

Section A.4.4: Summary of Fish Assessment.

Page 205: Many species of piscivorous fish in the LDW are long-lived and significantly different from the bull trout in their life history and exposure. The RI is not sufficiently conservative, and suggests that the list of COPCs carried forward in Phase 2 for the fish piscivore is incomplete. Can the exclusion of PAHs for bull trout be justified when there is data from the LDW for rockfish (West et al 2001) that indicate major exposure to PAHs through the measurement of biliary FACs?

It appears that The RI continues to underestimate exposure by omitting lifespan considerations in the exposure assessment. If so, this eliminates the existing body burden as an accumulated dosage for compounds that can be retained in the body and mobilized during spawning or passed onto offspring at effects levels (PCBs, mercury).

Exposure and Effects Assessment: Wildlife

Section A.5: Scaling and Safety Factors in HQ TRVs.

Page 208: In examining the previous documents, DRCC can only find a few instances of detailed discussions on the approach of scaling toxicity study data and applying safety factors to adjust for different species used in the effects studies. Where adequate factors are used to adjust exposure (Page 209), there is no effort to make adjustments in the toxicity data. In the King County CSO-WQA (Appedix D, 1998) there was a limited discussion on scaling and safety factors. Are there other locations where this issue was discussed? There are fewer complications in the scaling with the otter data, because of the similarity between otters and mink. The LDWG makes a point to demonstrate that otters may not be as sensitive as mink to PCBs, but neglects to mention that the toxicity data used may not be as applicable in birds. There are major scaling issues for birds and yet the safety factors used in the CSO-WQA were from 2 to 5, without much rationale. DRCC considers these to be quite low. Despite major phylogenetic differences between avian species used in most of the toxicity tests on birds, (the King County CSO-WQA mentions only one non-peer reviewed poster at SETAC as a reference), and there was no rationale given for the differences between herons and eagles. This is not a very systematic approach, as it seems to encompass two types of scaling factors which should be considered separately, body weight corrections/physiological factors, and factors to protect sensitive individuals in the populations. Why would a heron have a different safety factor to protect individuals than other species?

The three birds that have been chosen as ROCs in the LDW, the bald eagle (Falconiformes), spotted sandpiper (Charidiiformes) and great blue heron (Ciconiiformes) are all phylogentically different from the three orders of birds typically in toxicity studies, Galliformes (quail), Anseriformes (mallard) or passeriformes (perching songbirds). There should be some safety factors to address the differences in sensitivities. At a minimum, a factor of 5 should be used across all species for individual sensitivities, and a separate factor of at least 5 based upon size and metabolism should be applied to obtain a general scaling factor of at least 25.

Due to levels of sensitivity, and because the three avian ROCs are also surrogates for a large number of other bird species, this safety factor is conservative. If a screening level for the

HQ was also set at 0.1, a much larger number of COPCs would have been retained in the Problem Formulation.

Section A.5.1.2.3: Eggs

Page 214: Recent data provided from heron eggs collected in the LDW in 1998 point to the flaws in the exposure assessment. Not only does the LDWG underestimate the HQ from this recent data, but they also make several errors in their presentation. It is hoped that other such compilations of data are more carefully reviewed for substance.

Page 215, Table A-5-5: The calculation of the concentrations of the PCBs in two of the egg samples were listed in Table A-5-5 incorrectly. The concentrations in Samples 2 and 5 are based solely upon the chick concentration in the almost hatched eggs. The yolk sample for Sample 5 was lost and the yolk sample for Sample 2 was 70ppm. Depending upon the individual sample weights of the yolk and chick fractions, they need to be proportioned to estimate total egg concentration. Unfortunately, this analysis was not conducted. For example, if the chick and yolk portions were equal in weight, the true concentration of Sample 2 would be 50ppm. Because yolk has a much higher concentration than chick (Norman 1991), it can be predicted that both Samples 2 and 5 will be significantly higher in concentration. Yolk samples in a previous study were as much as 4 to 10 times higher than chicks, depending upon the development stage of the chick and whether the chorion-allantoin membrane (CAM) was attached to the chick or the yolk. The CAM holds a significant amount of contamination (Norman 1991, Cobb et al. 1994, Cobb et al. 2000). Sample 5's concentration could be as high as 10ppm. These samples are extremely high, but examining the samples collected in 1984 at the same location confirms these values, as the mean concentration was 15ppm based upon only the A-1260 concentration.

