

0047520



047908

STATE OF WASHINGTON
DEPARTMENT OF ECOLOGY

1315 W. 4th Avenue • Kennewick, Washington 99336-6018 • (509) 735-7581

June 24, 1997

Mr. Robert Stewart
U.S. Department of Energy
P.O. Box 550
Richland, WA 99352



Dear Mr. Stewart:

Re: Comments on *Screening Assessment and Requirements for a Comprehensive Assessment*, April 1997, DOE/RL-96-16, Rev. 0. Draft 47249

Enclosed are the Washington State Department of Ecology's comments on the draft *Screening Assessment and Requirements for a Comprehensive Assessment*.

If you have any questions or concerns, please contact David Holland at (509) 736-3027.

Sincerely,

David Holland
Nuclear Waste Program

DH:rb

- cc: G. deBruler, HAB
- L. Gadbois, EPA
- P. Danielson, NPT
- S. Harris, CTUIR
- T. Woods, YIN
- M. Blazek, ODOE
- R. Patt, ODOE

RECEIVED

JUN 26 1997

DOE-RL/DIS

Ecology's Review Comments on:
Screening Assessment and Requirements for a Comprehensive Assessment:
Columbia River Comprehensive Impact Assessment,
DOE/RL-96-16, Rev. 0, April 1997

Commenters: D. Delistraty, D. Holland, J. Yokel, W. Soper

June 24, 1997

Page, Paragraph	Comment
p. xvi, para 3	The outlier and trend analysis tests require further justification. Convincing rationale needs to be presented explaining why data outliers are not real and why temporal trends are real? For example, is a five or six year time interval sufficient to characterize a temporal trend? Furthermore, is it appropriate to conduct serial outlier and trend tests on small data sets for a given contaminant/segment/medium?
p. xvi, para 4	Elaborate on "spreadsheet" vs. "computer code," addressing advantages and disadvantages of each.
p. xvii, para 3	Use of LC50 as a toxicological benchmark to calculate EHQ is non-conservative and, therefore, inappropriate.
p. xviii, Figure S.1	Regarding the key at the top of the figure, the parenthetical information for the human risk threshold should be (>1E-6 lifetime risk or >0.01 hazard index).
p. xviii, Figure S.1	According to page xix, paragraph 2 this Figure S.1 only reflects Hanford related risk contribution rather than total risks (Hanford + background). I suggest inserting this clarification into the description.
p. xix, para 1	<p>Figure S.1 shows that ecological and human risks are often unique for a given contaminant in a given segment (e.g., only ecological risk flagged for Pb in segments 2, 3, 7, 9, 13, 21). This makes sense due to unique stressor characteristics (e.g., mode of action, bioaccumulation, frequency, timing, spatial and temporal scale).</p> <p>The observation that segments identified via human health analysis having increased potential risk were not always identified in the ecological analysis may largely reflect the reporting threshold scheme (which incorporates a very conservative 1E-6 cancer risk for human health) or lack of data to assess ecological risk (p. xxv, para 3).</p>
p. xix, para 2	Provide rationale for specific reporting thresholds in Figure S.1. For example, why 5% above background segment simulations exceeding the benchmark for chronic ecological effects or why >0.01 for human hazard index?
p. xix, para 4	Please explain why contaminant concentrations in pore water drive body burdens in aquatic species.
p. xxiii, Table S.1	The human cancer risk tabulated for Technetium-99 in Segment 9 is <1E-6 (9.61E-7), so it should not be included in this table, according to the footnote on p. xxiv.
p. xxv, para 3; p. xxvi, para 2	The data gaps acknowledged here make it difficult to interpret information presented in the Executive Summary without consulting the body of the report.

p. xxvi, para 3	Explain how certain aspects of the uncertainty is addressed via Monte Carlo simulation.
p. xxvi, para 4	Although delineating Hanford and non-Hanford sources of contaminants is relevant in terms of liability and potential remedial strategies, it is moot in terms of risk presented to human and ecological receptors.
p. xxvi, para 5	Provide a brief explanation as to why several contaminants in Figure S.1 (i.e., ammonia, diesel, kerosene, phosphate, xylene) were screened as contaminants of concern but were not flagged by the Figure S.1 reporting thresholds and, therefore, not discussed on pp. xxvi-xxix.
p. xxx, para 1	This final conclusion seems very important, yet it is not particularly highlighted. This conclusion on uncertainty in the ecological assessment should be expanded and emphasized to a greater degree.
p. xxxi, para 2	This correlation between sediment grain size and metal sorption is important and should have been considered in determining sediment background. Not including fractional considerations calls into question the validity of using Segment 1 to determine background levels for contaminants in the sediment (see Section 5.2.4.1). Of the 28 sediment sampling locations in Segment 1, three are clearly downstream from Priest Rapids Dam, 24 appear to be in the pool behind the dam, and one location has no coordinates given and cannot be determined (Miley). The Blanton et al study has shown that the sediment behind Priest Rapids Dam tend to be fine and very fine silt while most sediments collected in the Hanford Reach are predominantly medium and fine sand. This same study also that there is a direct correlation between grain size and TOC content with metals concentration. The net result is contaminant background levels for certain metals may be biased high when used for comparison to sediment from areas of the Hanford Reach where the grain size is coarser.
p. xxxi, para 3	Bioavailability of contaminants is a key consideration in predicting ecological risk. In addition to biotic regulation (e.g., metals), data on species composition and physical-chemical factors in environmental media may influence bioavailability. As a result, there may be spatial and temporal variations in bioavailability, as well.
p. xxxiii, para 2	Clarify how it can be ensured that no factor is eliminated or overlooked that would strongly influence study results. This seems difficult, considering current data gaps and fiscal limitations.
p. xxxvii, abiotic	Use of the word "inorganic" seems awkward in the definition for "abiotic."
p. xxxviii, bioconcentration factor	" where uptake is limited to water alone (e.g., respiration, dermal absorption)."
p. xxxviii, biomagnifying	The definition for biomagnifying should be amended to read "... 'primarily' through dietary accumulation," since biomagnification includes processes of both bioconcentration and bioaccumulation.
p. xxxviii, chemicals (toxic)	"Noncancer toxicity" is more appropriate than "poisonous agent," as used here.
p. xxxix, dose	Dose may also refer to chemical (i.e., non-radionuclide) dose.
p. xliv, outlier	"Unlikely" should be "likely."
p. xlvi, risk:hazard index	Note that "risk" here does not denote a probability.

