Review Comments:
Human Health Risk Assessment, GE/Housatonic River Site, Rest of River

Reviewer: Lee Shull PhD, MWH Global, Sacramento, CA. December 17, 2003

A. Phase 1 – Direct Contact Screening Risk Assessment (Volume IIA, Appendix A)

Charge Questions and Responses to these Questions:

Were the procedures used in Phase 1 of the HHRA to screen out properties and areas from further evaluation as well as the application of those procedures appropriate under the evaluation criteria?

Overall review conclusion: For screening purposes and as related to the specific, stated objectives of the Phase 1 assessment, the general procedures used are acceptable and appropriate. Also, for the most part, EPA's risk characterization criteria of transparency, clarity, consistency and reasonableness, and the additional "objectivity" criterion are met. A few items lacking some of these areas are noted under specific comments below.

In addressing this question, consider:

1. **The general procedures used:** Following are a few of the more key concerns and issues:

   - To the extent that the Consent Decree dictates procedures, methods, etc as related to the Phase 1 should be identified and briefly summarized in Section 2 to improve transparency. More than simply a reference to the Consent Decree (Appendix J) is needed.

   - Rationale for why the Phase 1 assessment did not utilize, or even make mention of, EPA's "Soil Screening Guidance for Developing Soil Screening Levels for Superfund Sites" should be presented. Also, a reference for this guidance is not included in the reference list (Section 9). This guidance is directly applicable to the process undertaken in this Phase 1 screening analysis. Without some justification for not following this guidance, lack of consistency with EPA guidance is an issue. Section 1.2 would be the appropriate place for this discussion.

   - Clarification is needed as to why only PCBs were addressed in the Phase 1 and not other chemicals detected in samples in the area. While I do not necessarily disagree with the approach, it is nonetheless inconsistent with EPA guidance. A better explanation as to why the deviation from standard screening practice is needed.

   - Further clarification is needed as to why only EPA/USACE-collected data, and not all data, are used in the Phase 1 screening evaluation. Also, the chemical form of PCB data used in this screening evaluation is unclear as to whether it is total PCBs (tPCBs), summed Aroclors, or something else.

   - Page 4-51, Section 4.5.3.9: It is unclear why no future agriculture land use was evaluated; only current agriculture land use. If it is assumed future land use of floodplain areas are not believed to be transformed into agriculture areas, rationale/justification for

this assumption should be provided here, or a reference to a location in the report where such information can be found should be provided.

2. **The SRBCs used for the COPCs:** The SRBCs used generally meet the evaluation criteria. Following are key concerns:

   - Page 2-6, lines 1-2: The application of the atypical target risk levels (e.g., 5×10^{-6} for residential and recreational; 1.6×10^{-6} for utility worker; 1.1×10^{-6} for groundskeeper) is not consistent with EPA guidance, and the explanation given is not transparent. Typically, selection of a target risk level (TRL) is either a site-specific risk management decision, or is codified in guidance. In this Phase 1 assessment, it appears these TRLs are risk management decisions, which is acceptable. A more thorough risk management explanation is needed.

   - In Section 2, in particular the tables, none of the exposure parameters and assumptions are referenced and should be. Parameters that are based on a source that can be referenced should be referenced. For those that are based on professional judgment, rationale should be given that the parameter or assumption meets EPA's criteria of transparency, clarity, reasonableness and consistency.

   - The screening of agricultural areas based on direct exposure to PCBs (soil ingestion and direct contact), which does not include food chain transfer exposure, could result in elimination of some parcels for consideration in Phase 2. The document should present clear rationale for not developing agricultural SRBCs, or should provide clear and transparent explanation as to why it is believed that the SRBCs will not result in elimination of parcels that should be included in the Phase 2 analysis.

   - Page 2-6, Section 2.5.1: Exposure frequencies for residential and recreational receptors assumes 5 d/wk for 7 months for high contact and 3 d/wk for 7 months for low contact. Additional rationale for these values should be provided. These values could be considered low, especially for children who are likely to play outdoors during good-weather months as much as 7 d/wk.

   - Page 2-6, lines 21-23: Text should indicate here that age-adjusted SRBCs are developed, as indicated in Tables 2-2 and 2-3.

   - Page 2-8, line 12: RAGS Part E (EPA, 2001) suggests 2,800 cm^2/d for a childs surface area rather than 2900 cm^2/d.

   - Tables 2-1 to 2-6: All values for individual parameters presented in these tables should be referenced. A column can be easily added to each table in which the appropriate reference can be cited.

   - Page 2-16, line 19: RAGS Part E (EPA, 2001) suggests a utility worker soil adherence factor of 0.9 mg/cm^2 instead of 0.8 mg/cm^2.

   - Page 2-19 and Table 2-6. Soil ingestion of 50 mg/d for the groundskeeper seems low. Suggest using at least 100 mg/d for outdoor worker (EPA, 2002 – Supplemental Soil Screening Guidance for Developing Soil Screening Levels for Superfund Sites).

3. **The land use and exposure categories considered and the classification of particular parcels and areas into those categories:** The land use and exposure categories considered and the classification of particular parcels and areas into those categories generally meets the evaluation criteria. Following are key concerns:

   - Although the stated focus of the Phase 1 screening is current land use (*e.g.*, page ES-9, line 10), it is not clear that all of the exposure scenarios assessed are, in fact, representative of current land uses vs future land uses. Further clarification is needed as to how future land use was factored into this screening analysis.

   - Page 2-23, line 8: The basis of these land use designations should be briefly discussed here, or reference made to a place in the document where such an explanation can be found.

Additional reviewer comments:

- The Executive Summary generally provides a very good overview of the screening process, including the purpose, the methods and assumptions, and the results.

- Page ES-2, lines 26-29: Because "integrated SRBCs" play such an important role in this screening risk assessment, the executive summary should better define and provide a bit more basic information on how these "integrated" SRBCs are derived, and explain why "integration" is relevant and appropriate.

- Page ES-8, lines 21-25: This sentence is unclear. Is the statement being made that no floodplain or riverbank soil samples were collected because there is no evidence of upstream contamination?

- Page ES-8, line 23. The statement refers to the "known relationship between sediment concentrations and associated floodplain concentrations", but does not explain what this relationship is or what downstream sediment concentrations mean relative to downstream floodplain soil concentrations. Clarification is needed.

- Page 1-2, Section 1.2 and elsewhere, as needed: Further explanation of differences in soils (e.g., floodplain vs riverbank) is needed. The document appears to treat these two floodplain and riverbank soils the same with the only distinction being high vs low contact. Why use floodplain and riverbank distinctions if high contact and low contact are the only distinctions that matter for the screening? It also appears that the entire 10-year floodplain was not included in this screening analysis (Ex. Fig 3-7 of Vol IIB).

- Page 2-2, lines 2-3: Further clarification is needed here as to why only EPA/USACE-collected data, and not all data, are used in the Phase 1 screening evaluation. Also, the chemical form of PCB data used in this screening evaluation is unclear; is it total PCBs (tPCBs), summed Aroclors, etc?

- Page 2-3, lines 8-24: Should indicate what summary statistics or 95% UCLs are chosen when the data fit neither a normal nor lognormal distribution.

- Page 2-6, line 18: EPA's assumption discussed here should carry a reference.

B. Phase 2 – Direct Contact Risk Assessment (Volume IIIA, Appendix B)

Charge Questions and Responses to these Questions:

1. Were the following aspects of the direct-contact exposure assessment appropriate under the evaluation criteria?

The exposure scenarios which were evaluated.

- In general, the exposure scenarios evaluated are appropriate.
- Page 4-3, Section 4.2.3: It is not clear why a construction worker scenario was not considered or discussed in the CSM. I believe a construction worker should be included in the analysis, at least evaluated (and dismissed) in the CSM, which is usual practice. A construction worker who is not a building construction worker (e.g., someone who constructs buildings, which would not be constructed in the floodplain), but rather an earth worker (e.g., earth moving equipment operator) such as a road construction worker should at least be identified in the CSM, and then dismissed, if appropriate, with supportive rationale.

The exposed populations which were selected for each scenario.

- In general and except for the following comments, the exposed populations evaluated for each scenario are appropriate.
- In the receptor matrix (Table 4-1) and in Section 4.2.3, three receptor groups are identified; the younger child, older child, and adult. The text in Section 4.3.5.1 (lines 13-15) then states that dose and risk estimates were generated for two groups; children (0-6) and adults (7-45). This difference needs to be clarified. Furthermore, an explanation should be provided why older children are lumped in with adults for the residential scenario and not for the recreational scenarios.
- Page 4-10, Section 4.3.5.2.1, Table 4-1: Regarding the ATV/dirt and mountain bike riding, it is not clear why an adult receptor was not considered for this scenario. Adults over the age of 30 routinely engage in such activities on a regular basis. Lines 15-17 implies that the adult is addressed, which is not the case for the ATV/dirt and mountain bike rider.

The exposure areas identified based upon potential current and future use(s).

- The exposure areas identified for potential current and future use are appropriate.

The routes of exposure for each scenario.

- The routes of exposure for each exposure scenario are appropriate.

2. Have the most important exposure pathways been identified and evaluated?

- In general, the most important exposure pathways have been identified and evaluated.
- The breast milk and transplacental pathways should be considered for inclusion in the residential population analysis. As was expressed at the public meeting November 18-20, potential health impacts associated with neonatal exposure to PCBs and dioxins/furans is

among the greatest concerns of residents and medical personnel in the area. Risk assessment methods are available for such an analysis; EPA's *"Methodology for Assessing Health Risks Associated with Multiple Exposure Pathways to Combustor Emissions" (2000?).*

3. Were the approaches and methods used to calculate and apply exposure point concentrations (EPCs) for the direct-contact exposure assessment appropriate under the evaluation criteria?

- In general and except for the following comments, EPC calculation methods are appropriate. Use of the 95% UCL or maximum whichever is lower is consistent with guidance and standard risk assessment practice.
- Page 4-15, lines 5-9: Please expand the explanation for how these "test runs" were done, or refer to a relevant section of the report(s) where the reader can find the information.
- Page 4-18, lines 24-page 4-19, line 6. Whereas all measured and interpolated data were used in the 95 UCL calculation, the value of "n" was restricted to measured data only. Please provide additional rationale or relevant citation(s) justifying/substantiating this approach to improve transparency.
- Page 4-19, line 7: Suggest providing a brief technical description of ProUCL in an attachment. The purpose is to inform readers who may be unfamiliar with this program confidence that it is a "black box", but that it is appropriate for performing statistical analyses. If there is EPA precedent for its use, this should be stated.
- Page 4-21, lines 9-13. Suggest expanding the discussion as to why the approach for deriving EPCs in Reaches 5 and 6 was not applied in Reach 7. It is not clear whether by not applying IDW to fill data gaps results in EPCs with greater uncertainty, meaning risk estimates will have greater uncertainty.
- Page 4-22, lines14-16: Should again briefly describe the 8 areas, or, ideally reference a figure that shows the 8 areas.
- Note comments on Attachments 1, 2, 3 and 4 below, all of which relate to the derivation of EPC estimates in this HHRA.

4. Were the values used to represent the exposure and absorption parameters used in the direct-contact exposure assessment appropriate under the evaluation criteria, specifically:

Exposure duration scenario.