Section A.5.2.1.3: Lead

Changes from the King County CSO-WQA for Lead Effects in Birds

Page 231: The lead TRV LOAEL values in birds ranged from 0.14 to 0.72mg/kg/d in the King County CSO-WQA (Table D-20 in the Appendix D of the 1998 Draft is unclear). This is significantly lower than the 20 mg/kg bw/day used in the RI. Please explain why the studies, Scheuhammer 1976 and Edens, et al 1976 (which were not cited in the draft CSO WQA document) were not used. These values are up to 100 fold lower.

Section A.5.2.1.4: Mercury

Changes from the King County CSO-WQA for Mercury Effects in Birds

Page 234, Table A-7-26: For mercury effects in birds, the DRCC urged examination of the ET&C issue on mercury in our Exposure and Effects Assessment comments, as there were lower effects studies listed. A study by Heinz (1979) used a LOAEL of 0.065mg/kg/day. No scaling

between species was performed and no safety factors, so the results from that study, performed on ducks, could have scaling factors for much smaller birds like shorebirds.

Section A.5.2.3.2: Arsenic

Changes from the King County CSO-WQA for Arsenic Effects in Otters

Page 247: The NOAEL for Arsenic in river otter is based upon a rat study by Shroder and Mitchener (1971). In the King County CSO-WQA (1998) Appendix D, the same study has a scaled NOAEL listed as 0.052mg/kg/day. Why was this scaled value ignored? The form of exposure also appears not to have been considered.

Section A.5.2.3.3: Lead

Changes from the King County CSO-WQA for Lead Effects in Otters

Page 247: The lead TRV NOAEL values in otters was given as 0.15mg/kg/d (Table D-20 in Appendix D) , which is significantly lower than the 0.33 mg/kg bw/day used in the RI. Please explain why the Eisler (1988) and Clark (1979) studies (which were not cited in the draft CSO WQA document) were not used? There was no information about the applicability of scaling given for Lead.

Risk Characterization and Uncertainty Assessment

Section A.7.3.1: Risk Characterization for Wildlife

Page 321, Table A–7–27: In the calculation of Egg HQs for heron, the values used by LDWG were different than those used in a Fox River study, surprisingly based on the same sources. The LDWG uses a NOEC TRV for PCBs based upon a TEQ method as 0.5µg/kg, and a LOEC of 1µg/kg, which is higher than the values given by Hart et al 1991 of 0.02µg/kg NOEC and 0.25µg/kg LOEC (though a later paper, Janz and Bellward 1996 has a NOEC of 2µg/kg). Where did the values of 7.1 and 16 used in Table A-7-27 come from?

Section A.7.3.2.2 Exposure Assessment

Page 327: The spotted sandpiper was assigned a high sediment intake as well as 100% of its diet as amphipods, which explains its retention of many of the COPCs. It is not clear whether many of its surrogates, such as dunlin, a common wintering sandpiper, would have a higher HQ if it were considered to eat 100% of its diet in molluscs (see previous comments from DRCC on Exposure and Effects Assessment). Are concentrations of As and Hg in small clams higher than amphipods in LDW samples? If so, then mercury and arsenic could have higher HQs.

Page 326: Regarding high levels of PCBs in heron eggs, a primary method of increased exposure to eggs through maternal transfer would occur from not only current exposure, but

historic exposure that is stored. While some attempts at addressing this issue are in the Exposure Assessment section, the approach needs to be systematic and consistent across species. It is difficult to determine the effectiveness of these data without having both a good appreciation for local BCFs, the food chain applicable to the local one in the LDW, and the variability of sample concentration. It is also equally important to address variability in diet in some species like bald eagle, as certain changes in diet may provide vast increases in exposure. This approach is not addressed. A table of all the BMFs, changes in diet, and uncertainty, similar to Table A-7-29, but with numbers, would be much easier to follow.

Page 329: Site Usage: The ERA appears to suggest that herons may be obtaining PCBs from other sites than the LDW. The logic here is transposed. While it is true that herons are known to forage as far as 10 miles from the nesting area and these sites may be contaminated with PCBs, it is unlikely that there are other locations where herons could accumulate such high levels of PCBs as occurred in two of the egg samples. In fact, the more time that herons are shown to forage at other clean locations, the more flawed is the exposure assessment regarding uptake of PCBs in the herons.