p. xlvi, risk:lifetime risk	EPA radionuclide slope factors in HEAST include fatal plus nonfatal cancers. Please document the definitions stated for carcinogenic chemicals and radionuclides.
p. xlix, toxicological benchmark	The definition of toxicological benchmark should be expanded to state that the benchmark represents an arithmetic or statistical summary of the observations that comprise the measurement endpoint (e.g., NOEC, LC50).
p. xlix, uncertainty	Regarding alternative definitions, it may be worthwhile to contrast uncertainty (lack of knowledge) with variability (variation in nature).
p. I-1.4, para 5	It does not appear that human acute toxicity was assessed in the screening protocol, as stated.
p. I-1.5, para 2	Cite references for risk due to cancer incidence vs. cancer fatality.
p. I-1.5, para 4	Potential limitations to restricting the contaminant database from 1990 to present may have included generation of data gaps for certain contaminants, smaller contaminant sample sizes for statistical characterization and stochastic calculations, and artificially constraining temporal variability of contaminant concentrations. This latter possibility could be important if certain contaminant concentrations may increase in the future due to transport into the area or chemical transformations. In this regard, it is critical that the proposed "comprehensive impact assessment" evaluates <i>future</i> risk along the Hanford reach.
p. I-2.2, box	Clarify when groundwater data were limited to within 500 feet of the river as stated here or a 0.5 mile corridor (p. I-1.2).
p. 2.20, para 5	State how the screening method evaluated chemicals that have no established toxicity values (i.e., no cancer slope factors, RfD, AWQC, TLM, nor LC50).
p. I-2.21, para 4	Equation 1 is missing the 0.25 retention term. Equation 2 is apparently missing the duration term (365 days/2). Please revise these equations and add some clarification in the text on how equilibrium conditions are determined in leaf and root. Also, it appears that the definition for the translocation factor (T) should refer to roots (not leaves), since T is in the root equation.
p. I-2.22, para 2	It appears that a second right parenthesis was mistakenly omitted after CR in equation 4. Although the contribution of contaminant exposure from sediment inhalation appears relatively insignificant, it is inappropriate to multiply this term by the ingestion slope factor, since the exposure is by inhalation. An inhalation slope factor should be inserted for this term. This comment applies to other similar equations, as well. Also, inhalation of radionuclides in air is not considered as an exposure pathway. Please explain.
p. I-2.22, para 4	Except for sediment inhalation, inhalation and dermal contact are not considered as exposure pathways in the screening equations for human carcinogens and noncarcinogens. Demonstrate that these pathways are not important.
p. I-2.23, para 1	Units for C_w should be $\mu\text{g/L}$, not g/L . The sediment inhalation mass loading should be $100 \mu\text{g/m}^3$, not 100g/m^3 . Also, the units for 2E-4 and 2E-6 should be kg/day , not kg . Check other similar equations for these same errors. CPF should be defined here as <i>oral</i> cancer potency factor.