- In general and except for the following comments, the ED values used seem appropriate.
- In Section 4.2.3, the age ranges for each of the identified exposed populations are given (e.g., older child 7-18 years of age). The sources (references) of these age ranges should be given and a definitive statement should be made that the ranges are reasonable for this risk assessment.

- Page 4-8, lines 12-15: Exposure duration for adults is defined as 7-45 years. This differs from EPA's default exposure duration (30 years). Rationale or a reference citation for this value should be provided.
- Page 4-48, lines 8-9 and Table 4-17: Seems like the CTE ED should be 19 years instead of 25 years, since a <12 yr old is not allowed to hunt (i.e., 31-12 = 19 yrs).

Exposure frequency and area use factors for each scenario and exposure area.

- In general and except for the following comments, the exposure frequency and area use factors seem appropriate.
- Section 4.4.1.1.1: Regarding area use weighting, the factors selected seem appropriate. However, given the overall impact of EF on the risk estimates, this section deserves more consideration in my opinion. GE presented information at the November 18-20, 2003 public meeting that described/documented the inaccessibility of some areas in Reaches 5 and 6. I strongly suggest this information be taken into consideration and that re-evaluation of the area use factors be done. As a minimum, the GE information should be presented in the uncertainty section with some level of judgment about the impact of the values used on the risk estimates.
- Page 4-24, Section 4.5.1.1: Age adjustment of body weight is common practice in risk assessment. Rationale should be presented here as to why this practice was not done.
- Page 4-32, Section 4.5.3.1.1: The EF for residential sites is typically 350 days/yr. The difference in the basis for the MDEP-referenced value of 150 days/yr and the 350 days/yr default (1998 Soil Screening Guidance [SSG] and 2002 SSG Supplement) should be provided.
- Page 4-33, line 2: Please state whether MADPH, 1997 is a peer-reviewed study. If it is not, stronger statement should be added stating why the risk assessors believe can be relied upon in this HHRA.
- Page 4-35, line 11: Better rationale for the two EF values should be provided. If the basis was professional judgment, a statement of same should be included. For example, later in the report (page 4-38, lines 12-13), a clear statement is made regarding the EF basis. This approach should be applied throughout Section 4.
- Page 4-42, lines 1-3: How can the mean be 18, and the 50^{th} percentile 2, and the 75^{th} percentile 7? Please clarify.
- Page 4-49, Section 4.5.3.8.1: Would it be possible to provide some additional rationale for these EF values (i.e., some minimal information to support the professional judgment).
- Page 4-51, Section 4.5.3.9.1: The EF for the farmer receptor of 10 days per year seems low. Many other management practices such as irrigation, tilling, side-dressing/fertilization, inspection, etc. occur for which direct soil contact occurs in addition to harvesting and planting.

- Page 4-54, Section 4.5.3.11.1: The utility worker EF of 5 days/yr seems low. Is the assumption of 5 days/yr for a single area, or all areas where PCB contamination exists?

Soil ingestion rates.

- In general and except for the following comments, soil ingestion rates are appropriate.
- Page 4-33, Section 4.5.3.1.3: Rationale for applying 0.5 of the 100 mg/day as the CTE ingestion rate should be provided.
- Page 4-55, Section 4.5.3.11.3: It doesn't seem accurate to assume an RME ingestion rate of 330 mg/day soil ingestion for a utility worker compared to 200 mg/day for a farmer. Either the utility worker is too high or the farmer is too low.

Exposure assumptions affecting dermal contact (e.g., soil adherence rates, skin surface areas assumed to contact soil or sediment).

- In general and except for the following few items, the exposure assumptions related to dermal contact are appropriate.
- There appears to be some inconsistency in the selection of some exposure parameters across similar, if not identical, exposure scenarios. For example, the skin surface areas for the marathon canoeist and recreational boater are different, but no rationale is given for the difference. Consistency across similar exposure scenarios would be preferred, or additional discussion to provide justification for the difference would meet the evaluation criteria.
- Page 4-25, line 29: Reference is made to two concerns by the EPA of the GE dermal absorption study in rhesus monkeys. Clarification is needed as to whether these "concerns" are reflected in a formal peer review conducted by either EPA or GE, or whether they are concerns of the authors of this risk assessment.
- Page 4-31, Section 4.5.2.4.2: Additional discussion of the available adherence factors (AFs) would improve clarity, specifically a discussion of rationale as to why the AFs selected are believed representative of Housatonic River soil and sediment.
- Page 4-48, line 25. Use of reed gatherer AFs for soil exposures seems overly conservative. A "moist soil" value, if available, would be more appropriate.

Oral and dermal absorption factors.

- The dermal absorption factors are appropriate for this risk assessment.

5. Is the approach used to estimate a Reasonable Maximum Exposure (RME) and a Central Tendency Exposure (CTE) for the direct-contact exposure assessment appropriate under the evaluation criteria?

- The approach used to estimate RME and CTE are considered appropriate.

6. Were the uncertainties adequately characterized and expressed?

- The qualitative uncertainty analysis presented is incomplete and inadequate. Not all factors that could impact risk estimates, either over- or under-estimates, are identified and evaluated. For many of the sources, the document states "risk may be either over- or under-estimated." In my view, the risk assessor should, if at all possible, use his/her judgment, experience, etc to put forward a position whether risks are either under- or over-estimated. If risks could go either way, reasons for same should be provided.

- In several places, confusing terminology is used. For example, on page 7-2, lines 22-24, the following statement is made: "Therefore, exposure to surface water was eliminated from further consideration and quantification, which would lead to an insignificant underestimate of risk." Does this mean there may be an underestimate of risk, but it is considered insignificant? In this case, since the conclusion was made based on scientific evidence in the exposure assessment that no PCB concentrations exist in water, this means there is no exposure. If the assessors believes scenarios exist whereby exposure and some level of risk associated with water could occur, these scenarios should, as a minimum, be identified and discussed qualitatively.

- Page 7-1, line 16: Suggest adding "…overview of sources of uncertainties…"

- Page 7-3, lines 20-23: Suggest stating mean concentrations are likely to be overestimated.

- Page 7-4, line 19: Should comment whether current land uses are likely to over- and/or under-estimate risks.

- Page 7-7, line 7: Suggest adding the words "….risk compared to the central tendency CSF."

- Page 7-7, Section 7.2.3.1: This section is sorely lacking. One of the greatest sources of uncertainty in human health risk assessment is in the areas of animal-to-human extrapolation, and high-to-low dose extrapolation. This section contains no mention of these factors, as well as no mention of human (e.g., epidemiology) information that helps understand the degree to which strict reliance on animal data impacts risk estimates. I strongly suggest the addition of this information to this section, with an assessment by the risk assessor as to whether these sources of uncertainty present an over- , no impact, and/or under-estimation of the risk estimates presented in these documents.

- Page 7-8, lines 6-7: It is my understanding that the TEFs are based primarily on CYP1A1 induction, which is a relative indicator of Ah binding in the liver. Please clarify.

- Page 7-8, section 7.2.3.3: This entire section should be eliminated from the document. In particular, I do not believe it is not appropriate to discuss EPA's proposed draft TCDD cancer slope factor (CSF) of 1E+06 $(mg/kg\text{-}d)^{-1}$. The EPA's Science Advisory Board (SAB) has provided comments to EPA on this issue, yet the EPA has not yet released a revised document for public or peer review. The derivation of this CSF is a highly controversial issue due to EPA's failure to clearly describe and document its derivation, and because it appears to be based largely on epidemiological studies for which there are substantial confounding factors and spurious associations. Furthermore, the uncertainty discussion specifically fails to make reference to other alternative CSFs for TCDD published in the peer-reviewed literature and by other federal regulatory agencies,

specifically those published by the FDA (1992) and Keenan et al. (1991). In the absence of a final consensus in the scientific and regulatory communities on the potential human carcinogenicity of TCDD, it is speculative and presumptuous to even discuss EPA's draft CSF for TCDD in this risk assessment. Should EPA decide to keep this discussion in the uncertainty section, I recommend that the full range of possible TCDD CSFs published since the Pathology Working Group reported its findings of the 1990 re-evaluation of the Kociba et al. (1978) rat liver pathology slides (Sauer 1990; Keenan et al. 1991; Goodman and Sauer 1992) be included also.

- A high quality HHRA should present relevant information that helps the risk manager to place the estimated risks in proper perspective. Information provided in GE's presentation at the November 18-20, 2003 public meeting could be used to accomplish this objective. The findings in two studies described at this meeting – the MDPH Exposure Assessment Study, 1997 and the ATSDR/MDPH Cancer Incidence Study, 2002 – provides excellent information to which the risk estimates can be compared. Without placing risk estimates in proper perspective, including identifying data gaps where they exist, can be result in misleading risk managers during the decision making process.

7. Overall, was the approach used to estimate risk from direct contact reasonable for evaluating the baseline risk?

- In general, the assessment does provide the information in the four areas stated on page ES-1. Although the Phase 2 report lacks a clear statement of objectives, if these four items are taken as "objectives", I believe the assessment generally meets the overall purpose of providing this information, in particular the first item: " a characterization of the potential human health risks under baseline conditions (i.e., no action) for current and future uses." Also, for the most part, EPA's risk characterization criteria of transparency, clarity, consistency and reasonableness, and the additional "objectivity" criterion are met, with the exception of the uncertainty analysis (Section 7). In my opinion, the greatest deficiencies are in the area of lack of transparency and clarity. Other key deficiencies exist in some parts of the assessment that could result in significant over-estimations of risk. As a minimum, the assessment should address these deficiencies, especially the uncertainty analysis, to ensure the results of the risk assessment are appropriate and are placed in proper perspective for risk management decision making.

- Because of the central role played by the Consent Decree in the performance of this HHRA, a similar comment as was made on the Phase 1 screening assessment is relevant to the Phase 2 assessment. A brief summary as to how the Consent Decree dictates procedures, methods, etc as related specifically to the Phase 2 Direct Contact assessment should be identified and briefly summarized in Section 2. This addition would improve transparency. Again, this summary should consist of more than simply a reference to the Consent Decree (Appendix J).

Additional Reviewer Comments

- Page ES-1, line 18-19 (also Page 1-1, lines 23-25): The 1 ppm isopleth and its equivalency to the 10-year floodplain is such an important component of this risk assessment that it warrants more discussion, even in the Executive Summary.

Alternatively, a reference to a section(s) of the report where the underlying rationale and essential information for this approach is given should be provided.