Page 330: Tissue Data: TBT should be investigated to be retained as a COPC for otters. Grove et al (2001) analyzed 22 trapped otters in Puget Sound in 1996, including one 7 year old animal that was trapped at Fort Ward on Bainbridge Island, which is only 13 km from the mouth of the LDW, an easily accessible distance. The otter has a liver concentration of 2.6 ppm (wet weight) total butyltins, of which the majority was monobutyl and dibutyl tin. How does this level relate to the concentrations in the liver in studies indicating effects needs to be addressed? Do levels of mono- and di-butly tins in the liver mean exposure to TBT?

Page 332: Tissue Data: The LDWG questions the quality of the heron egg data as a means to dispute it veracity. First, the HPLC-PDA method is a well established method (Krahn et al 1994) and is currently used by the PSAMP program as a standard protocol. While egg tissue is somewhat different than the previous fish tissue and marine mammal tissue, some of these samples also have similar extraction and recovery problems as the egg samples (G. Ylitalo, personal communication). Second, it is likely that substantial portions of the data used for the entire LDW study would be eliminated if recoveries were questioned as they are in section A.2.3.2.2 (Page 332, paragraph 3 of the ERA). While recoveries were problematic, this information was presented, which is not the case for any of the other data in the study. In fact, several of the heron egg samples were so high that they caused some careful examination of the samples, and because these samples were performed for the Damage Assessment Program, they were carefully analyzed. It is likely that they may be reanalyzed if they can be relocated to also help recalculate the whole egg concentration.

A.7.3.2.2: Great Blue Heron

Exposure Assessment for Birds Exposed to Lead

Page 340: Please explain why higher lead values from the PSAMP data set were not used, such as large dead rockfish or English Sole which could easily be scavenged by bald eagles. As with otters, meals from large dead rockfish may provide a much higher lead concentrations, as well as containing organs with higher concentrations. Using the mean value of English sole for muscle tissue (West et al 2001a), which ignores the consumption of bone and other tissues containing more lead, the exposure dose is 2X what is given in Table A-7-26. If a LOAEL of 0.14 is used, the HQ for the LOAEL is 0.14, much higher than the value in Table A-7-26 and at a screening HQ of 0.1, so it should have been retained into the RI. Using a NOAEL the value would be greater than 1.

A.7.3.3.4: River Otter

Exposure Assessment for Otters Exposed to Lead

Page 341: The assessment does not present any information on prey size, and it is well known that lead may increase with size in older fish. It is also not presented whether any incidental bone consumption is considered in the exposure assessment, as bone may have higher levels of lead.

Exposure Assessment for Otters Exposed to PCBs

Page 341 PCB concentrations in otters collected from Bremerton (Grove et al. 2001) were also quite high. Levels of Aroclor 1260 in otter liver ranged as high as 30ppm (ww). Some of the PCB congeners also indicated exposure to high TEF containing congeners such as BZ#126 (0.070-0.090 ug/kg) andBZ#169 (0.080-0.130 ug/kg ww), which could also be used to determine how reasonable the exposure assessments were estimated to be. This is an additional factor to be concerned with as otters may move between these locations already carrying a significant PCB body burden.

Section A.7.3.3.2: Great Blue Heron

Page 339: A major factor in formulating good risk assessments is not just looking at the highest values, but also at the lowest. What are the distributions like? How do they compare with other locations where samples have been taken? One of the surprising and disturbing facts of the heron egg PCB concentrations is the high concentrations of the lowest samples, which were 1.9 and 2.1 ppm (ww). These lowest concentrations are significantly higher than all of the recent PCB samples collected at the Samish Island colony in Padilla Bay in 1994 and 1995 (Thomas and Anthony 1999). The geometric mean for 5 heron eggs from 1994 was 0.22ppm (0.09-0.84 Range) and 0.4ppm (0.32-0.46 Range), both a background level. Coplanar PCB congeners (BZ#s 77, 126 and 169) are also available for these samples, as well as dioxins and dibenzofurans. Historic samples of PCBs tend to have a bimodal distribution, which has not been explained.

DRAFT PHASE I HUMAN HEALTH RISK ASSESSMENT

GENERAL COMMENTS

The Human Health Risk Assessment (HHRA) follows generally accepted practices and guidelines for human health risk assessments, notwithstanding the weaknesses of same. The HHRA uses existing data to survey concentrations of chemicals in water, sediment and fish tissues and then estimates exposures through several pathways. No new data were gathered in this effort. Existing data on chemical toxicity and regulatory standards are compared to the estimated or modeled exposures to derive a risk. Not all of the chemicals on the list of potential chemicals of concern were assessed. The pathway analysis is not complete, the list of chemicals is reduced greatly and the toxicity data are selected, rather than comprehensive. However, it seems that the charge to the authors was to proceed as written, rather than to comprehensively predict risks.