p. I-2.24, para 4	Provide a reference for TLM. Care needs to be taken with this terminology, since TL_m is the median tolerance limit (i.e., LC50) which is obviously different from how CRCIA defines TLM. A more conventional term for the concentration at which effects are first observed is lowest observed effects level (LOEL).
p. I-2.25, para 1	The text indicates that TLM and LC50 address "essentially the same endpoint." This is not necessarily correct, since TLM may reflect sublethal effects (e.g., growth, reproduction), while LC50 is a lethal effect.
p. I-2.25, para 3	Provide citations addressing developmental effects.
p. I-2.26, para 1	Equation 10 should include an ecological benchmark with a citation. An appropriate benchmark may be 1 rad/day which would not affect the numerical "score" value, but would make the screening equation more logical as an ecological hazard quotient. This same comment would apply to other occurrences of this equation too.
p. I-2.35, para 4	Kd in equation 21 should have units L/kg, not L.
p. I-2.43, para 5	Please clarify what is meant by accounting "for over 99% of the relative risk." Is this based on the ranking distribution? Because chemicals appear to be selected on a relative ranking basis and not on the absolute screening "score" per se (i.e., human cancer risks, human and ecological hazard quotients), the screening method may identify chemicals with very low absolute cancer risks or hazard quotients which rate high on a relative ranking.
p. I-2.45, para 1	Please clarify what is meant by accounting "for over 99% of the relative risk."
p. I-2.50, para 3	Regarding terminology, it might be helpful to the reader to contrast radiation exposure (ionization in air in units of μ roentgen/hr) with dose (absorbed energy in any specified medium in units of rad).
p. I-2.51, para 3	Unless additional work has been done, the dose rates in the pipelines should not be given. In the work done by Dunks (see ref.) the suspected electrical problem with the survey equipment in 100-D and 100-DR pipelines invalidated the radiological monitoring effort. If additional work has been done, please reference.
p. I-2.51, para 3	Give the concentrations for Fe, Cr and Hg or reference where they can be found. I did not find them in the data report.
p. I-2.55, box	It is stated that 23 contaminants "passed" the screen. Please explain in the text in more detail the passing criterion after screening scores were calculated.
Section 3.0, general	This is probably one of the weakest sections of the whole report due to the fact that data quality objectives were not defined and therefore data quality cannot be properly assessed. Explain in the report that not all of the data has the same pedigree and this is a large contributor of uncertainty in the assessment.
p. I-3.5, Fig. 3.1	The boundary lines between segments 11/12 and 15/16 are not accurately portrayed.
p. I-3.8, para 1	Again, the radiological surveys on the pipelines (Dunks 1995) were invalidated due to suspected electrical problems which gave some rather unrealistic readings. I recommend the radiological surveys not be mentioned.
p. I-3.9, para 1, sent. 1	I agree with the intent of this statement. However "near-term," is probably not as accurate as "imminent" or "immediate." As one of the Tri-Party managers, I think

	there should be some resolution on the pipelines issue in the "near-term" as compared to other "mid-term" or "long-term" remedial decisions that need to be made at Hanford.
p. I-3.9, Figures 3.6 and 3.7	It appears that the concentration units for cobalt-60 should be pCi/L, rather than $\mu\text{g/L}$? As a clarification to the reader, please explain the negative concentration units.
p. I-3.14, para 2	Pinza 1992, as referenced in section 6.0 synthesis, should have sediment data that represents the Boise Cascade area. Please explain why this data was not included.
p. I-3.16, para 1	Discuss the uncertainty introduced by combining disparate data sets, reflecting differences in data quality (e.g., analytical methods, detection limits, censored data, filtered vs. unfiltered samples). For example, time series data should be of consistent quality in order to perform trend analyses. Are these data consistent? Have analytical methods remained relatively uniform over the data collection period?
p. I-3.31, para 2; p. I-3 32, para 2	It is stated that the confidence level (α) for the Dixon outlier test is 0.05, whereas the α level for the Mann-Kendall trend test was chosen to be 0.01. Please explain why different α levels were selected for these two tests. Furthermore, performing multiple outlier and trend tests may inflate the nominal α level. It seems that the Bonferroni inequality should be applied to guarantee the nominal α level.
p. I-3.42, para 4	If the geometric standard deviation for a contaminant in a river segment is calculated from a set of median values, the true variability is not captured. That is, the input data are median data, not the full range of raw data.
p. I-3.43, para 1	Explain the inverse normal statistic in greater detail, as a clarification to the reader.
p. I-3.52, sec 3.5.2	Why did Diesel and Kerosene show up on the contaminant of concern list if they were never detected?
p. I-3.52, sec. 3.5.2	What does, "considered in reduced detail," mean? I found no discussion of it in Section 5.2 (82 pages long). Please clarify or be more specific as to where in Section 5.2 this information is located.
p. I-3.56, para 1	The emphasis on the geometric mean value for Cr in segment 7 obscures the recognition of preferential channels for contaminant flow resulting in localized areas of significantly higher contaminant concentration. The high pore water values (which appear not to have been used) indicate the likely presence of a previously unidentified Cr plume. Were there any seep samples taken from the area. If not why were the pore water data not included as a surrogate for Cr in seep water? This would be more representative than using groundwater data.
p. I-4.1, para 4	Explain how indirect effects at the population and community levels are addressed.
p. I-4.7, para 1	Thank you for including rationale for the exclusion of microorganisms (except fungi). Despite their ubiquity and purported redundancy in function, however, the uncertainty introduced by excluding this group of organisms should be acknowledged.
p. I-4.10, para 3	Regarding the fifth criterion (i.e., availability of toxicological data), note that lack of data does not necessarily equate with lack of ecological significance. That is, it should be explicitly acknowledged that some species may lack toxicological data but are ecologically significant.

