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STATE OF WASHINGTON

DEPARTMENT OF ECOLOGY

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May 7, 1996

Mr. Robert K. Stewart
CRCIA Project Manager
U. S. Department of Energy
P.O. Box 550, MSIN: HO-12
Richland, WA 99352

Dear Mr. Stewart:

Re: "Species for the Screening Assessment: CRCIA," Draft

Enclosed are comments provided by the Washington State Department of Ecology on the "Species for the Screening Assessment: CRCIA," DOE/RL-96-16-b Rev. 0 UC-630 Draft. The comments are provided in the requested review comment form, and are being sent separately to you, and your contractor staff, in electronic format. 43304

Thank you for your willingness to work with the Columbia River Impact Assessment Team in developing and reviewing these documents. If you have any questions regarding these comments, please contact me at (509) 736-3027.

Sincerely,

David Holland
Nuclear Waste Program

DH:skr
Enclosure

cc: Larry Gadbois, EPA
Stuart Harris, CTUIR
Paul Danielson, Nez Perce Tribe
Dan Landeen, Nez Perce Tribe
Lino Niccoli, YIN
Tom Woods, YIN
Ralph Patt; ODOE
Amoret Bunn, Dames and Moore
Administrative Record:
DOE/RL-96-16-b Rev. 0 UC-630 Draft

Review Comments on Technical Reports for the Columbia River Comprehensive Impact Assessment

Compiled by: Damon Delistraty, David Holland, and Wayne Soper

Report Title: *Species for the Screening Assessment* (Becker et al, March 1996)

Page, Paragraph	Comment	Resolutions
vi, para 1	The Tri-Parties should be recognized as members of the CRCIA Team.	
vi-vii	This entire section needs to be rewritten. See comments given previously on this section in the *Human Scenarios* report.	
ix, para 2	Does this assessment concern itself only with specie populations, or will it address threats to individuals within some species?	
ix, para 3	Consider listing the six criteria here.	
ix, para 4	Briefly explain rationale for excluding species "within the same foraging guild that have the largest body weight" in the process of Tier II species selection. See comment for page 3.16, para 3.	
x, table	Use of the asterisk in the fourth column of the table is inappropriate, due to the definition of the asterisk in the table footnote.	
xiii	The definition for biomagnifying should be amended to read " . . . . 'primarily' through dietary accumulation," since biomagnification includes processes of both bioconcentration and bioaccumulation.	
xiv	The definition for foraging guild might be expanded to include " . . . . similar composition and a similar response to environmental impacts (e.g., exposure to chemicals) as a result of similarities in food habits."	
xiv	The definition for hazardous chemicals indicates this term is generally used to differentiate from carcinogenic chemicals. This is arguably not true and serves only to confuse terminology (e.g., "hazardous waste" may include carcinogens).	

xv	The definition for sensitivity analysis should be amended to state, it is a method to examine the variation in model output resulting from systematic changes to individual model inputs. Sensitivity analysis is most often defined as one component of uncertainty analysis. Another component of uncertainty analysis is probability analysis (e.g., Monte Carlo simulation).	
xv	The definition of toxicological benchmark (i.e., measurement endpoint) should be expanded to state, the benchmark represents an arithmetic or statistical summary of the observations which comprise the measurement (e.g., NOEC, LC50).	
xv	The definition for uncertainty should state, uncertainty is a lack of precise knowledge to what the truth is, whether qualitative or quantitative. This should be distinguished from variability. Variability, in turn, should be defined separately as a measure of heterogeneity or data dispersion. Variability describes the scatter of measurements around the center of a distribution (e.g., range, variance). It is recognized in practice, however, and it is often difficult to treat uncertainty and variability separately.	
1.1, para 3	The final sentence should read, " . . . 2) hazards posed by past, present, and future contaminant fluxes, . . . "	
1.2, figure	The figure is missing the microbial community.	
1.3, figure	The figure is missing the microbial community.	
2.1, para 1	Since this section is entitled, "Ecosystem," the abiotic component of the ecosystem should be briefly described, in addition to biological resources.	
2.1-2.3	No mention is made of the microbial community within either the riparian or aquatic communities. These organisms (i.e., bacteria, protozoa, fungi) perform important processes (e.g., carbon mineralization, redox, nitrification, denitrification, sulfide precipitation, methylation, dealkylation), which influence the mobility and toxicity of many substances. These microbes are suspended in surface waters, either freely or sorbed, to suspended particles and on soil or sediment particles. Although fungi (as a broad taxon) has been included as a	