It is not clear how the HHRA takes into account the existing body burdens, and effects of contaminants on human health. Supposedly, the lead modeling includes blood lead levels for age 6 months at a national level, but this is not indicated in the HHRA. Some chemicals will remain for long periods in body tissues and slowly release into the blood, other tissues, etc. so that the new exposures (that this HHRA seeks to estimate) will add on to some dose greater than zero. In that case, the incremental exposure and effects results in cumulative exposure and effects that is not predicted by standard models. The other problem is that some effects are additive or synergistic, so that once damage occurs, the body does not recover, as in neurodevelopment. This is the case with lead.

1. Blood Lead and lead exposure estimates

The HHRA does not comment on how the IEUBK model was used and specifically what data were used as inputs or assumptions. According to personal communications (EPA), the model used data from national surveys for children 6 months of age. The data from CDC (National Report on Human Exposure to Environmental Chemicals; Centers for Disease Control and Prevention; Atlanta, Georgia; March 2001) show no data for children that age. Data from CDC do indicate that mean blood lead level is 2.0 ug/dL, but the 90^{th} percentile, a more conservative measure of population blood lead for estimating exposures, is 4.7 ug/dL.

The CDC web site and fact sheet indicate that low levels of lead in blood can cause significant effects, and that no truly “safe” level of lead exposure has been found to date. Current literature provides data on lower lead levels that are associated with effects in young children. These data need to be included in the HHRA. If, in fact, existing blood lead levels

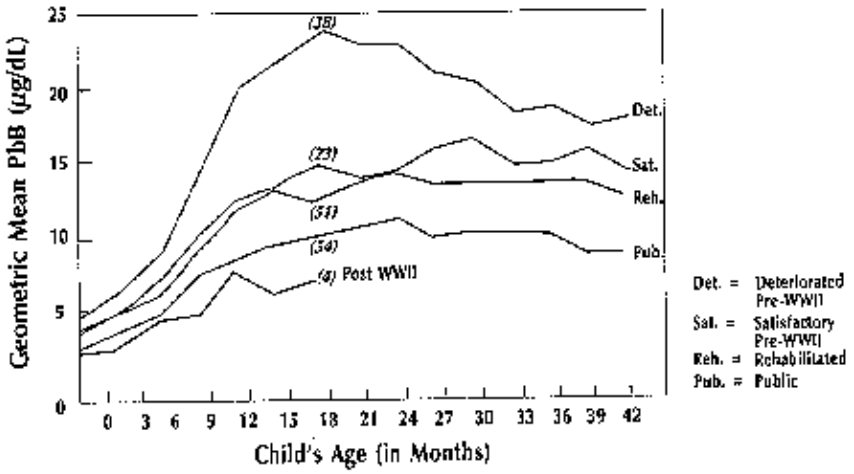
are already problematic and any additional exposure will further exacerbate an unacceptable public health situation, then the HHRA needs to take these facts into account.

The HHRA should use the 90th or 95th percentile for blood lead in children, as a more conservative estimate, unless and until data are collected from Washington State that are more specific to the local population.

Washington State Department of Health (DOH) completed a report on childhood lead levels (Washington State Childhood Blood Lead Screening Recommendations; November 2000.) and found that 4.0% of children in King County had lead in excess of 10 mg/dL, the value recommended by CDC as needing treatment.

Furthermore, Clark et al (1985) demonstrated that blood lead does not peak in young children until 18 – 30 months of age (Figure inserted below).

Relationship between children's blood lead levels and housing age and condition, Cincinnati



Note: Number of children at 18 months of age indicated in parentheses.
Source: Clark et al., 1985.

Clark CS, Bornschein RL, Succop P, et al. 1985. Conditions and type of housing as an indicator of potential environmental lead exposure and pediatric blood lead levels. Environ Res 38:46-53.

Clearly, the data from CDC and Washington State are consistent and indicate that a significant portion of the child population already has blood lead in excess of a "safe level" of 10 ug/dL and that an even greater proportion has blood lead likely in the range of 4.0 ug/dL or greater. The IEUBK model must account for these existing elevated blood lead

levels. If possible, the model should use data from the Washington State Department of Health.