p. I-4.15, Table 4.5	Dermal exposure is an awkward term for primary producers here and in other Tables.
p. I-4.18, para 1	Biomagnification does not apply to the first trophic level (i.e., primary producers). Explain the basis for grouping contaminants as biomagnifying vs. non-biomagnifying. Is the octanol-water partition coefficient (K_{ow}) used for organics in this regard? How are inorganics (e.g., metals) assessed for the presence or absence of biomagnification potential? Note too the role of biotransformation in opposing biomagnification (e.g., polycyclic aromatic hydrocarbons).
p. I-4.23, para 1	Use of the LD50 as an endpoint for ranking acute radiation sensitivity may not be a good predictor of chronic toxicity, the more ecologically relevant concern. Also, were LD50 values utilized derived from the same route of exposure with similar exposure duration? For example, comparing oral LD50s (e.g., mammals) vs. aquatic immersion LC50s (e.g., fish) is not strictly valid.
p. I-4.23, Table 4.16	According to HSRAM (Table 4-3), fish and reptiles are more sensitive than birds to ionizing radiation. This conflicts with Table 4.16 scores.
p. I-4.30, bottom box	For completeness, "toxic chemicals" (i.e., noncarcinogens) should be mentioned too in relation to measurement endpoints (in addition to radionuclides and carcinogenic chemicals).
p. I-4.30, para 3	If the objective is to evaluate both acute (LD50) and chronic (LOEL) toxic effects separately, clarify this here.
p. I-4.34, para 2	" $LOEL_{Lo}$ " and " $LOEL_{Hi}$ " appear to be reversed in the sentence.
p. I-4.34, para 6	Why is only a single LOEL estimated for aquatic species but a range ($LOEL_{Lo}$ and $LOEL_{Hi}$) is estimated for terrestrial species?
p. I-4.38, para 2	Explain the pros and cons of assuming equilibrium conditions in food web modeling. That is, contrast steady-state vs. dynamic modeling. Note too the role non-equilibrium processes (e.g., metabolic regulation or episodic meteorological events) in sometimes driving field results.
p. I-4.38, box	You might clarify that the use of the term "risk" here with EHQ does not denote probability, as it does with cancer risk.
p. I-4.39, para 4	What is the rationale for using only LOELs as a measurement endpoint in deterministic analyses vs. using both LOELs and LD/LC50s in stochastic analyses. Selection of benchmarks would appear to be a sensitive variable in determining final results in the screening risk assessment.
p. I-4.39, para 5	Describe "population health data" in greater detail.
p. I-4.39, para 6	Since "much of the ambiguity resulted from lack of information for the specific species evaluated in this risk assessment," the assessment is not particularly benefited by including many of these data-deficient species.
p. I-4.40, para 5	In their uncertainty analysis, MacIntosh et al (1994) distinguish between knowledge uncertainty (incomplete understanding) and stochastic variability (random natural variability). The "comprehensive impact assessment" should identify variables with high knowledge uncertainty and conduct further research to reduce this source of uncertainty.

p. I-4.40, para 6	Explain the basis for the factors 2, 5, and 10 as fractions of the mean.
p. I-4.42, Table 4.20	Define "likeliest." Is this the mean (arithmetic or geo), median, mode, or some other measure of central tendency?
p. I-4.44, para 3	Please give the underestimation value for contaminants in paragraph 3. They are given for other contaminants in paragraph 7 and overestimate values are given in paragraph 5.
p. I-4.44, para 5	From Figure 4.6, it appears that cobalt-60 was overestimated in fish by a factor greater than 5 times, as stated. Also, it is stated that zinc was overestimated in fish, which appears to be the case from Figure 4.6, although Table 4.23 indicates that the model correctly predicted zinc concentration in fish. Please clarify.
p. I-4.47, Figure 4.7	Delete the bars in the figure. They serve no purpose.
p. I-4.47, para 1	<p>Although it is stated that errors generally favored a conservative estimate of exposure, the model underestimated more contaminant concentrations in herbivorous mammals than it overestimated (Table 4.23).</p> <p>It may be appropriate to evaluate model discrepancies (Figure 4.6 and Table 4.23) by providing a brief comparison here as to how the model computed transfer factors (i.e., summarize modeled exposure pathways for fish and mammals) vs. how the studies cited in Table 4.22 calculated these same transfer factors. This comment would also apply to the modeled and published (Thomann et al, 1995) transfer factors in Figure 4.7 for sediment.</p>
p. I-4.48, para 4	Note that "baseline" levels of nutrient metals in Segment 1 (e.g., Ni, Pb, Zn) are not necessarily associated with no adverse effects.
p. I-4.48, para 4 and 5	Using Segment 1 as a baseline for background contaminant levels in sediment is inappropriate unless grain size is taken into account. (See comments on p. xxxi, para 2.) Having high metal concentrations in Segment 1 should be expected given that most of the samples came from the Priest Rapids pool. Given that most of these samples should tend to the fine-sized particles a higher concentration of metals is expected. I doubt the sediment values derived from Segment 1 are valid as a background baseline for most Hanford Reach segments since particle size fractionation was not taken into account.
p. I-4.48, para 5	Is the Mann-Whitney U test the best test to compare contaminant concentrations in Segment 1 to those in other segments? Dunnett's test or a nonparametric version (e.g., Dunn's test) may be better suited to do repeated comparison to a control (i.e., Segment 1). Also, repeated tests require a Bonferroni adjustment, so as not to inflate the overall alpha level.
p. I-4.49, Table 4.24	Define "Period." Beeby (1991) noted that this is a corruption of part of the periodic table.
p. I-4.49, Table 4.25	Provide rationale for selecting a non-conventional significance level of 0.1. Also, what is the χ^2 statistic? The Mann-Whitney U test typically calculates a U statistic.
p. I-4.50, Figure 4.8	I am assuming the nutrient curve is the dotted line and the EHQ curve is the solid line U-shaped curve (my graph is not in color, as indicated in the text). I do not understand why $EHQ > 1$ when $BC < LOEL$ for nutrient metals (left side of the curve), given Equation 4.2. It would seem that the two curves should parallel each other throughout their range, since LOEL is constant. Please clarify.