- Page ES-5, lines 5-6: The role of the two agencies (EPA and MDEP) should be either elaborated here, or a reference to a description provided in a place elsewhere in the documents should be cited here.
- Page ES-5, line 21: An explanation of the PSA should be included, or a reference to a definition provided elsewhere in the documents should be cited here.
- Page ES-6, line 8: Given the significance of SRBCs in this risk assessment, it is important to add some additional description of their basis in the Executive Summary (e.g., reference to toxicity criteria used, citing key references, methods used in derivation).
- Page ES-9, lines 1-3: I believe a construction worker should be included in the analysis. This is not a building construction worker (e.g., someone who constructs buildings, which would not be constructed in the floodplain), but rather an earth worker such as a road construction worker. I see nothing in the HHRA that indicates such work would not be done. This is different than a utility worker who may be involved in short-term activity (e.g., trenching). If EPA believes an earth work type of construction worker is not significantly different than a utility worker in regards to the exposure assumptions, then documentation/rationale should be provided.
- Page ES-20, line 9: Additional information should be provided here on the rationale for 10% of all soil and sediment samples to be analyzed for PCB congeners, dioxins, and furans. Because of its significance to the risk assessment, rather than reference the SIWP, the rationale should be provided/described in sufficient detail here.
- Page 1-2, lines 7-8: The issue of cumulative risk (e.g., summing risks for multiple exposure pathways) should be addressed here. I know EPA has a good explanation for not developing cumulative risk scenarios, and this would be a good place in the Phase 2 Direct Contact HHRA to provide this rationale.
- Page 1-3, lines 16-19: It would be helpful to readers to better understand land use trends in the flood plain area (i.e., what land uses are diminishing and disappearing and what land uses seem to be increasing or emerging). A better profile of what the land uses in the area are likely to be over the next 30-50 years would be helpful.
- Page 1-5, lines 12-20: It would be helpful to readers to explain here the rationale for not applying the Phase 1 screening, as is the usual case, to COPC selection instead of using it only to narrow the study area to be assessed in Phase 2.
- Page 1-5, lines 24-26: Rationale for including pesticides as COPCs in the fish and waterfowl consumption risk assessment and not in other assessments should be provided here. This explanation should include whether the assessors do or do not consider the GE facility as a source of these pesticides.
- Page 2-2, line 15: Suggest adding a brief statement of rationale for how the analyte list was originally selected should be given here.

- Page 2-2, lines 25-27: Suggest adding to the end of this sentence the words "…and to do a quality human health risk assessment."

- Page 2-3, Section 2.3: This section should be expanded and more explanation of DQO provided, or, alternatively, reference made to another section of one of the other reports where the information is given. In particular, a discussion of DQO as related to human health risk assessment and confirmation that the data soil and sediment data were evaluated in accordance with EPA's DQO requirements for risk assessment (EPA, 1992).

- Page 2-3, line 34-page 2-4, lines 1-2: As stated above, this discussion should be expanded, or a document referenced that presents the methods and results of the DQO evaluation in which EPA data are documented as having met all DQOs pursuant to EPA DQO guidance.

- Page 2-4, line 8: Should begin this section by explaining briefly what is meant by "data reduction" and why it is done as a standard practice in risk assessment.

- Page 2-5, lines 10-12. This sentence implies that other chemicals were screened out (eliminated) in the Phase 1, which is not the case. Please clarify that only "areas" were screened in the Phase 1.

- Page 2-5, line 20: What constitutes the PSA should be defined, or reference made to a section of the report that does define the PSA.

- Page 2-5, lines 27-29: The discussion presented here on how background concentrations were addressed seems inconsistent with Section 2.5.2.2. Also, it would be helpful perhaps if some discussion about why EPA's 2002a guidance on dealing with background concentrations is not applied in this HHRA.

- Page 2-8, line 7: This sentence is confusing. What is meant by "the initial step"? Also, does "evaluated" mean comparison to background concentrations? Please clarify.

- Page 2-8, line 21-23: Rationale for 0-1 ft depth for background samples should be provided. Also, are residential PRGs as shown in Table 2-5 used consistently for background comparisons?

- Page 2-9, lines10-13: Rationale for not applying standard statistical methods in determining whether concentrations are or are not significantly different from background should be presented. Also, the basis for applying a ratio of 5 for determining whether site and background concentrations differ should be given.

- Page 2-9, Section 2.5.2.2.2: Should provide some explanation on where the MDEP samples were collected, so that the reader has some basis for evaluating relevancy of these data.

- Page 2-10, lines 18-19: The statement that PAHs above background are not site related because they are not widespread in distribution should be eliminated unless better supportive rationale/information can be provided. For example, if there is no historical information related to GE operations that would suggest GE as a PAH source, such additional statements should be added here.

- Page 2-10, Section 2.5.3 and Table 2-8: In keeping with the development of SRBCs for other purposes in this risk assessment, rationale as to why SRBCs for evaluating sediments could not have been developed also. Lack of sediment PRGs is not an acceptable basis for not developing SRBCs (as was done in the Phase 1 assessment), unless solid scientific reasons can be given. If EPA chooses to retain the soil PRGs as screening criteria for sediments, then more justification should be given regarding the appropriateness of using soil PRGs for this purpose.

- Page 3-4, lines 7-8: The basis for the statement that the 1996 PCB cancer re-assessment "remains consistent with the 1999 Revised Carcinogen Guidelines" should be provided. If EPA or some other body has issued a written statement that this is true, an appropriate reference should be provided. Also, EPA (2003b) should be referenced on line 8.

- Page 3-4, lines 8-9: Reference to EPA (2003b) should be included.

- Page 4-8, line 20: Please clarify what is meant by "reasonable potential" (page 4-8, line 20) versus "realistic potential" (page 4-9, line 1).

- Page 4-8, line 26: Define more specifically what are these restrictions. If they are zoning restrictions, EPA as a rule does not consider zoning as a basis for not addressing a future hypothetical land use.

- Page 4-11, line 16: Please clarify what is meant by a "high quality fishery" (i.e., why is it described as a high quality fisher?).

- Page 4-13, lines 5-7: This statement should be repeated in (or moved entirely to) the uncertainty section (Section 7).

- Except for Section 6.2, the rest of Section 6 presents a good overall summary of calculated risks for each of the EAs, exposure scenarios and receptors.

- Section 6.2: Suggest moving most of the information in this section into Section 5 (risk characterization). A summary of the information should be presented in Section 6. Because the information presented is very difficult to grasp, also strongly suggest developing a graphic or diagram of some sort to clearly convey the dioxin TEQ process, and how site data are used to derive "predicted" and "expected" dioxin TEQ concentrations. The equation presented on page 6-3 is helpful, but doesn't go far enough. Also, should add a column in the table on page 6-3 showing the "expected TEQ" concentration. To better illustrate the process, suggest showing an example calculation of the recreation exposure scenario example through to completion (data in Table 6-2).

- Page 6-2, lines 15-22: Need further explanation of "expected" vs "predicted." Also, for consistency, an equation showing calculation of adjusted TEQ (as is done for calculating expected TEQ) is suggested.

Comments on Volume 1, HHRA Attachments:

Attachment 1: Approach for Treating Non-Detects

- The approach presented in this section for dealing with analytical non-detects is adequate and consistent with EPA guidance.

- Page 6, line 12 and page 7, line 19: What is mechanistic knowledge and how is it applied? Please clarify.
- Page 8, line 11: The statement is made that "The data in area H6 do not appear to be skewed…" What is the basis of this statement?

Attachment 2: Congener vs. Aroclor Regression Analyses

General Comments

- In general and except for the following comments, Attachment 2 is well written and provides a succinct description of the Congener *vs*. Aroclor regression analyses for floodplain soils.
- Suggest providing soil physicochemical information to justify the prediction/extrapolation of congener concentrations from soil samples used in the regression data set to other floodplain soils. Although extrapolations may be supported from a statistical point-of-view, it may not be supported based on soil science. For example, are there soil physicochemical characteristics that suggest that degradation rates of PCBs might be the same/different between soils used in the regression analyses and soils for which predicted congener concentrations were performed (*i.e.,* soils from other geographic areas where congener concentrations were not measured)?
- Suggest EPA consider adding simple descriptive statistics and scatter-plots of the non-transformed data, which would be helpful (though not absolutely necessary) to provide the reader a better understanding of the distribution of the data and to support the log-transformation of the data. This information can be placed in an appendix to Attachment 2 to maintain the "readability" of this section.
- Suggest adding a discussion on the consequences of using the sum of Aroclors as a measure of tPCBs as related to (a) the regression analyses and (b) the prediction of PCB congeners. Are there specific Aroclors that would be better indicators of congener concentrations (*e.g.,* due to initial composition of PCB congeners, degradation rates)? Multivariate analyses (*e.g.,* principal component analysis [PCA]) can be performed to explore/identify possible "best" indicators that could undergo more focused analyses (*e.g.,* regressions).
- Suggest a discussion be added as to whether the four assumptions of linear regression (*i.e.,* "fixed" independent variable, linearity, dependent variable normality, homoscedasticity) are met for the analyses, and, if not met, whether the regression is robust enough for the purpose of predicting congener concentrations. For example, do the regressions meet the assumption that variance around the regression line is constant [homoscedastic] and, hence, independent of the magnitude of the independent and dependent variables)?
- Additional rationale should be added regarding the selection of regression models for predicting congener concentrations based only on the p-value (*i.e.,* $p = 0.01$) and sample size (when more than one regression met the p-value selection criterion). It is appears that the p-value referred to in the Results and Recommendations (page 4, lines 14-17) is related to whether the slope is significantly different than a slope of zero. Given that the

only purpose of these regression analyses is to predict congener concentrations, EPA should consider including the r^2-value in selecting regression models as well. The r^2-value is a measure of the variance in the dependent variable that is explained by the regression model—the "tighter" the fit to the regression line, the higher the r^2 (r^2 ranges from 0 (0%) to 1.0 (100%) of the variance that is explained by the regression model). For example, if the slope is significant ($p = 0.01$), but the regression model only explains a small proportion of the variance in the congener concentration [dependent variable] (as indicted by the r^2-value), one might not select the regression model as an adequate predictive tool.

- Suggest clarifying and adding rationale for not using a specified prediction limit (*e.g.*, 95% upper prediction limit) for obtaining an estimate of the dependent variable value (*i.e.*, congener concentration). Given the stated purpose of the regression models, use of this metric is (a) directly relevant to the use of the regression models, (b) provides a measure of confidence/assurance that the "actual" congener concentration is no greater than the predicted concentration, and (c) accounts for the fact that the further away from the mean, the less reliable are the estimates of the independent variable (this is because of the uncertainty about the true slope of the regression model).

- It is recommended that the reader be clearly informed that predictions from these regressions are best applied over the range of tPCB concentrations considered in the regression analyses. Predicting congener concentrations outside the range of the regression models (*i.e.*, outside the bounds of the data set) is problematic.

- To further ensure the clarity and transparency of the proposed use of the regression models, it is recommended that an example calculation be added to Attachment 2 that shows how the regression results are used to derive a congener concentration (including the use of the TEQ).

Specific comments

- Page 2, lines 18-27 (bullet #4): It is suggested that the 4th bullet, *Used Only Reach 5 Data*, be moved to the #1 or #2 bullet position under METHODS as it is a key [first-cut] exclusion of the data.

- Page 2, lines 12-13 (bullet #3): A reference should be added for the statement: "This additional cleanup step is not needed for other dioxin-like PCB congeners and does not affect their quantification." This reference will further support exclusion of these data from the regression analyses.

- Page 2, lines 28-32 (last bullet): Further clarification is needed as to why Aroclor concentrations quantified by GERG were not used. The method used by GERG should be specified and the rationale why this method is not comparable to Method 8082 should be provided. Method 8082 is the method "more commonly used to analyze soils samples from the site" (Last Bullet, Lines 31-32, Page 2). Were methods (other than Method 8082 and those used by GERG) used to quantify Aroclors at the site?