2. Arsenic

Two issues with the arsenic contamination arise in this RI/HHRA. The first is whether fish and sediment samples indicate elevated arsenic levels over ambient or background (which are not the same) levels. The second is whether the 20 ppm standard in residential soils is sufficiently protective of public health.

The existing data indicate arsenic levels are significantly elevated over reference conditions, the reference conditions being in the region but outside the study site. The HHRA questions whether the elevated levels can be attributed to the site and site related sources, or whether the arsenic is actually from other sources, specifically, Asarco. The HHRA proceeds to dismiss the elevated arsenic levels, attributing them to another source. The nature, location and type of source are not the question in the HHRA. The question at hand is whether the existing conditions pose a health risk. Clearly, the arsenic levels in the sediments are elevated and exceed regulatory values. The percentage of the arsenic due to a specific source can be assessed in the subsequent analysis by using fingerprinting of chemicals in soils and sediments.

The second question is whether the 20 ppm soil standard is protective. The National Research Council/ National Academy of Sciences recently completed two reports on arsenic in drinking water (NRC, 2000; *Arsenic in Drinking Water*; National Academy Press, Washington DC and NRC 2001; *Arsenic in Drinking Water 2001;* National Academy Press, Washington DC), in which the NRC concluded that the toxicity of arsenic was greater than previously estimated, based in large part on cancer potency in several human populations around the world. In addition, EPA examined risks posed by arsenic in CCA treated wood, commonly used in playground equipment for children (U.S. Consumer Products Safety Commission Memorandum, August 2, 1990). Similar assumptions about ingestion of soil in playgrounds can be applied to ingestion of sediment at parks and beaches.

The HHRA/RI must consider that arsenic may be more potent than presently estimated and evaluate lower clean-up levels than the 20 ppm.

Furthermore, the speciation of arsenic in the environment and living tissues makes the analysis more complicated, requiring either additional safety factors or different assumptions. Arsenic is considered toxic in the metallic form only, yet it can be converted between

metallic, oxidized and organic forms, so that the conversion has to be taken into account when evaluating site-wide toxicity issues.

3. Children's Health

The major issues here are that the HHRA relies on toxicity endpoints and values that were not set for children. The safety factors are insufficient to account for the differences. Children are not just little adults, as EPA observed when forming the Office of Children's Health. Lead poisoning is an excellent example. Children are sensitive to lead poisoning and the effects are permanent. Adults are not sensitive to lead and tolerate orders of magnitude more than children, in large part because the most sensitive tissue – the developing brain – has already formed in adults, and is no longer amenable to developmental alteration. The health effects in the databases are not protective for children unless the experimental and epidemiological data used to set the standards (RfD's, RfC's) were intended to model children.

4. Soil Standards vs Washington SMS and sediment standards

The HHRA uses EPA risk based standards as the basis for screening marine sediments and fish tissue in the LDW. The HHRA notes that EPA values are both more complete and lower than Washington State's and that Region X does not have such values for fish tissue or soil. Instead, the HHRA uses Region IX soil and Region III fish tissue values. Tissue and soil screening values are divided by 10 because Region X uses an HQ of 0.1 for screening COCs. However, no adjustment is made to adjust for or compensate for the difference between soil and sediment.

This method is questionable. The use of soil standards raises a host of bio-geochemical process issues that are not addressed. One substantial difference is the fact that soil is largely aerobic (oxygen is present), while marine sediments are anaerobic (no oxygen) below a certain depth. Other differences include the temperatures, native bacteria and other microbes that breakdown or absorb contaminants, presence of vascular plants in soil, etc. The HHRA uses EPA's standard screening level of 0.1 as the total of its safety and uncertainty factors by dividing these soil values by 10 – this has to account for *all* of the differences between soil and sediment, as well as the usual uncertainties. The HHRA needs to offer a more conservative and plausible approach. At the very least, an additional safety factor should be considered to account for the methodological unknowns.

5. PCBs & Salmon

PCBs and other contaminants in LDW salmon need to be considered in relation to the HHRA and the Washington State Department of Health Public Health Assessment. Both the DRCC and WA DOH (Rob Duff, personal communication) consider the lack of congener specific data in salmon to be a substantial data gap with potentially significant implications for human health.

New data for salmonids in the Columbia River emphasize need to re-evaluate PCB levels in salmon for possible human health effects. The recent findings on the Columbia attribute highly elevated cancer risks to PCBs in salmon that are consumed by tribal fishermen. The consumption rate used is 48 meals/month, which may be comparable to certain population segments' consumption of salmon on the Duwamish. An initial review of the data show that the Columbia River salmon that have been determined unsafe for tribal consumption may have tissue concentrations lower than LDW salmon.