	final Tier II receptor species, other important microbes have not been considered.	
3.1, para 2	What are the six criteria? Consider listing them here.	
3.4, para 1	Regarding the fifth criterion (i.e., availability of toxicological data), note lack of data does not necessarily equate with lack of ecological significance. It should be explicitly acknowledged some species may lack toxicological data but are ecologically significant.	
3.4, para 2	Clarify the 88th percentile as a threshold. Was this the percentile of the cumulative frequency distribution which was empirically observed to delineate species with three or more yes responses?	
3.4, para 4	State rationale for excluding species with the largest body weight within the same foraging guild. See comment for page 3.16, para 3.	
3.6, para 2	It appears media scores were scaled from 0 to 4 (e.g., Table 3.8), not 1 to 4. Clarify.	
3.6, para 2	Discussion of the sensitivity score might be expanded to include what the organism does to the contaminant (i.e., chemical disposition). Mention the influence of absorption, distribution, biotransformation, and excretion on potential toxicity. In addition, sensitivity may relate to a unique mode of toxic action (e.g., eggshell thinning), or a critical life stage.	
3.6, para 2	LD50 is only an appropriate endpoint for ranking sensitivity to contaminants on an acute basis. An endpoint, such as a no observed effect level (NOEL), would more appropriately measure chronic toxic effects.	
3.7, table	Dermal and ingestion exposure are awkward terms for primary producers. Explain what is meant by ingestion exposure for macrophytes (uptake by roots?). May be clarified in a table footnote.	
3.8, table	Same comment for page 3.7.	
3.9, table	Same comment for page 3.7.	
3.10, table	Biomagnification, defined as magnification primarily through dietary accumulation, does not really apply to primary producers.	

	Table 3.8 should be disagreggated by sediment/soil and pore water/groundwater to correspond more directly with the examples given.	
3.12, para 1	Same comment for table 3.10 and for table 3.8 on page 3.11.	
3.13, para 3	Prey are not media, why are they included here?	
3.14, para 2	Document the statement, most of the contaminants are radionuclides.	
3.14, para 2	Use of the LD50, as an endpoint for ranking sensitivity, may not be the best measure (see LD50 comment for page 3.6.). Were LD50 values utlilized derived from the same route of exposure with similar exposure duration? For example, comparability of oral LD50s (e.g., mammals) with aquatic immersion LC50s (e.g., fish) is invalid.	
3.14, table 3.15	According to HSRAM (table 4-3), fish and reptiles are more sensitive than birds to ionizing radiation. This conflicts with table 3.15 scores.	
3.15	The grand average exposure scores (row 25, Appendix C) should have been divided by 10.25 (rather than 10) if the intent was to keep 4.0 the maximum score (i.e., 41/10.25=4.0). Please clarify. Doing this assigns equal weight to grand average exposure (row 25), exposure duration (row 28), and sensitivity (row 29) scores. Is this the intent?	
3.16, para 2	Regarding the preference of grand average exposure scores (row 25, Appendix C) over composite effect scores (row 31), are you saying it is inappropriate to mix (composite) acute toxicity data with chronic exposure data? If so, please clarify.	
3.16, para 3	The influence of body size on toxicity is variable, and not easily predicted, due to the interplay among metabolism, detoxification, and surface area to volume ratio. Much of what has been attributed to sensitivity differences may be, in fact, artifacts of the bioassay process caused by size differences affecting the kinetics of accumulation (Rand et al, 1995). For example, when exposed to a constant lethal water concentration of a chemical, a small fish usually dies much sooner than a	

	large fish because the small fish reaches a lethal body residue sooner. (Opresko et al, 1995) indicates smaller animals have higher metabolic rates and usually are more resistant to toxic chemicals because of more rapid rates of detoxification. However, this may not be true, if the toxic effects of the compound are produced primarily by a metabolite. It has been stated, birds and mammals may be more susceptible than their larger counterparts to the effects of organophosphate pesticides because their high metabolic rates require them to ingest large quantities of contaminated food per unit body weight, or make them less tolerant to pesticide-induced anorexia (Grue, 1994). Excluding species with larger relative body weight within the same foraging guild and focusing on smaller species appears inappropriate, as a general rule.	
3.17	Same comment for page x.	
4.1, para 1	Give more detail on how exposure models will integrate exposure over all pathways and media to yield an overall total dose. It is stated, exposure estimates will be compared to toxicological benchmarks which reflect mortality (e.g., LD50), or the lowest observed adverse effect level (LOAEL). Benchmarks based on acute mortality should be given the lowest preference in comparison to benchmarks based on more sensitive sublethal and chronic effects (e.g., NOECs for growth, reproduction). It might be explained, endpoints for ecological risk assessments should ideally be at the population or community level (e.g., species shifts), instead of at the individual organism level (e.g., mortality). An exception to this would be when the assessment involves a threatened or endangered species where value has been assigned to the individual species.	
4.1, para 3	Elaborate on the deterministic and stochastic models. For the deterministic approach, give rationale for using the maximum source term data. Will point estimates for other exposure variables be at a maximum? For the stochastic model, will the analysis be adjusted for internal correlations among input variables? How will a	

	probability distribution be constructed for toxicological benchmark data? In reference to the mention of "high ratio of exposure to benchmark," introduce the term "environmental hazard quotient" (EHQ), as defined in HSRAM. Are you saying the initial EHQ will be calculated with a deterministic approach? Are you using the deterministic model as a screen for the stochastic model? Is this appropriate? Please clarify this.	
C.2	The composite rank (row 32) is incorrect for amphibians. It should read "1,2,2," not "1,1,1." I would suggest careful review of all table calculations and data.	