- Page 2, line 38: It is stated that for PCB congeners other than PCB77, PCB81, PCB126, and PCB169, there was "consistency" between (a) regression models based on 2002 data

and (b) models based on pre-2002 and 2002 data. Regression models for PCB congeners PCB77, PCB81, PCB126, and PCB169 are based on 2002 data only). This "consistency" rationale is provided to support the use of regression models for PCB77, PCB81, PCB126, and PCB169 based on 2002 data only. The meaning of "consistency" (*e.g.*, consistency based on model predictions, r^2-values, other parameters?) needs to be defined and why this consistency is sufficient to support the use of regressions based on a small data set further clarified. Results of power analyses may also provide helpful supporting information.

- Page 4, lines 7-13, Table 2: Please clarify why was the tPCB concentration of 10 mg/kg used to illustrate the comparability of predicted congener concentrations using the two regression models. It appears that 10 mg tPCB/kg is near the midpoint for most of the tPCB ranges for the "2002 data only" model. Differences between predictions using the two models tend to be minimized using midpoints as compared to a concentration near either end of the tPCB range (see spread of 95% confidence intervals).

- Figure 1: It appears that the 95% confidence intervals provided in Figure 1 are related to the slope. If this is true, for clarity, suggest specifying on the regression plots that the "associated 95% confidence intervals" are related to the slope and not related to predicted values from the regression (*i.e.*, prediction limits).

Attachment 3: Approach to Spatial Weighting of Contaminant Concentrations in the Housatonic River Floodplain

- Whereas inverse-distance weighting (IDW) is the geostatistical approach selected for spatial weighting, the document states that different approaches (e.g., kriging) were evaluated. Although I do not question that IDW may be the best approach, information should be provided on how this determination was made. A discussion on the strengths and limitations of IDW (in general and versus other approaches [e.g., kriging]) would be useful.

- How the habitat mapping was used in focusing the spatial weighting lacks transparency. Was it only used to define separate areas that were spatially weighted separately, or was it also somehow factored into the weighting (which could have been done if it wasn't--especially if these are tied to topography as indicated)? Also, how were the separate areas combined (if at all) to obtain the overall concentration map.

- Whereas IDW doesn't accommodate anisotrophy (direction), kriging does. Since this is a river floodplain, it seems as though the direction of contamination would be a factor to consider in geostatistics. Kriging would have allowed this. Was anistrophy considered in selecting IDW (vs Kriging) in selecting a spatial weighting approach?

Attachment 4: Calculation of Exposure Point Concentrations:

- Whereas the approach, methods and discussion presented in this section appears to be thorough and accurate, the presentation is cumbersome and lacks tranparency. It does not follow a straightforward path, and tends to be redundant. A flowchart of the process would improve transparency. For example, although the decisions on what methods to use in conjunction with a specific type of data distribution, a discussion of how

distributions are determined in the first place would improve transparency of the methods.

C. Phase 2 – Consumption of Fish and Waterfowl Exposure Assessment (Volume IV, Appendix C)

Charge Questions and Responses to these Questions:

1. Were the approaches and methods used to calculate EPCs for the fish and waterfowl consumption scenarios appropriate under the evaluation criteria?

- In general and except for the following comments, the approaches and methods used to calculate EPCs for fish and waterfowl consumption are considered appropriate.
- This section describes the fish and waterfowl tissue data available for use in this HHRA. Mention is made of other data that are available, but were not considered for use in the assessment. All available data should be described and subjected to the data usability evaluation, or clear explanation as to why not.
- Three criteria for selecting fish tissue data for use are identified. It is unclear as to whether these criteria were applied for an initial compilation of data, or if these criteria were applied to determine data usability in Section 2.3. These criteria include: 1) species typical of those consumed by humans, 2) tissue type representative of those consumed by humans, and 3) consistency with data quality objectives. It is unclear as to whether temporal trends were considered in the selection of fish and waterfowl tissue data for risk assessment, since this was not listed as a criterion, and is not discussed in Section 2. However, it appears from comparison of Table 2-1 (Sources of Data Used in the Fish and Waterfowl Risk Assessment) with the data sets described in Section 3 (Data Usability and Validation) that fish tissue data collected before 1984 were not considered for use in the HHRA. The selection of applicable and usable data for HHRA purposes should follow a systematic and transparent process.
- Page 1-1, lines 23-25: Should refer to some other document or source regarding the discussion on the floodplain extending to the 1 ppm isopleth and its correspondence to the 10-year floodplain.
- Page 1-19, lines 9-10: Further discussion of the probability bounds analysis is needed, or reference to another section of the report where this additional discussion can be found.
- In Section 2.2.1.1, it is stated that Total PCB (tPCB) concentrations for EPA Supplemental Investigation Data were calculated from PCB congener concentrations, but in Section 2.2 (Recent GE Data), tPCBs were analyzed as Aroclors. If tPCBs, as described in Section 2.2.1.1, was calculated as the sum of the PCB congeners, it is important to understand which congeners were specifically included in the summation. Section 2.3.1 indicates that 120 congeners were quantified in the EPA Supplemental Investigation data set, but it is unclear whether all 120 congeners were summed to estimate tPCB concentrations. If tPCB referred to in Section 2.2 are actually Aroclor data (or the sum of Aroclor concentrations), then the data should be described as such. Throughout the risk assessments, the term "total PCB" and "tPCB" should be defined more definitively. Since the laboratory animal toxicity studies of Arcolor mixtures reflect toxicity associated with very specific and well-defined mixtures (e.g., Aroclor 1254), the uncertainty associated with the application of these toxicity data to PCB data represented

by different analytical methods should be described in detail, including the implications of the assumptions used to combine data from different sources. To that end, the specific PCB analytical methods used for each data set used in the risk assessment and the methods and rationale for combining data should be described in detail.

- Data usability was determined by assignment of a "score" to each data set based on criteria presented in Table 2-6, and the scores for each data set are presented in Table 2-7. However, there is no indication of the individual criteria scores for each data set, and therefore, it is not possible to ascertain the basis for selecting specific data sets as usable for risk assessment.

- In Section 2.3 (Usability and Validation), there is no discussion of Data Validation for any data set, nor is there any detailed discussion of data quality with respect to field duplicate samples, laboratory quality control samples, or estimated and rejected data.

- Tables 2-2, 2-4, and 2-5 summarize EPA Supplemental Investigation Data, Recent GE Data, and Historical Data for fish tissues. These data include fillet (skin-on and skin-off), composite, whole body, offal, and ovaries data. These tables should indicate which data were used in the quantitative risk assessment.

- Page 2-1, line 25: Again, an explanation should be provided as to why other edible body parts were not sampled (see comment in ES-5, line 7 above).

- Table 2-1: Of the data sources described in this Table, those determined to be usable for risk assessment include "EPA data, recent GE data, and the data from Coles, 1996." (see Section 2.3). The discussion of these data is not balanced. There is an extensive discussion of the EPA Supplemental Investigation Data, a brief discussion of the Recent GE Data, and no discussion of the Coles (1996) data. Furthermore, there is no explanation as to why the Coles (1996) data were included in the risk assessment, but the Smith and Coles (1997) data, the MADEP data, and the State of Connecticut data were excluded.

- Page 2-4, lines 19-20: Should reference a section where the tPCB concentrations were calculated from congener concentrations are explained.

- Page 2-6, lines 1-8: When referring to tissue concentration data, please clarify whether all concentrations are lipid normalized, or expressed on some other basis.

- Page 2-7, line 1: Before (pg 2-6, line 18), the statement is made that waterfowl are year-round inhabitants of the area. Here, reference is made to migration. Please clarify whether the birds of interest migratory or non-migratory.

- Page 2-7, lines 9-10: Again, no thigh or gizzard analyses were conducted. Is it assumed that concentrations in breast represent thigh as well?

- Page 2-7, line 20: Please clarify how/whether fish data were used in the revised RCRA permitting process. Further explanation here warranted.

- Page 2-11 to14, Tables 2-5. Given the fish analyses go back as far as 1977, the RA should contain more information verifying that all the data are usable for RA

purposes…DQO process. Please clarify the makeup of the DQO team, and whether a separate DQO report was issued.

- Page 2-15, lines 4-5; 11-13: This DQO statement is very brief. Please clarify whether there is a separate DQO report. A reference to Attachment C.2 should be added.
- Page 2-23, line 9: Ice fishing has not been addressed in this HHRA. Please add information on the relative frequency of winter ice fishing compared to other times of the fishing season.
- Page 2-25, line 18: Please be specific as to what criteria are be referred to here.
- Page 2-27, line 4: Should comment on other edible tissues (see comment under ES-5, line 7 above).
- Page 2-29, line 10: The reference of this USGS study done for EPA should be provided.
- Page 2-31, line 1-2: Should comment/discuss the applicability of these RBCs and the basis of their derivation. Reference table 2-12, which provides some basis. Should explain that their use in this HRA is in accordance with their intent.
- Page 2-35, table 2-12: Please provide the basis for the 54 g/day fish ingestion rate, which seems very high.
- Page 2-41, lines 24: Combining perch and sunfish was done in Reach 5/6, but not in Rising Pond. An explanation of why this was not done in Rising Pond is warranted.

2. Were the exposure assumptions and parameters used in both the assessments of fish and waterfowl consumption appropriate under the evaluation criteria?

- In general and except for the following comments, exposure assumptions and parameters used in this HHRA are appropriate..
- Figure 1-5: It is not clear why an air/inhalation exposure pathway is identified as a complete pathway associated with the 'fish and waterfowl consumption' pathway on the CSM. As far as I can tell, it is not addressed with further (i.e., no qualitative discussion as to why it is eliminated from quantitative analysis.
- Page 4-2, lines 17-18: The basis for the potentially exposed population being anglers who consume at least one meal per year from the Housatonic River should be explained.
- Page 4-3, lines 24-25: The basis for the potentially exposed population being hunters who consume at least one meal per year of waterfowl from the Housatonic River should be provided, along with relevant discussion.
- Page 4-16, lines 6-8: EPA's conclusion regarding the lack of subsistence fishing should be explained in more detail or a study referenced.
- Page 4-16, lines 11-13: The basis (reference?) of this statement that fetuses and young children are particularly sensitive to PCB adverse effects should be provided.