If LDW fishers are eating PCB contaminated salmon (regardless of source of the PCBs), this exposure must be considered in an analysis of pre-existing body burdens.

6. Fish/Shellfish Consumption Rates May be Underestimated

DRCC remains concerned that fish and shellfish consumption rates may still be underestimated, especially when Asian American and Pacific Islander consumption habits are taken into account. In addition, the HHRA should provide specific health risk information for different consumer groups.

SPECIFIC COMMENTS

Executive Summary

Page 1: Will Phase I include actions to protect people from further contamination? Such actions may include issuing warnings against fish consumption or prohibitions against recreational or commercial use in any of the potentially affected waters until "safety" has been assured. Will actions be taken to prevent further pollution, e.g. eliminate combined sewer overflows, control discharges of toxic chemicals, and install best management practices on stormwater systems?

Page 2, top: Surface sediment samples are likely not sufficient for unconsolidated sediments that will easily resuspend in propeller wash.

Muscle tissue analysis and composite sampling is clearly insufficient for crab, as noted in this HHRA on page 53, due to methods of cooking and consumption.

On the bottom of the page, please provide a reference for the statement on Suquamish fish consumption.

Page 3, ES 4: The statement that non-carcinogenic chemicals all have a threshold for effects is either probably or certainly not true for lead, mercury, dioxins, PCB's and may not be true for other chemicals.

Page 4, middle: The RI states that the HHRA is conservative *and* may underestimate some risks. This pair of statements is at least confusing, and at worst is contradictory. It should be amended to articulate the facts: that the HHRA used as many conservative assumptions as possible, yet still may not accurately estimate risks – some may be greater, others less than estimated here.

Page 5, top: Mussels and fish do not take up PAH's in similar enough fashion to treat them the same.

Page 6, footnote 6: 15 cm is not deep enough for sediment samples. Unconsolidated sediments will be disturbed by prop wash, resuspending material from that depth or greater, depending on the vessel and speed.

Pages 11, 14: Excluding whole crab, hepatopancreas etc., and using the muscle only, is not appropriate for several reasons. Crabs are cooked whole, in water, or steamed in water, and the process can remove (leach) contaminants out of the organs, even if the organs are not consumed. Second, the HHRA has to provide data that the internal organs will never be consumed by anyone in the area, because in some regions and among some consumer groups, the hepatopancreas is considered a favored item and may be consumed.

There is a statement in the HHRA that some groups are known to consume the internal organs of crustaceans, and of course, all the parts of a mussel are consumed. In addition, the ERA must use whole animals to accurately reflect prey item body burdens; osprey and eagles do not fillet fish before eating.

Page 16: The % inorganic arsenic in food – specifically in fish and shellfish – is as per the National Research Council (1999; *Arsenic in Drinking Water*; National Academy Press, Washington DC).

Section B.2.3.1.2: Tissue

Page 18: The HHRA observes that non-resident fish that move in and out of the study area (LDW and Elliot Bay) will give an indication of contamination throughout their normal migratory range. This point is true, as is the fact that animals indicate local conditions to the extent that they reside and interact with the media of interest. The HHRA must not confuse the issues of the fish indicating local conditions, and the fish being "safe" for human consumption. *The HHRA needs to address both.*

B.3.1: Summary of Previous Exposure Assessments

Page 22: This section gives some information on the decision to exclude surface water as an exposure pathway and to select only a small number of chemicals for analysis. Neither is sufficiently complete to convince DRCC that the omission and data reduction are appropriate for this activity. Indeed, it seems that the Washington Department of Health (Draft Public Health Assessment, Lower Duwamish Waterway, July 9, 2002) considered a more comprehensive list of chemicals in their health assessment.

Page 24: The Conceptual Model includes surface water, but this pathway is not examined in detail. In addition, the breathing pathway is ignored entirely. While breathing is not usually a major pathway for exposure of metals and non-volatile organic chemicals, the extent of activities on the LDW and the size of the site make the possibility real that inhalation may occur.

It is not clear why the worker on the waterway exposure pathway is listed as not complete. By LDWG's reasoning, the pathway should be listed as complete, but insignificant

Section B.3.2.6: Selection of Exposure Scenarios

Page 26: The HHRA wisely focuses on highly exposed individuals, not the average or central tendency. There is no mention, however, of pregnant women and fetal exposures, which are the most sensitive persons for some chemicals such as lead, mercury, PCBs and dioxins. This is an essential segment of the potentially exposed population that needs to be added.