p. I-4.51, Equation 4.2	Define: BC=body concentration.
p. I-4.54, para 2	Alternatively to tie into Figure 4.11 more clearly, the sentence referring to Figure 4.11 might state that the K_d method produced sediment/pore water ratios three orders of magnitude less than those obtained using groundwater or seep/spring values. Also, Segment 8 should read Segment 18 in the last sentence of this paragraph.
p. I-4.55, para 1	It is stated, "In all cases, ingestion exposure for inorganic contaminants was from one to several orders of magnitude greater than exposures via other routes (Figure 4.12). Consequently, daily ingestion exposures were used as the basis for comparison to measurement endpoints in the chemical EHQs for terrestrial animals." However, data for only five inorganic contaminants and three species are shown in Figure 4.12. Furthermore, ingestion does not dominate exposure for the single organic contaminant shown (benzene) in Bufflehead and coyote. Please clarify. Explain the double standard for treatment of chemical vs. radiological doses for terrestrial species.
p. I-4.56, Figure 4.12	Be consistent on y-axis label (i.e., either use scientific notation or conventional numbering but not both).
p. I-4.57, Figure 4.27	Where are fungi results?
p. I-4.58, Figure 4.28	Where are algae results?
p. I-4.59, box	References to aquatic and terrestrial species are reversed in the description of Figures 4.13 and 4.14.
p. I-4.60, para 1	Not unexpectedly, it is apparent from Figures 4.13 and 4.14 that risk scores were higher for more contaminants when LOELs were used, as compared to LD/LC50s. This should be mentioned and again demonstrates the insensitivity of acute toxicity benchmarks.
p. 4.60, box	It should also be pointed out that the term "uncertainty" has been used to refer to stochastic variation vs. knowledge uncertainty or parameter uncertainty vs. model uncertainty.
p. 4.60, para 3	The paradox is moot if the error in perception is recognized. "All models are wrong, some are useful" (George Box, 1979). The reference to Table 4.28 appears wrong. I suspect this should be Table 4.26.
p. I-4.63, para 3	Estimated (modeled) Cs-137 in whitefish also did not exceed observed body burden, according to Figure 4.15.
p. I-4.64, Figure 4.15	Label y axis with units.
p. I-4.64, para 2	Explain how the sensitivity analysis was performed. It appears that the 25% of variability in body burden accounted for by pore water concentration (described in the text) is only part of the variability contributed by media, since Figure 4.16 indicates 34 and 42% for media for sturgeon and coyote, respectively. I presume the difference is related to contributions to the variability in body burden by other media. Please clarify.
p. I-4.65, para 3	Please elaborate more on toxicokinetic and bioavailability issues, since these are

	key contributors to variation in body burdens.
p. I-4.69, para 2	Cite some literature and explain why dynamic models are not practical for food web models that involve multiple species simultaneously.
p. I-4.69, para 4	What are the "other two methods?" Although one is described as the traditional wildlife approach (straight function of body weight), the other does not seem to be specified? Please clarify.
p. I-4.69, para 5	Reference should be EPA (1996a), not EPA (1996).
p. I-4.70, para 3	It should be noted that contaminants with concentrations below corresponding Segment 1 concentrations may still be associated with adverse effects. That is, non-Hanford-derived contaminants (e.g., Cu, Pb, Zn) may contribute significantly to ecological risk. The issues of ecological risk and Hanford-derived contamination are independent in this regard.
p. I-4.70, para 4	Table 4.31 also shows that nitrate poses no significant ecological risk, relative to Segment 1.
p. I-4.70, para 5	"Elevated" should be "evaluated."
p. I-4.71, Table 4.31	In the table title, "Exceed 1" should be inserted after "EHQs," since LOELs are part of the EHQ ratio.
p. I-4.71, para 2	Cite references for Levene and Welch tests.
I-4.71	Using the Bechtel data to evaluate the adequacy of the surrogated pore water is not a fair comparison. The Bechtel data specifically looks at Hexavalent Chromium at a much lower detection level and delineates more thoroughly the river shoreline where a hotspot was found. The surrogated values are based on a few well locations of mainly total chromium. For screening purposes it would be better to use the Bechtel data for a deterministic assessment of Chromium related risk.
p. I-4.72, Table 4.32	It would be of interest to list arithmetic mean and standard deviations for surrogated vs. measured Cr VI levels, as well.
p. I-4.72, para 2	To be more complete, the statement might add too that the variance and maxima estimates may be biased low.
p. I-4.72, para 5	The first five lines of this paragraph were transposed from the first paragraph on this page in error. Check Segment numbers (and omissions) for cited contaminants in the remaining part of this paragraph. They do not all match data in Table 4.33.
I-4.73, 1st full sentence 1st paragraph	According to Thornton, filtered versus unfiltered data for Hexavalent chromium should not matter since it is water soluble. Gill exposure and risk should be comparable. Hexavalent chromium vs. total chromium should be discussed. Thornton, E. C. 1995. <i>Speciation and Transport Characteristics of Chromium in the 100D/H Areas of the Hanford Site</i> . WHC-SD-EN-TI-302
p. I-4.73, Table 4.33	In theory, filtered contaminant concentration data should always be \leq unfiltered data. This is not the case in this table (e.g., chromium in Segment 3, nitrate in Segment 1). Is this a result of surrogation (combining disparate data sets)? Please clarify.