- Page 4-22, Table 4-4: This table should reference page 4-31 where an explanation is given re. exposure prevalence study vs volunteer study.
- Page 4-31, lines 1-2: It is not clear how the prevalence data were used in the HHRA.
- Page 40 (Parameter Selection). The adult fish consumption rates used are taken from the Maine Angler Survey, which is clearly the most robust angler study available for the northeast. The 90th percentile and arithmetic mean adult consumption rates were selected to represent the RME and CTE rates, respectively, which seems reasonable. However, the study reported rates for "all waters" and for "streams/rivers", and the higher "all waters" rates were selected for use in the risk assessment. The 90th percentile rates are 32 and 15 g/day for "all waters" and "streams/rivers", respectively. The arithmetic mean rates are 14 and 8.9 g/day for "all waters" and "streams/rivers", respectively. Note that for trout, the "streams/rivers" rates were used. The rationale for selecting "all waters" fish consumption rate data should be described, including the difference between "all waters" (does this include marine waters?) and "streams/rivers".
- Page 4-41, lines 10-17: Please clarify why the 90th percentile is applied instead of the 95th. Should provide additional rationale. Also, it is not whether the 90th vs 95th is based on professional judgment or some other basis.
- Page 4-41 (Child Consumption Rate): The child fish consumption rates were estimated as approximately one-half of the adult rates. This assumption seems to be arbitrary given that there is no information provided to support the assumption. Given that the hypothetical child weighs 4.7 times less than the hypothetical adult (15 kg versus 70 kg), the differences in body weight alone do not support the assumption that a child consumes one-half the amount of fish as an adult. However, differences in dietary requirements and consumption rates for other foods may provide basis for a more reliable estimate of a child fish consumption rate.
- Page 4-44 (Cooking Loss): Cooking loss for the RME scenario was assumed to be zero "based on data from several studies showing no cooking loss, and that individuals may consume the drippings/pan sauce." The studies showing no cooking loss are not cited. Furthermore, it does not seem logical that there would not be some cooking loss. Explanation as to why some studies showed cooking loss and others did not should be provided. Also, clarification as to whether detection limits in those studies that did not detect cooking loss were sufficiently low. The statement that "individuals may consume the drippings/pan sauce" seems to be based more on speculation than on actual facts. Note that CTE cooking loss percentages ranged from 21 to 24% for MA and CT, respectively, based on cooking preferences.
- Page 4-50, lines 12-13: Please clarify why no cooking loss was assumed for other COPCs, especially one like DDT.
- Page 5-50: (Fraction Ingested). The fraction of fish ingested from the Housatonic River was assumed to be 100% based on Ebert et al. (1996). However, as noted in the risk assessment, Ebert et al. reported that only 1.5% of 1,515 respondents to the Connecticut Housatonic River Creel Survey caught all their fish on the Housatonic River, and only 9.9% of these individuals indicated that at least 95% of their fishing trips were on the

Housatonic River. They noted that the assumption is reasonable since the survey was conducted while the fish consumption advisory was in place.

- Table 4-22: Recommend showing a calculation in a footnote for the preference weighting…its not immediately clear from the table.

3. Was the basis for the selection of point estimate RME and CTE exposure parameter values appropriate under the evaluation criteria, and were they clearly described and referenced?

- In general, I believe the RME and CTE exposure parameter values are appropriate for purposes of this HHRA.

- An explanation as to why it is reasonable to combine the 90th percentile fish consumption rate with the 95th percentile exposure duration for fish consumption should be provided.

4. Were the probabilistic approaches used clearly described, and were they appropriate under the evaluation criteria?

- In general and except for the comments below, the probabilistic approaches used are clearly described and appropriate.

- Knowing that probabilistic risk assessment is difficult for even practitioners to grasp, I suggest an expanded discussion on some of the basics, specifically a clearer discussion on how the results of the probabilistic analysis and Monte Carlo simulations are used in risk management decision making. For example, in the introduction to Section 6, it would be helpful to readers not familiar with probabilistic analyses to have an explanation as to what is meant by a probabilistic uncertainty analysis versus a probabilistic risk assessment (PRA). These terms are intermingled throughout, and are identical, but how the results are used in decision making differs. The explanatory information that is presented in Section 6 is excellent, but it doesn't go far enough. More examples to illustrate and convey PRA concepts would be helpful.

- This section presents many figures, which contain a substantial amount of information. However, the text does not adequately explain how all this information is used in the risk assessment, and in risk-based decision making. Perhaps a figure similar to Figure 8-1 should be placed in this section to help educate readers.

- The rates of fish ingestion used in the probabilistic risk assessment are based on the exposure frequency (meals per year) and ingestion rate (grams per meal). For ingestion rate, separate CTEs are used for the adult and child; however, both the child and adult scenarios use the same exposure frequency. Thus, the result of a child consuming up to 2.9 meals per day is equivalent to a maximum fish ingestion rate of 331 g/day. This does not make sense.

- Page 6-1, lines 19-24. Again, this place in the document would seem to be a good place to state with appropriate citation that EPA's default percentile is the 95th percentile.

- Page 6-12, lines 20-23. These two sentence are an example of the use of two terms (probability distribution, Monte Carlo input variable) that may mean the same. The result

is confusion. Consistency of use of terms is very important, especially in a PRA because of the inherent complexity.

- Page 6-15, lines 1-3. This statement is poorly worded and thus confusing. Please clarify what is meant by "…accounting for sampling uncertainty by using the EPC in place of the sample mean in probabilistic risk analyses." The same comment applies to page 6-49, lines 17-18.

- Page 6-17,lines 11-16. This would be a good place to explain how "exceedance probability" is used in this risk assessment (i.e., in terms that a risk manager can understand). Perhaps using an example to explain would be helpful.

- Page 6-36, lines 4-7: Again, this would be a good place to explain in practical terms how the information is used in the risk assessment…to assist with transparency and clarity.

5. Were the distributions used in the probabilistic assessments clearly described, and were they appropriate under the evaluation criteria?

- In general and except for the following comments, I believe the distributions used in the probabilistic assessment are well described and appropriate.

- Page 6-13, Table 6-2 (input parameters for probabilistic exposure assessment). The maximum exposure frequencies presented for bass and trout are 1,042 and 761 meals per year, respectively, based on empirical data distribution. The adult ingestion rate is the CTE point estimate of 227 g/meal, which is EPA's default meal size value. The maximum EF for bass of 1,042 meals/year correlates to 2.9 meals per day, and essentially assumes that this individual consumes fish with every meal. At a rate of 2.9 meals per day and an ingestion rate of 227 g/meal, this individual would consume 648 g/day. This rate greatly exceeds the 90^{th} percentile adult fish ingestion rate of 32 g/day selected as the RME point estimate ingestion rate for adults. Also, please clarify why tPCB concentrations were not expressed as a range or distribution, rather than a point value.

- Page 6-58, lines 16-17. Although the 90^{th} and 99^{th} percentiles can be more or less visualized from the inset boxes, it is not accurate to say the percentiles are highlighted. Also, showing an example (e.g., Figure 8-1) would help clarify for readers how to read these inset boxes.

- Table 6-14 to 6-19. Suggest a more visual way, in addition to the tables, to show the relative uncertainty contribution of the variables.

- Page 6-106, Section 6.10. In my opinion, the method used here to assess and convey professional judgment regarding each of the sources of uncertainty is not very effective. Why not apply a standard qualitative approach per RAGS. Also, the fact that so many of the parameters in Table 6-23 are assigned a "?", which, as is explained on page 106 "…have mixed or uncertain bias consequences for the analysis", will likely be confusing to readers (e.g., risk managers). Is it not possible for professional judgment to be applied a assign either a "C" or "O" to the parameters assigned a "?"?

6. Were the uncertainties in the data and models adequately characterized and expressed?

- Uncertainties in the data and models are not adequately characterized and expressed. Section 7 needs improvement. In general, Section 7 does not present uncertainty information in a way useful to risk managers in risk management decision making. There does not appear to be consistency in statements as to whether items discussed are considered to either under- or over-estimate risks. Section 7 could greatly benefit from an overall qualitative analysis of uncertainties and whether, in the view of the risk assessor, overall risk estimates are considered as either under- or over-estimating health risks.

- Section 7.2: A general comment: There is no discussion of the uncertainty associated with the PCB analytical data and reporting of PCB concentrations as total PCBs (see additional discussion regarding total PCBs in Volume IV, Appendix C comments).

- Page 7-2, line 25-page 7-3 line 5: The discussion of "Excess" PCB Congener Calculations describes the uncertainties associated with double counting Aroclor and TEQ risks, but does not address the uncertainty associated with the use of PCB TEFs relative to the use to Aroclor toxicity data. There is likely to be substantially more uncertainty associated with the estimation of risks using the PCB TEFs than using the Aroclor toxicity data. For Aroclor mixtures, toxicity data are based on in vivo studies focused on frank toxicological responses such as cancer, reproductive, development, and target organ effects. The congener-specific PCB TEFs are largely based on biological responses, not necessarily frank toxic effects, such as Ah receptor binding affinity and enzyme induction. Extrapolation from these types of biological responses to frank toxic effects is highly uncertain. Furthermore, the in vitro assays used to develop the PCB TEFs were largely based on studies of individual congeners, not mixtures as found in the environment or in the original PCB formulations, and therefore, due not account for synergistic and antagonistic interactions between the various congeners.

- Page 7-3, line 10: Please clarify whether the reference to "pesticide" applies to all COPCs or just to pesticides?

- Page 7-4, line 3: The phrase "…changing substitution values…" is unclear. The text should define substitution values, or reference a location in the document where this terminology is defined.

- Page 7-4, lines 33-39: Risks associated with pesticides were not estimated for this pathway. Therefore, it does not seem relevant to state that pesticides "…contribute less than 1% of the cancer risk and HI…" Moreover, nowhere in the document was it stated that the GE plant in Pittsfield is considered a source of pesticides in the Housatonic River.

- Page 7-5, section on Skin-off Filets: It is my opinion that a risk manager will have difficulty understanding the uncertainty associated with the skin-off fillet vs skin-on fillet from the discussion presented. Too much information is given. A more concise discussion should be provided, with greater emphasis on how this issue impacts the calculated risk estimates.

- Page 7-5, line 38-page 7-6, line 2: This section is too brief and deserves greater explanation. Specifically, a better understanding as to why the author(s) believe the dioxin/furan congener patterns are approximately the same in CT and MA is needed.
- Page 7-6, lines 5-7: The authors should state that the reason they expect concentrations in smaller fish to have lower COPC concentrations, thus lower EPC and possible risk underestimation, is that the bioaccumulation time is less in younger fish.
- Page 7-6, lines 30-39: It is difficult to ascertain what a risk manager would do with the information presented in this section. Some further clarification is suggested.
- Page 7-8, lines 22-23: The assumption referred to in this sentence is not clear. It appears the assumption is that tissue concentrations measured in dabbling and perching ducks is the same as what would be found, if measured, in diving ducks. Clarification is needed. Also, if diving ducks are migratory, it would seem lower concentrations in tissues would be expected because of the briefer exposure duration.
- Page 7-9, lines 1-12: Because frogs are harvested in the area and because frog leg tissue data were collected from Housatonic River frogs, it is surprising that risk estimates were not derived. The information could have been directly useful in risk communication with people who may wonder whether consuming frog legs harvested from the area poses an unacceptable health risk or not.
- Page 7-9, lines 13-28: This section does not convey whether, in the author's view, that the way cooking loss was dealt with in the risk assessment leads to an over- or under-estimation of health risk. This should be corrected.
- Page 7-9, lines 29-page 7-11, line 6: Part of this section seems redundant with the "fish preference" section on page 7-6. Also, it is not clear how "sharing" of a catch impacts individual fish consumption rates. The implication is that an individual's consumption rate is reduced through sharing the catch with others. Please clarify the basis for this assumption.
- Page 7-11, lines 8-11: The use of the words "significantly underestimates exposure duration" is unclear in this statement. An increased exposure duration from 30 to 45 or even 63 years does not seem like a significant increase in risk.
- Section 7.2.3: This section is too brief and doesn't convey uncertainty associated with toxicity information from laboratory animals used in deriving toxicity criteria used in the HHRA (e.g., CSFs, RfDs).
- Section 7.2.3.1: Numerous commentaries and reviews have been published over the past 15-20 years describing the uncertainty associated with relying on toxicology data from laboratory animals administered high dosages in humans exposed to much lower dosages. This section is completely lacking any such discussion. Furthermore, no epidemiology information in humans is given to assist the risk manager in evaluating the uncertainty (or over-estimation of risk estimates) associated with relying solely on animal toxicology data for assessing human health risk.