In selecting the highly exposed individual, the HHRA uses the 95% upper confidence limit, but not the highest value recorded in field measurements. It may not be appropriate to eliminate the upper 5%, as this may represent an especially sensitive component of the

population. Would the risk estimates be higher if the HHRA had used the highest values recorded? Please provide an analysis and comparison, as well as justification for using the 95%ile when considering human populations.

Section B.3.3 Contaminant Screening and Evaluation

Page 30: Two weaknesses in the selection process cause exclusion of chemicals from this HHRA. (1) Chemicals without risk-based concentrations (RBC's) are not evaluated, and (2) those chemicals that are present in especially high concentrations are excluded if only a few (<10% very high values) samples exceeded the RBC. Lack of toxicity data is another reason for excluding contaminants from consideration, although it is not clear what data are lacking.

Relegating these chemicals to the uncertainty analysis is not sufficient when the uncertainty analysis is qualitative and risk decisions are based on quantitative analysis. Excluding data based on the toxicity data presents a circular argument. Toxicity values for some of the contaminants should be extrapolated from related compounds (e.g. PAH's, butyltin's).

The HHRA should not be constrained by the availability or lack thereof of EPA's data on risk-based concentrations. Alternative approaches can be used, using other data in toxicity databases or in the literature. Certainly, NOAELs are effective screening levels when based on endpoints for human health.

Section B.3.3.2: Tissue

Page 34: This section describes the manner in which the HHRA translated the RBC's for tissue to Region X application. LDWG did correct for greater body weight, longer exposure, higher fish consumption and used the HQ of 0.1 instead of 1.0.

Section B.3.3: Contaminant Screening and Evaluation

Page 25: This section describes the method used to select COC's from the longer list of chemicals measured and reported in previous studies of sediments in LDW. While the screening in this case may be intended to only identify the sites for early action, it nonetheless gives a preliminary assessment of the site(s) and should not unduly exclude locations or chemicals. The approach of excluding chemicals without toxicity data must be considered a last resort and alternatives can be considered, such as using the data for a chemically related compound, with an uncertainty factor. In cases where absolutely no toxicity data exist in the formal databases, literature searches may yield additional information. Other data may yield important non-toxicological information on the sites and

extent of contamination, such as location of pollutants. The compound coprostanol is a breakdown product of cholesterol and is commonly used as an indicator of fecal pollution; the high levels of coprosterol reported at some sites (HHRA Subappendix B.2, Table 1, page 136) is important information on pollution location.

Some of the information in the chemical selection Table 1 (page 136 of Subappendix B.2) is not correct or entered wrong. All the TCDD and TCDF congeners should all be included in the TEQ analysis, none should be listed as bsl.

The 10% occurrence criterion has excluded chemicals that are in extremely high concentrations at a few sites, e.g. 1,3 dichlorobenzene was found at only 13/609 samples, yet was present at 190 ppm, vs the 2.1 ppm screening level. Clearly some sites have levels of this chemical requiring analysis and likely cleanup. Similar patterns exist for other chemicals (4-methylphenol, 2-mehtylphenol, dichloromethane). If a few locations have high levels of several compounds, but these all fail the 10% occurrence criterion, as seems to be the case, then the sites will not be identified for cleanup at this stage. This is completely inappropriate, and may result in significant, localized risks remaining in the sediments.

Some compounds have toxicity data but are listed as not having data in Table 1. This is true for 2- hexanone (ATSDR has data), alpha endosulfan (IRIS, ATSDR), acenapthylene (IRIS), benzoperylene (IRIS), and others.

Page 36, top: The HHRA admits that data availability is a problematic issue in stating that 180 chemicals were found in sediments but never measured in tissues.

B.3.4.1: Fish and shellfish consumption rates

Page 43, bottom: The assumption that no clams are present and consumed is not be correct, though the need for more data should be discussed in both the uncertainty analysis and the data gaps report. In fact, Windward's own reconnaissance survey (2000) found butter clams, softshell clams, sand clams, bent-nose clams, and inconspicuous macoma using randomly placed transects and investigations of siphon holes, reporting the highest abundance at Kellogg Island. The HHRA also need to recognize the likelihood of expanded clam habitat and colonization in the future.