p. I-4.73, para 2	With surrogate pore water data presented in Table 4.33, it is not true that "in each case" exposure and risk would have been reduced if filtered data were used. This is because a number of filtered/unfiltered ratios in Table 4.33 exceed 100%. Thus, even though this must be erroneous data, using these "filtered data" would have lead to higher calculated exposures and risk.
p. I-4.74, Figure 4.17	Converting and expressing the y axis in units of "ppm" would more clearly relate to the text. The same is true for Figure 4.18.
p. I-4.74, para 1	Figure 4.19 also shows an acute risk from Cu in Segment 20 for aquatic species, and a chronic risk from Zn in Segments 7, 17, and 20 for terrestrial species. In addition, Figure 4.19 does not show a risk in Segment 21 for aquatic species, as stated.
p. I-4.74, para 2	Explain the rationale for grouping Segments according to areas where filtered concentrations were one third or less of unfiltered pore water? Why was one third selected as the cut-off? Does this mean that data were excluded where filtered concentrations exceeded one third of unfiltered concentration? This may not be appropriate. Please clarify.
p. I-4.75, para 1	Note that where acute effects are identified, chronic effects may also ensue over time (but not necessarily the reverse), since $LOEL \ll LC50$. This is the case, since the exposure model is based only on concentration or dose and not on mechanism of action.
p. I-4.76, Figure 4.19	Explain the figure legend in the text. I see no mention in the text of " $\geq 5\%$ above background" for chronic effects and " $\geq 2\Sigma(RRI)$ above background" for acute effects. Also, define RRI. Phosphate is PO_4 , not PO_3 . Also, please explain the white areas in the figure that are not labeled with no data (e.g., Sr-90 in Segment 12).
p. I-4.77, para 1	Reference to Figure 4.14 should be Figure 4.13. Also, ">55" should be ">65," according to Figure 4.13. It would be helpful to explain the LOEL used for Tc-99, since the text specifies a toxic rather than a radiological effect to plants.
p. I-4.77, para 5	Cite Table 4.25 with statistical comparisons. Note too that Co-60 and Cs-137 sediment concentrations were elevated, relative to Segment 1 (Table 4.25).
p. I-4.77, para 6	Note that risks should only be added for chemicals which exert a common mode of action.
p. I-4.78, Figure 4.20	This figure is very confusing, as a result of the number of species and segments. It is difficult to see many of the symbols due to overlap. The word "Segment" should be inserted in the figure legend to correspond to numbers 1-21. The y-axis label should read "Sum of" (not "Sum or"). This figure illustrates that the inclusion of the relatively large number of species seems more to have confused than to have clarified the assessment, especially since most of the toxicity benchmarks, species attributes, and species-by-chemical attributes have been extrapolated.
p. I-4.79, para 1	Explain in more detail the "correction" for filtered vs. unfiltered pore water exposures.

p. I-4.79, para 3	If results were corrected for filtered vs. unfiltered data (as stated at the top of p. I-4.79), why then does the risk attributed to Cu and Zn in Segment 4 remain suspect? Please clarify.
p. I-4.79, para 4	Again, I thought unfiltered data were corrected, as in preceding comment. Explain please.
p. I-4.80, Figure 4.21	Again, "Segment" should be inserted in the figure legend to correspond to numbers 1-21.
p. I-5.1, para 4	Thus, exposure scenarios for human health risk assessment are largely hypothetical, as opposed to ecological exposures. This is an important distinction between the human health and ecological screening assessments.
p. I-5.2, Table 5.1	Water ingestion should have units of L/day.
p. I-5.6, para 1	Explain in more detail the omission of groundwater as a drinking water source.
p. I-5.7, para 3	Describe more directly that both deterministic and stochastic analyses will be conducted.
p. I-5.20, para 2	Keenan et al in Paustenbach (1989) list deer venison as 45 kg. Thus, the 50% reduction factor should not be used. This would boost deer consumption to 30 g/day. HSRAM lists 1 g/day for game ingestion, considering only the fat content in venison and a two person family.
p. I-5.28, para 2	The fish consumption rate of 540 g wet wt/day seems high. For example, assuming salmon is roughly 20% protein of wet wt, this would yield 108 g protein/day which is approximately 1.9 times the recommended dietary allowance (RDA) for protein intake for adult males.
p. I-5.28, para 4	Similarly, the "animal protein" intake (150 g wet wt/day) appears high, especially if it is in addition to the fish protein intake. "Animal protein" is a misnomer here, since it is described as containing fat and marrow, in addition to protein. At any rate, note that in combination with the relatively high incidence of diabetes in Native Americans, excessively high protein intake could lead to renal failure in this population.
p. I-5.29, para 7	As mentioned, exposure to nursing infants from mother's breast milk is potentially significant. The lactation pathway should be included in the screening assessment, since lipid soluble substances may compartmentalize into milk and infants represent a sensitive subpopulation.
p. I-5.31, footnote h	Again, "animal protein" is a misnomer if it includes non-protein constituents.
p. I-5.38, para 2	The specific equations for cobalt-60 particle exposure should be developed more formally mathematically and incorporated into Section 5.2.1 of the report, along with other equations.
p. I-5.38, para 1	Clarify what the toxic endpoint is for inhalation exposure. By not including a slope factor in the inhalation equation, I am assuming the endpoint is a noncancer effect (e.g., burn, ulceration). Is this correct?.
p. I-5.44, para 2	According to HEAST, radionuclide slope factors (and hence, risk outputs) reflect combined fatal and nonfatal cancer incidences (not solely cancer fatality, as stated). Please clarify.