- Page 7-14, lines 3-6: Statements such as this should be referenced, even if a personal communication. The statement is: "EPA is currently reviewing new studies on noncancer effects of PCBs as part of the ongoing IRIS review process. These studies report possible associations between developmental and neurotoxic effects in children from pre-natal or post-natal exposures to PCBs." Without dose-response information provided simultaneously, such statements fuel public concerns unnecessarily.

- Section 7.2.3.2: In view of the overall importance of the use of TEFs in this HHRA, expanded discussion of TEFs and uncertainty associated with them is warranted. For example, the basis of the TEFs is Ah binding and CYP1A1 induction. The association between enzyme induction (AHH) and toxicity, and extrapolation of this effect in animals to humans, should be discussed.

- Section 7.2.3.3: This section refers to the EPA's proposed draft TCDD cancer slope factor (CSF) of 1E+06 $(mg/kg\text{-}d)^{-1}$. The EPA's Science Advisory Board (SAB) has provided comments to EPA on this issue, yet the EPA has not yet released a revised document for public or peer review. The derivation of this CSF is a highly controversial issue due to EPA's failure to clearly describe and document its derivation, and because it appears to be based largely on epidemiological studies for which there are substantial confounding factors and spurious associations. Furthermore, the uncertainty discussion specifically fails to make reference to other alternative CSFs for TCDD published in the peer-reviewed literature and by other federal regulatory agencies, specifically those published by the FDA (1992) and Keenan et al. (1991). In the absence of a final consensus in the scientific and regulatory communities on the potential human carcinogenicity of TCDD, it is speculative and presumptuous to even discuss EPA's draft CSF for TCDD in this risk assessment. Should EPA decide to keep this discussion in the uncertainty section, it is recommended that the full range of possible TCDD CSFs published since the Pathology Working Group reported its findings of the 1990 re-evaluation of the Kociba et al. (1978) rat liver pathology slides (Sauer 1990; Keenan et al. 1991; Goodman and Sauer 1992) be included also.

- Section 7.3.2: Further clarification of this section is needed. For example, how would a risk manager use the information presented in the rightmost column of table 7-5 in his/her decision making? Information in this column is described as an "average effect" (page 7-16, line 12), which implies there's a range.

7. Were variability and uncertainty in the risk estimates adequately characterized and expressed?

- In general, as described in 6 above, uncertainty and variability were not adequately characterized.

8. Overall, was the approach used to assess risk from consumption of fish and waterfowl and other wild food items reasonable for evaluating the baseline risk?

In general, I believe the approach used to assess potential human health risks associated with consumption of fish and waterfowl and other wild food items in the Housatonic River area is reasonable, but lacks transparency and consistency in a number of instances. The assessment, as done, is highly likely to over-estimate risks to the receptors assessed via these

pathways, as a result of the application of muliple upper-bound assumptions. This is all the more reason that a thorough identification and analysis of uncertainty should be included.

I also have concerns about the apparent miss-use of the Ebert et al (1993) fish consumption data; a number of incorrect applications of these data were presented by Dr. Ebert herself at the November 18-20, 2003 public meeting. Although it is highly unlikely that EPA has underestimated risks to people who might consume fish or waterfowl taken from the Housatonic River area, I believe it is highly likely these risk have been grossly over-estimated. I strongly recommend that EPA re-assess this fish/waterfowl consumption pathway, taking into consideration comments especially related to exposure assessment.

Additional Reviewer Comments

- Table 3-4 indicates that an RfD of 1E-04 for methyl mercury will be used, but instead an RfD of 3E-04 for mercury chloride is used in the Risk Characterization to calculate HQs.
- All Tables should be checked to make sure that footnotes are added, as needed, to reference where in the text relevant explanations/discussion are given.
- In Section 5, risk characterization methods and results are clearly and well presented, both in graphic and tabular form.
- All cancer risk estimates should be termed "theoretical upperbound cancer risk estimates", rather than simply cancer risk. Readers need to understand the estimates are both theoretical and upperbound.
- Section 5 (risk characterization) lacks a proper discussion that would assist readers in placing the results in proper perspective. With theoretical upperbound cancer and non-cancer risks as high as these are, readers could benefit from the addition of some information that might help place these results in perspective (e.g., comparison of the calculated exposure levels associated with fish and waterfowl consumption with actual human exposures reported in the literature and data on associated toxic effects, if any). As was mentioned in regards to the Phase 2 Direct Contact HHRA, comparison of estimated risks with study results such as those in MADH (1997) and ATSDR/MDPH (2002) would be appropriate. These studies, as presented by GE at the November 18-20, 2003 public meeting, indicate that no increase in neither human blood levels nor cancer incidence rates were measured in the Housatonic River area. Comparison to these kinds of studies help place the HHRA estimates in proper perspective.
- In Table 5-9, the RfD of 2E-05 mg/kg-day for Aroclor 1254 was used to estimate noncancer effects associated with Housatonic River PCBs, which are predominately Aroclor 1260. Based on available data, EPA specifically derived RfDs for Aroclor 1016 and 1254. EPA has not recommended the use of these RfDs for assessing noncancer risk to other Aroclor mixtures (IRIS Verification Date 2/16/1994). Neither EPA Region 3 nor Region 9 utilize the Aroclor 1016 and 1254 RfDs for deriving Risk Based Concentrations (RBCs) or Preliminary Remediation Goals PRGs), respectively, for other Aroclor mixtures (EPA 2002, 2003).

Attachment C.4. Total TEQ Calculations

- PCB risks are calculated separately, and then summed, for tPCB and PCB congeners. To avoid double counting exposure/risk, the contribution of TEQ from tPCB (based on the TEQ fraction) was subtracted from the total PCB congener TEQ. Thus, Aroclor exposure/risks are based on the full measured concentration of tPCB, whereas the PCB congener risk is based on the fraction of exposure/risk not accounted for in the tPCB exposure/risk estimate. As an example, Table 5-3, Cancer Risks from Fish Consumption for Each COPC, gives RME cancer risks of 8E-03 for tPCBs and 5E-03 for TEQ risks (minus the contribution from tPCBs). Theoretically, since the source of the 12 PCB congeners is the Aroclor mixtures released, then the TEQ contribution from the Aroclor mixture subtracted from the TEQ based on measured congener concentrations should equal zero. However, the fraction of TEQ in the tPCBs appears to represent only 6.1% of the measured TEQs. There are several possible reasons:

  - PCB mixtures have weathered over time resulting in a relative increased proportion of the 12 coplanar congeners compared to other congeners either by transformation or degradation processes.

  - The TEFs upon which the TEQs are based may overstate the dioxin-like potency of individual congeners: Aroclor toxicity values are based on in vivo animal bioassays of the PCB mixture and therefore assess not only actual toxic endpoints but also account for possible synergistic and antagonistic relationships among congeners. Conversely, TEFs are based largely on Ah binding and AHH induction of individual congeners, and therefore, do not assess actual toxic endpoints and do not account for possible synergistic and antagonistic relationships among congeners.

  - The CSF for TCDD may be overstated to the extent that PCB congener TEQ risks are substantially higher than estimated Aroclor risks.

  The description of the TEQ method of applying the TEQ method to avoid double counting suffers from both lack of clarification and transparency. Based on information presented by Dr. Russ Keenan at the November 18-20, 2003 public meeting, it appears that there is in fact incorrect. I strongly recommend that EPA consider Dr. Keenan's information and re-evaluate the necessity of using the TEQ approach in this HHRA.

- For a number of samples, the TEQ value for some dioxin, furan, and PCB congeners is 0.00. For these samples, the reported concentration in Attachment C.3 (Raw Data) is also shown as 0.00, sometimes with either a J or U data qualifier. It is unclear why these concentrations are shown as 0.00, and how these data are used in the risk assessment. If these analytes were not analyzed for or if these data were rejected during data validation, then this should be indicated. If these values of 0.00 were included in summary statistics or EPC calculations, then there may be errors in the calculations.

D. Phase II – Agricultural Exposures (Volume V, Appendix D)

Charge Questions and Responses to these Questions:

1. Were the exposure scenarios evaluated appropriate and reasonable for current and reasonably foreseeable future use of the floodplain?

- In general and except for the comments below, I believe the exposure scenarios evaluated for the current and future land use of the floodplain are appropriate and reasonable. The following comments relate primarily to lack of clarity and transparency for some items.
- Page ES-5, lines 8-15: A more definitive statement about the potential for future backyard farms is needed. The extent of the potential for these farms should be more clearly discussed.
- Page ES-8, line 21: It is important, even in the executive summary, to explain the basis of the assumed 0.5 mg/kg and 2 mg/kg soil concentrations of tPCBs. Also, a reference should be provided for the statement made in lines 21-22 that 2 mg/kg "…is the current remediation goal for current residential properties." The relevance this level to the HHRA should be clearly stated; whether or not any level greater than 2 mg/kg tPCBs means remediation will be done.
- Page 1-1, line 25: As already indicated, it is somewhat confusing as to why 2 mg/kg was the assumed soil concentration for risk assessment purposes instead of 1 mg/kg, which seems more logical. The 1 mg/kg concentration is equated to the 10-year floodplain area.
- In Section 2, various food and livestock feed media that were sampled and analyzed for PCBs and sometimes PCDD/PCDFs are described. Some sections note that data were reported on a wet weight basis and some on a dry weight basis. Please clarify whether these data are all normalized to a standard weight basis consistent with applicable consumption rates. Also, there is no data usability discussion and very little mention of data validation. In some cases, the analytical method used is not identified (e.g., milk samples analyzed by the USFDA, which is mentioned in Section 2.3.1.2).
- Page 2-1, lines 24-25: Please clarify whether cattle access to the river, which has been observed near the CT border, represents a potential exposure pathway in the MA Reaches.
- Page 2-2, line 12: The farmer interview information, which is compiled and summarized elsewhere in the HHRA (I don't recall where), should be referenced here.
- Page 2-3, lines 10-11: Please clarify and discuss the potential significance of the growing of field corn for sale as corn silage to other farmers (e.g., a commercial dairy outside the floodplain area). Also, please note any analytical data that may have been collected on this corn.
- Page 2-4, lines 25-26: While it is highly likely that the assumption that high-moisture corn grown on the floodplain does not contain levels of COPCs greater than background concentrations, this assumption should be verified at some point with sampling data.

- Page 2-6, Section 2.1.2: In light of the presentations made at the November 18-20, 2003 public meeting, this section needs to be more concise as to the potential for non-commercial, backyard farms. The point was made rather strongly at the meeting that significant backyard farming interest exists in the Housatonic River area.

- Page 2-8, Section 2.2.1: Better justification for using residential PRG values for screening agricultural produce should be provided. It is interesting that the COPC selection for agricultural and livestock exposure pathways utilized a screen against Region 9 PRGs whereby chemicals were excluded as COPCs if less than 10% of the samples exceeded PRGs. Since Region 9 PRGs do not address any food consumption pathways, this screen is not relevant. Please provide rationale for not developing agricultural SRBCs for this purpose.