B.3.4.4: Lead modeling

Page 62: The lead model does not take into account the existing body burden. The HHRA is silent on the matter; therefore, we assume that the existing body burden has

probably been omitted. Children with 9.9 ug/dL would be considered in the "safe" range, yet only 0.15 ug/dL more would raise blood lead to the level of concern. The average lead blood level for 18–30 month old children in the U.S. is 2.1 ug/dL (CDC web site) It is also quite likely that existing blood levels are higher in the urban/industrial region surrounding the LDW. The HHRA must account for existing exposures and body burdens in determining incremental risk.

The HHRA uses the IEUBK Model to predict blood lead an hence children's risk of lead poisoning, as in many other such assessments throughout the country. The assumptions in the model have to be more carefully screened for relevance to the case at hand. There are two major issues that either have not been addressed in the present analysis or are at least not explained, if they have been considered. First, the IEUBK model does not necessarily account for current blood lead levels in the population in question. The children of the US already carry a lead burden, with some effects and costs. The present position of the CDC is that blood lead less than 10 ug/dL is without serious effect. But if 10 ug/dL is the threshold, then a child with 90% of that dose only requires a dose of 1 mg/dL to raise their blood lead to a level of concern. Because lead is retained in the body, the body burden is critical in determining the total exposure, as measured by blood lead levels. It is not clear from the HHRA if an attempt is made to take existing body burdens account, or what assumptions are made in any effort to do this. (see Additional Info for Blood lead and lead exposure estimates, below)

Second, it is not clear that 10 ug/dL is low enough. The CDC also states that there may be no "safe" level of exposure, and current research indicates that measurable effects can be found at blood lead levels as low as 5 ug/dL. Blood lead levels less than 10 ug/dL can cause neurological problems in children and adolescents according to a recent paper by Lamphear et al (2000), reinforcing the conclusions of CDC. At the very least, the HHRA should estimate the levels or actions needed to achieve the 5 ug/dL blood level in all resident children.

The HHRA assumes that it is acceptable if 1% of the resident children have lead poisoning, as indicated by blood lead <10 ug/dL. DRCC finds this unacceptable.

The HHRA also does not include fetal exposure. For lead, this exposure is critical and contributes to total exposure. Fetal exposures must be assessed.

Page 69, Table B–21: Dioxin is not listed as having a non-cancer RfD, etc. Nor is TCDD listed in Table B-23 for the health effects of chemicals (COPC's) – toxicological endpoints. Both these are known and EPA and ATSDR each published and used 1 pg/kg/day as an RfD.

Lead is not listed in Table 5-21 as having non-cancer effects, despite the fact that EPA and ATSDR each have described the neuro-developmental effects, particularly in children.

Page 89: The Risk Characterization concludes that six chemicals are a health problem: "… the following chemicals were identified as COCs [chemicals of concern] 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 HHRA identifies fish consumption as the principle pathway by which people are exposed.

The basic elements of these conclusions are likely correct – that a small number of chemicals can be quantified as health threats and that fish consumption is the principle way people are exposed. But the HHRA may be in error that other exposure pathways are not as important and that other chemicals are not a health threat. The biggest single issue may well be the toxicity of chemicals with unknown toxicity information or that interact in unknown ways with other chemicals.

Section B.6: Uncertainty Analysis

Page 89: The Uncertainty analysis is qualitative, as expected and in line with the explanation given in the previous chapters of the HHRA. Notwithstanding the qualitative nature, the uncertainty analysis certainly could and should provide *quantitative* analysis of certain assumptions, such as interactions among metals that may have a common mode of action. The uncertainty analysis should also present the results of modifying the input assumptions and other such factors that affect the calculation of risk. Suppose, as an example, that the method of estimating contaminant levels in sediments is actually in error by 25% and that the sediment levels of contaminants are 25% greater than estimated? Such quantitative elements can be addressed in the uncertainty analysis.

Background levels, other sources, and ambient concentrations are all problematic issues in this HHRA. In the case of arsenic, the uncertainty analysis concludes that the presence of and risk from arsenic may be due to other sources or a high natural level. The data do not support this conclusion.

Pages 90 et seq , Table B-30: The uncertainty analysis summary is a good summary of the types of issues that may be a problem and could introduce errors into the HHRA. However, the table has some unsubstantiated conclusions. For example, the arsenic data are not so clearly biased toward allowing the background levels to dominate the risk estimates. Yet, it

is correct that the failure to include chemicals for which EPA has not yet determined toxicity reference values likely causes a substantial underestimate of the actual risks to human health.

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