p. I-5.44, bottom box	It states that concentration of contaminants in five media (i.e., sediment, seep water, surface water, food products, cultural materials) are used in the exposure equations. Soil is not mentioned, yet it appears in the exposure equations in Section 5.2.1. Please clarify.
p. I-5.45, para 2	Note that for noncarcinogens, $ED=AT$, so that these terms cancel. For carcinogens, dose is averaged over lifetime ($AT=70$ yrs), so that ED does not necessarily equal AT. This comment applies to all non-radiological equations (dermal, inhalation, ingestion). Note that the dermal exposure equation is for systemic effects, not point of entry effects.
p. I-5.50, para 2	For consistency, define C_{other} and C_{seep} in Equation 5.9.
p. I-5.51, Equation 5.15	EPA recommends that when a dose is expressed as an absorbed dose (e.g., dermal absorbed dose), rather than an administered dose, then an oral cancer potency factor or an oral reference dose (which are typically expressed for an administered dose) should be adjusted by an oral absorption factor to be compatible with the absorbed dose. Although percutaneous and gastrointestinal absorption are likely not equal, Equations 5.15 through 5.20 lack this adjustment for the dermal exposure term.
p. I-5.53, para 2	Are the min, max, and mean (deterministic) values for transfer factors for each contaminant (Table 5.14) derived from the 27 river segments? Please clarify.
p. I-5.55, para 6	Distributions of internal dose conversion factors for ingestion and inhalation are specified in the text as "loguniform" but in Table 5.15 as "lognormal." Please clarify.
p. I-5.57, para 2	The method of establishing distributions for cancer potency factors and reference doses is largely arbitrary, since the shapes of these distributions are unknown due to data deficiencies. Bounding estimates provide range information but not shape information. This same comment would apply to skin absorption and skin permeability coefficient variables. These types of uncertain inputs generate unquantified uncertainty in risk outputs, making interpretation difficult.
p. I-5.57, para 6	The inhalation cancer potency factor for Cr VI is in both IRIS and HEAST.
p. I-5.58, Table 5.16	The assumption of dose equivalency across different exposure routes (e.g., oral vs. inhalation) is uncertain due to potential differences in factors such as absorption and metabolism (e.g., first-pass effect, detoxification pathways, activation, etc.). Furthermore, HEAST recommends that inhalation reference doses be expressed in terms of concentration in air (mg/m^3) rather than in terms of dose ($mg/kg/day$). The basis for this recommendation is twofold: 1) it is more accurate to base toxicity values on measured air concentrations instead of making the metabolic pharmacokinetic and/or surface area adjustments required to estimate an internal dose, and 2) there are compounds which elicit route of entry effects (e.g., sensitizers, irritants) where the toxic effect is to the respiratory system or exchange boundary where a measure of internal dose might inappropriately imply effects to other organ systems or effects from other exposure routes. Explain why loguniform distributions are assumed for the skin absorption factor

	(ABS) and skin permeability coefficient (Kp).
p. I-5.59, para 1	Should the inhalation range for ammonia be defined by a factor of 30 up (not 3, as stated), to correspond with the uncertainty factor of 30 given in IRIS? Uncertainty factors are controversial in their derivation and are in part, arbitrary, in terms of their magnitude. More generally, please explain the basis for using uncertainty factors as bounding estimates for a distribution, as well as rationale for departing from this rule.
p. I-5.59, para 5	What is the rationale for the factor of 25 used here?
p. I-5.60, para 2	The ingestion and inhalation reference doses for lead given here are based on relatively old EPA literature. More recent information, based on more subtle neurological endpoints, may suggest reducing these values, especially for sensitive subpopulations (e.g., children).
p. I-5.60, para 4	How was the maximum value (0.06 mg/kg/day) set for nickel's inhalation reference dose, since ingestion/inhalation dose equivalency is assumed and the uncertainty factor is 300 for the ingestion reference dose? Note too that some nickel compounds are carcinogenic (e.g., nickel subsulfide).
p. I-5.60, para 5	EPA's Risk Assessment Guidance for Superfund (RAGS), indicates that a reference dose is accurate to a factor of 10, not 3 as stated here.
p. I-5.61, para 3	Contrast the use of ABS and Kp in terms estimating dermal exposure to contaminants in soil and water matrices, respectively. Provide additional rationale in text for Kp values listed in Table 5.16.
p. I-5.61, para 4	Provide more rationale for the dose to risk conversion factor distribution. How were values determined in Table 5.17? Note that the averaging time specified (70 yrs) is for carcinogens. Averaging time cancels out of noncarcinogen equations, since it equals exposure duration.
p. 5.61, section 5.2.3	Somewhere in this section the overall risk for all segments and all scenarios should be given either by segment, such as in Figures 5.1-5.1, or by scenario such as in Figures 5.5-5.6. Assessment information of potential impacts for a currently applicable scenario such as the Casual Recreational Visitor Scenario would then be clearly expressed in this assessment. See comment for Appendix E.
p. I-5.62, para 1	Note that linear summation is only appropriate for chemicals with a similar mode of action. Thus, it may not be appropriate for the "toxic chemical" category.
p. I-5.62, box	According to HEAST, radionuclide slope factors (and hence, risk outputs) reflect combined fatal and nonfatal cancer incidences.
p. I-5.62, para 1	In terms of carcinogenic risk, hazard index, or radionuclide cancer risk, it can be seen that inclusion of 11 human scenarios is repetitive (Figures 5.1-5.3). The assessment could have been simplified by analyzing only two or three scenarios (e.g., a low, medium, and high exposure scenario), rather than 11 overlapping scenarios. This is exemplified by the complexity in Figure 5.4 without parallel information gain vs. the relative simplicity and information-rich graphics portrayed in Figures 5.5 and 5.6.
p. I-5.68, Figure 5.5	"Deterministic" was omitted from the figure legend in the center and bottom panels.