- Page 2-10, Section 2.3.1: This section suggests that milk samples were analyzed for tPCBs. Since there exists different analytical methods for Aroclors, congeners, and total PCBs, all reference to PCB analysis should specify exactly what was analyzed for so that reviewers and risk managers can put into perspective the data used in the risk assessment. This is extremely important for understanding risk characterization findings since the toxicity values used to estimate risk are based on specific congeners or PCB mixtures, not tPCB.

- Page 4-1, Section 4.1: This comment relates to the 0.5 mg/kg and 2 mg/kg assumed concentrations. Exposures were estimated based on assumed average soil tPCB concentrations of 0.5 and 2 mg/kg; the 2 mg/kg tPCB soil concentration correlates to the residential cleanup goal and the 0.5 mg/kg tPCB soil concentration was selected represent a lower tPCB concentration. The rationale for not using actual data for the agricultural exposure pathways was that it would be too difficult to assess every parcel individually. It was further assumed that 100% of pasture and cultivation areas are within the 1 ppm tPCB isopleth. If the assumption was to use average tPCB concentrations of 0.5 and 2 mg/kg, what does this latter statement mean? Also, isn't soil concentration directly (linearly) related to exposure and risk? Thus, what is the point of this exercise? The report further states that, "the result obtained by assuming a tPCB soil concentration of 0.5 mg/kg would also be obtained for a parcel where 10% of the land cultivated with corn silage was contaminated with 5 mg/kg tPCBs, and the remaining 90% of the cultivated land was not contaminated with tPCBs." What is the rationale for this analogy? It seems that regardless of the percentage of land contaminated with PCBs, the total acreage of land that is contaminated with an average tPCB concentration of 5 mg/kg may still support feed for all livestock raised on a particular farm.

- Page 4-2, lines 13-15: Soil PCB and PCDD/PCDF congener concentrations were estimated from regression equations that related tPCB concentrations to congener concentrations. This approach seems reasonable for predicting PCB congener concentrations, however, only specific PCDF congeners (not PCDD congeners) have been associated with PCBs, and this association has only been demonstrated in cases where PCBs have been subjected to elevated temperatures such as in the Yusho and Yu-Cheng rice oil incidents, and as demonstrated in studies of combustion and incineration of PCB mixtures.

- Page 4-2, lines 16-27: The report indicates that only the following food exposure pathways were quantitatively evaluated: (1) commercial dairy, beef, and poultry; (2) backyard dairy, beef, and poultry; (3) home gardens. However, Section 4.2 also describes methods for estimating PCB and PCDD/PCDF concentrations in goats, sheep, and deer. In Section 4.2.1.3 (Other Mammalian Species), it is stated that "the BCFs for milk and beef in cattle are used to estimate the milk and meat accumulation by these species [goats, sheep, and deer] in this assessment." It appears that the BCF selected for cattle is based on a dietary exposure (feeding study) that directly reflects the animals' rate of intake of food. Rationale should be provided explaining how this BCF is applicable to other livestock species that have different food intake rates and dietary requirements.

2. Were the approaches used to estimate transfer of COPCs from soil to plants appropriate under the evaluation criteria?

- In general and except for the following comments, the approaches used to estimate transfer of COPCs from soil to plants are appropriate.

- Page 4-24, Section 4.3.3.2: Soil-corn transfer factors were estimated for PCB congeners based on the ratio of the tPCB soil-grass to tPCB soil-corn transfer factor and then application of the ratio to the PCB congener soil-grass transfer factors. It is not clear why this approach was not also applied in deriving soil-exposed vegetable transfer factors. Clarification is needed.

- Page 4-25, Section 4.3.4: Because neither PCBs nor PCDD/PCDFs are highly volatile and neither group of chemicals appear to accumulate significantly by translocation via the roots, the only remaining viable pathway is airborne deposition of soil dust (containing PCBs and PCDD/PCDFs). Thus, bioaccumulation does not technically occur to any great extent, but rather these compounds are adsorbed to the surfaces of plants and fruits. Since such deposition is dependent on the generation of fugitive soil dust and subsequent deposition on plant and fruit surfaces, which is virtually 100% a function of site-specific conditions, it seems then that only site-specific data should be used for deriving transfer factors for assessing soil-to-plant exposure pathways. The relevance of applying soil-to-plant transfer not based on site-specific data is questionable. As a minimum, this subject should be addressed in the uncertainty discussion.

- Page 4-26, lines 27-29: The tPCB soil-exposed vegetable transfer factor is based on the "mean of the soil samples corresponding to corn ear and stalk and leafy material samples." Does this suggest that individuals consume corn leaf and stalk material?

- Page 4-28, lines 5-6: The soil-exposed fruit transfer factor was set equal to the soil-exposed vegetable transfer factor since no data were available for transfer of PCBs and PCDD/PCDFs from soil to exposed fruits. The authors note that this is "…a conservative estimate…". The authors further note that this pathway may not be important. The authors should consider eliminating this pathway as an insignificant pathway, and also because data are insufficient for making reliable exposure estimates.

- Figure 4-3 should specify (on the figure) that the plant uptake factors and vapor pressures are for PCB congeners. Note that this relationship should not be considered causal based

on the data presented (i.e., it should not be inferred from this association that plant uptake of PCBs is caused by volatilization of PCBs).

- Figure 4-5 presents several soil-plant transfer factor values of 0.00. Values of 0.00 were also observed in several other tables in the risk assessment. In most cases, a value of 0.00 is meaningless; rather than using a value of 0.00, it is recommended that a footnote or other character such as "N/A" be used to indicate that a value could not be determined or that the measure is not applicable.

3. Were the approaches used to estimate the bioaccumulation of COPCs in animal tissue appropriate under the evaluation criteria?

- In general and except for the comments below, the approaches used to estimate the bioaccumulation of COPCs in animal tissues is appropriate. However, the discussion of all BCFs needs improvement in clarity and transparency. For example, the weight basis upon which BCFs are derived and are applicable, and specifically what the BCF represents (e.g., soil-to-whole egg BCF, grass-based feed to beef fat BCF, etc.). It appears that some BCFs are based on lipid or fat data, and it needs to be clarified that all data and rates were adjusted to a whole edible tissue basis.

- Page 4-3, Section 4.2.1: Two "important" pathways are described; Soil ? Vapor/Particulate ? Plant ? Animal ? Product, and Soil ? Animal ? Product. The "Soil ? Vapor/Particulate ? Plant" pathway is quantitatively evaluated using soil-to-plant transfer factors. The "Soil ? Animal and Plant ? Animal Product" pathways are quantitatively evaluated using BCFs. The discussion of BCFs (Section 4.2.2.2) precedes the discussion of soil-plant transfer factors (Section 4.3.3.1). From a systematic perspective, it seems more logical to present the soil-plant transfer factors before the BCFs.

- Page 4-8, Section 4.2.2: The equation used to estimate livestock animal fat PCB and PCDD/PCDF concentrations is presented in Equation 5:

$$Cprod = (BCF*R*D_{soil}*C_{soil}) + (BCF*D_{sil}*C_{sil}) + (BCF*D_{grass}*C_{grass}) + (BCF*D_{con}*C_{con})$$

Where:

R	= bioavailability, assumed to equal 1.0
D_{soil}	= fraction of dry matter intake assumed to be soil
C_{soil}	= PCB or PCDD/PCDF soil concentration
D_{sil}	= fraction of dry matter intake assumed to be corn silage
C_{sil}	= PCB or PCDD/PCDF corn silage concentration
D_{grass}	= fraction of dry matter intake assumed to be grass based foods
C_{grass}	= PCB or PCDD/PCDF grass based food concentration
D_{con}	= fraction of dry matter intake assumed to be concentrate
C_{con}	= PCB or PCDD/PCDF concentrate concentration

From this equation, it appears that the same BCF, which also appears to be based on unspecified dietary exposure, was applied to soil, corn silage, and grass-based feed. Since concentrates were assumed to be grown outside the floodplain, C_{con} was set to zero, thus

canceling the concentrate term. PCB and PCDD/PCDF concentrations in corn silage and grass-based feed were estimated using soil-plant transfer factors. It is not transparent as to how the soil term in equation 5 was addressed with respect to ingestion of soil particles in feed. It is not clear whether the BCFs for grass-fed animals, for example, already include the soil component (i.e., were the BCFs derived based on unwashed feed). Clarification is needed.

- Table 4-4a: This table, which is a summary of BCFs used in the risk assessment, should indicate (footnote) the weight basis of the BCFs presented and the primary study(s) upon which each BCF is based. Also, it should be made clear whether the mammal BCFs are on a whole body basis or on a fat basis.

- Page 4-17, line 28-page 4-18, line17: A multi-step process was used to estimate BCFs for PCB congeners using the equation and information given. Estimating the BCF by this method utilizes a substantial number of factors, each with an unknown degree of variability and uncertainty, including K_{ow}, percent absorption, K_{ow} – absorption regression, intake rate of dry matter, milk production rate, and degree of metabolism. The multiplication of these uncertainties in the BCF equation is likely to result in a highly uncertain estimate of the BCF. Apparently, the rates for dry matter intake and milk production were averages from the Thomas et al. (1999) study. In view of the fact that Thomas et al. (1999) reported metabolism scores for each of the 12 PCB congeners assessed in the risk assessment, it is unclear why the scores were used to estimate either 50% or 0% metabolism for specific PCB congeners rather than using the actual measured percent metabolism values from the Thomas et al. (1999) study. Similarly, Thomas et al. (1999) apparently also measured percent absorption for each of the 12 PCB congeners, as presented in Figure 4-1; it seems that the actual measured values would be preferred over values estimated from K_{ow}.

- Figure 4-1: It would be useful to identify each of the PCB congeners presented in Figure 4-1.

- Page 4-18, Section 4.2.2.2.3: The BCFs for 2,3,7,8-CDF; 1,2,3,7,8-CDF; and 1,2,3,7,8,9-CDF presented on Table 4-4a are 0.0. No discussion is given in the text regarding these data gaps and how they will be addressed in the risk assessment. However, the footnote on the table indicates that the concentrations of these congeners in the experimental studies were below detection limits, and that these congeners have not been detected in milk and beef surveys. Nevertheless, it is highly improbable that actual bioaccumulation is zero.

- Page 4-19, Section 4.2.2.2.4: The approach for estimating BCFs for Aroclor 1260 seem reasonable. However, more detail should be provided on the variation in the ratios of PCDD/PCDF BCFs for spiked and non-spiked data, and how it was applied to reduce the 2,3,7,8-TCDD spiked BCF to an estimated non-spiked BCF. The statement is made on page 4-20, lines 7-10 that "Results for 1,2,3,7,8-PCDF differed significantly between the two dose groups even though this congener was not spiked. The soil concentrations in both dose groups were near or below the quantitation limits; therefore, a BCF of zero was selected for use in this risk assessment." If the soil concentrations in one of the dose

groups were near the quantitation limit, this suggests that it was detected, and data were available to calculate a BCF. This issue should be clarified.

- Page 4-20, lines 11-22: The extrapolation of poultry meat (adipose tissue) and whole egg BCFs from dairy cow BCFs seems quite a stretch across not only species, media, but also chemicals. Perhaps greater supportive rationale could be provided.

4. Were the exposure assumptions and parameter values appropriate under the evaluation criteria?

- The exposure assumptions and parameter values are considered appropriate and reasonable for this HHRA.

- Page ES-20, line 7: Since ED is such an important factor in this HHRA, a brief explanation of the basis of the 45 year ED should be provided, even in the executive summary.

5. Was the basis for selection of values clearly described and referenced?

- In general, all values are either clearly described and/or referenced.

6. Is the approach used to estimate the RME and CTE appropriate under the evaluation criteria?

- In general, RME and CTE estimates are considered appropriate for this HHRA.

- Page 4-41, Section 4.5.2.3: The authors applied cooking loss factors for both the CTE and RME exposure for poultry and beef, yet in the fish consumption exposure scenario, applied cooking loss was applied only to the CTE. Clarification is needed.

7. Were the uncertainties in assessment adequately characterized and expressed?

- Generally no, for the reasons given below.

- In general, the qualitative uncertainty analysis presented is very incomplete. Not all factors that could impact risk estimates, either over- or under-estimates, are identified and evaluated. For the sources that are identified, an informative discussion of information is presented, but no conclusion is presented as to whether the source of uncertainty is believed to result in either an under-estimation, no affect, or over-estimation of human health risks. In my view, the risk assessor should, if at all possible, use his/her judgment, experience, etc to put forward a position whether risks are under- or over-estimated or not affected by sources of uncertainty.

- Page 6-1, lines 7-9: While I agree with this statement, the uncertainty analysis does not present enough information about the relative contribution of individual sources of uncertainty to support such a conclusionary statement.

- Section 6.1: This section is too brief. It does not identify the sources of uncertainty related to hazard identification (e.g., data) and evaluate the degree that each source either underestimates risk, has not impact on risk estimates, or overestimates risk.

- Page 6-1, Section 6.2: Comments on uncertainty associated with dose-response have been provided in uncertainty sections of other volumes. In general, the section is sorely lacking and provides risk managers little to no useful information on the uncertainties associated with PCB and dioxin/furan toxicity criteria. The discussion on EPA's dioxin cancer reassessment should be removed in its entirety.

- Page 6-2, Section 6.3: No information is provided about the degree to which each source of uncertainty is believed to affect estimated human health risks.

8. Overall, was the approach used to assess risk from consumption of agricultural products and other wild food items reasonable for evaluating the baseline risk?

Considering the reliance on numerous assumptions because of lack of data in this particular risk assessment, the approach employed generally represents as reasonable an estimation of potential health risks associated with COPCs in the floodplain as possible. However, I believe a risk manager will have great difficulty determining how the information should be applied in risk management decision making. The exceptional amount of uncertainty is all the more reason for a much more extensive uncertainty analysis than has been presented. Most of the concerns embodied in my comments are the result of limited data, and, as a result, the application of many upperbound assumptions, which would be expected to over-estimate potential health risks by perhaps several orders of magnitude. It is highly recommended that further studies of agricultural products, both plant and animal, in the flood plain be done to validate some of the assumptions in the HHRA.

Additional Reviewer Comments

- Page ES-10, line 5: The use of the word "consideration" (here and elsewhere) is misleading. In the context of this sentence, "consideration" conveys the idea that a particular exposure scenario is given careful thought, and then a decision made as to it should be assessed or not. The sentence should explicitly state that this exposure scenario WAS assessed in this risk assessment.

- Page ES-17, lines 10-11: This sentence deserves greater explanation. Also, Attachment 2 of Volume 1 should be referenced here.

- Page ES-17, lines 19-20: In this sentence, reference is made to cattle grazing on soil containing 1 mg/kg, whereas the risk assessment assumed 2 mg/kg. Please clarify.

- Page ES-18, lines 8-10: Rationale for this statement should be provided.

- Page ES-18, lines 16-24: Should state whether or not any COPC concentration data have been collected for poultry eggs in the study area.

- ES-21, line 23: A statement of this importance should be accompanied by at least a brief explanation by the risk assessor as to why "all cancer risk estimates were dominated by 2,3,7,8-TCDD TEQ from PCB-126."

- Page 2-8, lines 17-18: Should reference the appropriate table where these data are given.

- In characterizing risks (Section 5), it appears that risk associated with tPCBs and PCB congeners were summed without any adjustment for the contribution to risk from PCB

congeners within tPCBs (i.e., potential double counting) as was done in the fish consumption pathway risk assessment. For consistency, this approach should have also been applied in this risk assessment.

E. Phase II – Integrated Risk Evaluation

Charge Questions and Responses to these Questions:

1. **Were the bases for the toxicity assessment adequately described including the cancer slope factors, reference doses, and calculations of TEQ?** Generally yes. A few comments on TEQ appear in the comments on Attachment C.4 Phase II – Direct Contact HHRA. Comments on toxicity criteria (CSFs, RfDs) generally appear under "other reviewer comments." I have concerns about the TEQ process, which are expressed in comments on Attachment C.4. In general, all toxicity criteria employed are standard EPA criteria.

2. **Did the risk characterization describe the methods and risk summary at an adequate and appropriate level of detail?** Generally yes. Comments on Risk Characterization sections in each of the risk assessments are provided under "other reviewer comments." In particular, comments on the Risk Characterization section of the Phase 2 – Consumption of Fish and Waterfowl Exposure Assessment is noted. The presentation of risk results in graphic and tabular format is particularly well done and clearly conveys the results.

3. **Were the potential risks associated with exposure to a combination of pathways and COPCs (direct contact, fish and waterfowl consumption, and agricultural product consumption) adequately characterized?** Except in Section 7.3 of Volume I (HHRA), no cumulative risk scenarios were assessed. Mention was made of the difficulty and complexity associated with deriving cumulative risk estimates in several places in the various reports. Rationale presented for not deriving these estimates is reasonable and clear. Regarding Section 7.3, the information for the two scenarios presented is clear and reasonable. However, further basic instruction and cautions should be provided for non-technical readers who may attempt to derive cumulative risk estimates based on a personal exposure scenario.

4. **Were the uncertainties associated with both cancer and non-cancer health effects adequately characterized and expressed?** With the exception of the Phase 2 – Consumption of Fish and Waterfowl Risk Assessment, which included quantitative uncertainty analysis, characterization of uncertainty is the most disappointing part of all the risk assessments presented. My comments on uncertainty analysis are quite extensive as related to all of the risk assessments. Perhaps the most extensive comments are presented on the Phase 2 – Consumption of Fish and Waterfowl Risk Assessment report.

F. General

Charge Questions and Responses to these Questions:

Were the EPA toxicity approaches and values (e.g. IRIS and HEAST) used for the COPCs applied appropriately under the evaluation criteria?

Generally yes. All of the toxicity criteria referenced in Table 2-1 of the HHRA are either listed in IRIS or HEAST. Whereas Table 2-1 shows no non-cancer value for alpha-BHC, NCEA has derived a value of 0.0005 mg/kg/day. Also, as noted in several comments (e.g., comment 8.b.xix under "additional reviewer comments" on Phase 2 – Consumption of Fish and Waterfowl Exposure Assessment (Volume IV, Appendix C), the documents should not discuss EPA proposed CSF for 2,3,7,8-TCDD.

Were the calculations of carcinogenic and non-carcinogenic risks performed properly and consistent with EPA guidance?

Yes. Theoretical upperbound cancer risks and hazard indices for non-cancer effects were calculated properly and consistent with EPA guidance.

Were the significant uncertainties inherent in the risk evaluation properly addressed and characterized? If not, please identify those that were not properly addressed or characterized and how they should be addressed in the HHRA.

No. As noted under "Charge Question E" above, characterization of uncertainty is the most disappointing part of all the risk assessments presented. The exception is the uncertainty analysis presented in the Phase 2 – Consumption of Fish and Waterfowl Risk Assessment, which is greatly improved because of the inclusion of a quantitative uncertainty analysis. My comments on uncertainty analysis are quite extensive as related to all of the risk assessments. Perhaps the most extensive comments are presented on the Phase 2 – Consumption of Fish and Waterfowl Risk Assessment report.

In general, the other risk assessments presented only a very cursory and inadequate qualitative uncertainty analysis. Not all factors that could impact risk estimates, either over- or under-estimates, are identified and evaluated. For the sources that are identified, an informative discussion of information is generally presented, but no conclusion is presented as to whether the source of uncertainty is believed to result in either an under-estimation, no affect, or over-estimation of human health risks. In my view, the risk assessor should, if at all possible, use his/her judgment, experience, etc to put forward a position whether risks are under- or over-estimated or not affected by sources of uncertainty.

The general approach for qualitative uncertainty analysis presented in RAGS is recommended.

To the best of the Panel's knowledge, have relevant peer-reviewed studies that support, are directly relevant to, or fail to support any estimate of risk been identified and considered, and has an appropriate methodology been used to reconcile inconsistencies in the scientific data?

For the most part, the HHRA does attempt to incorporate and rely on relevant peer-reviewed scientific literature throughout.

A concern is the fact that the risk assessments made no attempt to reconcile the derived estimates of risk with actual public health studies conducted in the Housatonic River area. Two studies, which have been mentioned in my comments in regards to risk characterization and uncertainty analysis, are the MDPH (1997) exposure assessment study and the ATSDR/MDPH (2002) cancer incidence study. These studies were also discussed in the November 18-20, 2003 public meeting. Considering the overall importance of the findings of the HHRA, I recommend that EPA afford greater emphasis on placing the risk estimates in proper perspective. Certainly, the one way to accomplish this objective is to compare the risks estimated in the HHRA with epidemiology studies in the literature.

Another concern that was also mentioned in my comments is the failure of the HHRA to quantitatively address potential impacts associated with neonatal exposure (transplacental transfer of PCBs, breast milk pathway). The recent scientific literature on developmental effects in children (e.g., Schantz, 2003; Stewart et al., 2003) strongly supports the notion that EPA should assess neonatal risks associated with PCBs in the Housatonic River area. In addition to assessing theoretical neonatal health risks, I recommend that EPA consider a public health study consisting of sampling fetal blood and breast milk. These data, in addition to a risk assessment of neonates, would provide a basis for evaluating the potential impacts of PCBs in the area on neonates.

To the best of the Panel's knowledge, is there other pertinent information available that was not considered in the HHRA? If so, please identify the studies or data that could have been considered, the relevance of such studies or data, and how they could have been used in the HHRA.

None noted that have not already been brought to EPA's attention.

With respect to the conclusions in the HHRA report:

- **Are the conclusions (risk characterization) supported by the information presented in the other sections of the report?**

  Generally yes.

- **Do the conclusions (risk characterization) objectively and reasonably characterize potential current and reasonably foreseeable future risks to human health in the Rest of River area?**

  Generally yes. As noted elsewhere in this report, I have concerns that neonatal risks have not been adequately characterized. As noted, I recommend EPA design and undertake a study to better understand the level of exposure (and theoretical risks) of human neonates to PCBs in the Housatonic River area.

  In addition, the concerns raised in regards to the Schaghticoke Tribe should be addressed through the performance of a quantitative human health risk assessment focused on this group of people and their surroundings.