p. I-5.68, Figure 5.6	"Deterministic" was omitted from the figure legend in the center panel. An incorrect label was inserted here (1.47E-01).
p. I-5.71, para 1	Again, the issue of identifying sources of contamination (i.e., Hanford vs. non-Hanford) is critical to assigning liability for remedial actions but is moot to impacted human and ecological receptors.
p. I-5.73	see comments for p. xxxi paragraph 2.
p. I-5.73, para 4	The validity of using Segment 1 as background (unimpacted by Hanford operations) is unclear. For example, the text indicates that a tritium plume may impact Segment 1 groundwater. Has any aerial deposition of contaminants in Segment 1 occurred in the past from Hanford operations? Although the primary objective of the CRCIA is to evaluate risk attributed to Hanford-derived contaminants, contaminant origin is ultimately irrelevant to human and ecological risk.
p. I-5.74, Table 5.18	If these are stochastic median values, are they really "Maximum Human Health Risk" values? It seems they would be central tendency values. Please clarify.
p. I-5.79	Hexavalent chromium should be looked at separately since background contribution to the environment is negligible and its increased toxicity as compared to trivalent chromium.
p. I-5.101, para 4	Is the Mann-Whitney U test the best test to compare contaminant concentrations in Segment 1 to those in other segments? Dunnett's test or a nonparametric version (e.g., Dunn's test) may be better suited to do repeated comparison to a control (i.e., Segment 1). Also, repeated tests require a Bonferroni adjustment, so as not to inflate the overall alpha level.
I-5.88	The raw data should be looked at to see if there was a misidentification. Americium would typically be found also since Np 237 is a decay product of Am241.
p. I-5.103, para 1	When exactly is the Kruskal-Wallis test used? It does not seem that the objective is to compare a given contaminant among all segments which is what Kruskal-Wallis would do. I suppose that Kruskal-Wallis could be applied, and if significant, followed by a nonparametric multiple comparison test (e.g., nonparametric Tukey) to tease out Segment 1 differences. Please clarify. Again, a Bonferroni adjustment appears warranted.
p. I-5.108, Table 5.19	Although this table is useful, reference back to Table 5.18 is needed to see actual estimated cancer risks and hazard quotients (e.g., chromium cancer risk is 2.7E-1 from Table 5.18).
I-5.113, Table 5.20	Table 5.20 does not give units. They should be added.
I-6.1., last sentence	It is not clear what the remainder of the study is. The contaminants of concern were taken from the contaminant identification process and assessed for risk.
I-6.9, para 1	Was the determination to run the stochastic based on the deterministic results?
p. I-6.12, para 5	It is noted that, "contaminant metal tend to sorb to fine-grained sediment, which deposit in slackwater areas," such as behind Priest Rapids Dam in Segment 1. To use these fine-grained sediments as a baseline for the higher energy environment containing coarser-grained sediment found in most of the Reach is inappropriate. It

	tends to overestimate the background level of some metals.
Appendix I-E	This appendix contains some of the most valuable and clear information in the document. While the primary purpose of this assessment is to examine potential risk for Hanford related contaminants, total potential risk calculations are of significant interest. Total risk potential helps to more accurately evaluate excess risks from Hanford related contaminants as contributors to an ecosystem that already has a significant background contaminant load. I highly recommend Figures E.1-E.18, or risk range figures for each segment (like Figures 5.1-5.3), be pulled up into section 5.2 of the report. One gets the impression that this pertinent information is tucked away rather than prominently displayed.
Appendix II-D, Fig. II-D.1	This diagram attributes funding to the box containing the NRTC, Tri-Party, and Sponsoring Organization. Are all three required to fund this project? The authors should revisit this diagram and either delete the funding reference or be more specific.
p. I-7.7 Section 3.0 Data	Reference to Miley's work listed below is noticeably absent. It (along with its associated disks) are the primary source for raw data used in this document. Please include: Miley, T. B., T. K. O'Neil, R. O. Gilbert, L. A. Klevgard, T. B. Walters. 1997. <i>Data for the Screening Assessment Volume II: Appendices</i> . DOE/RL-96-16-c, Rev I, Vol. II. Pacific Northwest National Laboratory, Richland Washington.

Selected References:

Blanton, M. L., W. W. Gardiner, and R. L. Dirkes. 1995. *Environmental Monitoring of Columbia River Sediments: Grain-Size Distribution and Contaminant Association*. PNL- 10535, Pacific Northwest Laboratory, Richland, Washington.

Dunks, K. L., 1995. *100 Area River Effluent Pipelines Characterization Report*. BHI-00538, rev. 00, Bechtel Hanford, Inc., Richland, Washington.

Miley, T. B., T. K. O'Neil, R. O. Gilbert, L. A. Klevgard, T. B. Walters. 1997. *Data for the Screening Assessment Volume II: Appendices*. DOE/RL-96-16-c, Rev I, Vol. II. Pacific Northwest National Laboratory, Richland Washington.

Thornton, E. C. 1995. *Speciation and Transport Characteristics of Chromium in the 100D/H Areas of the Hanford Site*. WHC-SD-EN-TI-302