REVIEW OF THE HUMAN HEALTH RISK ASSESSMENT
GE/HOUSATONIC RIVER SITE
REST OF RIVER
FINAL COMMENTS
December 17, 2003

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GENERAL COMMENTS

Overall, the risk assessment documents provide a thorough evaluation of potential risks for a very complicated site. I commend the authors. My comments are intended to be a source of constructive criticism from an individual who has prepared several assessments for other large Superfund sites as well. Since risk assessment is an evolving, flowing process, there are varying approaches that can be adopted that would be considered reasonable and appropriate. While I may disagree with an approach adopted in this assessment does not necessarily mean that it is wrong or inappropriate.

My biggest criticism of this report is that lack of discussion on uncertainty. Several complicated approaches were used (e.g., spatial weighting and regression analyses to name the two most obvious) and little or no attempt was made to characterize in a meaningful way the extent to which such an approach may have over or underestimated risks. I offer more detailed comments on this issue throughout my review.

RESPONSE TO CHARGE QUESTIONS

Please refer to the Charge for specific, detailed questions. Charge questions are paraphrased here.

A. Phase I – Direct Contact Exposure Screening

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? In addressing this question, consider:

- the general procedures used;
- the SRBCs used for the COPCs: and
- the land use and exposure categories considered and the classification of particular parcels and areas into the categories.

The Phase I assessment uses SRBCs (either calculated or those dictated in the Consent Decree) based on direct exposure to PCBs only. Receptors evaluated include residential, agricultural, and commercial/industrial. Except for the following comments, the receptors and procedures used are acceptable.

It is acceptable to focus the Phase I screening on PCBs only, but the process needs to be sufficiently conservative to ensure that areas where risks could occur are not eliminated. Thus, I recommend that the Phase I screening use primarily upper-bound exposure factors and

assumptions to reduce the possibility of getting a false negative result.

Page 2-3: In the Phase 1 screening process, if the maximum measured concentration exceeded the SRBC, the 95% UCL was calculated and compared to the SRBC for data sets with a sample size of five or greater. I disagree with this approach in a Phase 1 screening. It would be more conservative (and health-protective) to use the maximum measured concentration for comparison to the SRBC, which is done in some cases. Given the small number of measured samples relative to the large area affected, I recommend that this approach be adopted. If the UCL value is used, I recommend using the maximum value for data sets with a sample size of 10 or less.

Within Section 2.5.1 and associated tables, references for many of the exposure parameters used as well as justification as to why a particular value was selected are lacking and should be incorporated.

Table 2-1: I disagree with the use of a target risk level (TRL) of $5x10^{-6}$ (versus $1x10^{-6}$) in the calculation of SRBCs. The reason given for using $5x10^{-6}$ (that there was so much data from such a large area to evaluate that a higher TRL would screen out more areas more quickly) is not acceptable. The more conservative target risk level of $1x10^{-6}$ should be used in the Phase 1 screening for all scenarios and receptor groups.

Tables 2-5 and 2-6: A TRL of $1.6x10^{-6}$ was used for the utility worker, while a TRL of $1.1x10^{-6}$ was used for the groundskeeper. The text needs to justify why different TRLs were used for workers versus residential and recreational receptors, or use a consistent TRL for all receptor groups. I recommend a TRL of $1x10^{-6}$ be used for all receptor groups.

Section 2.6.1.1, page 2-23: The text states that "if the 2 mg/kg benchmark value was exceeded on a high-contact, residentially zoned but undeveloped property, it was retained for analysis in Phase 2." The text needs to clarify which specific EAs that are zoned residential or that could be used for residential purposes in the future were retained. A similar comment applies to agricultural areas.

While the assumption that individuals spend 100% of their recreational time in areas (i.e., FI=1) contaminated at the upper-bound (95% UCL) level is very conservative. I do agree that a Phase 1 assessment should err on the side of conservatism; hence, these values seem reasonable for Phase I (but would be overly conservative for Phase II).

Section 2.5.1.1, page 2-6: I disagree with the exposure frequencies used to calculate SRBCs for high contact residential areas. Residential land use means that individuals live on that property; hence they could potentially be exposed to floodplain soils 7 days a week (versus 5 days a week) for 7 months a year (the number of months the ground is not frozen or covered by snow).

Section 3: It would be helpful if the tables in Section 3 also included information on the size of the parcel or EA under evaluation.

Section 3.2.3: The process used to screen agricultural areas is confusing. My understanding is that agricultural areas were screened based on exposure to PCBs in soil via ingestion and direct contact with soil only (i.e., screening did not include possible consumption of crops affected by site soils). If agricultural areas were eliminated based on direct contact with soil only, I strongly disagree with this approach. If agricultural areas were screened for direct contact only and retained for Phase II analysis, this fact needs to be clarified in the Phase I report. Results of the agricultural exposure analysis (Phase II) show that consumption of some agricultural products

originating from areas with a soil concentration of 2 mg/kg (the SRBC) could result in a risk level as high as $1x10^{-3}$. Since the consumption of agricultural products originating from areas with PCB levels greater than 2 mg/kg could result in elevated risk levels, all areas that are or could be used for agricultural purposes in the future should be retained for Phase II analysis. I recommend including a summary table of EAs that are or could be used for agricultural purposes in the future as well as information on whether each area was eliminated in Phase 1 or retained for further analysis in Phase II.

Section 7: Phase 1 screenings were based primarily on current land uses (and zonings). How future land use conditions were incorporated into or affected the results of the Phase 1 analysis is confusing. Page 7-4, lines 30-33 state that only Reaches 7 & 8 have areas where land use could change and Reaches 5 & 6 have "no properties that could have their screening result changed based on realistic future land use." It appears that all of the EAs listed in Table 7-1 would be retained for further analysis if screenings were based on potential future land uses, yet all of these EAs were eliminated from further analysis based on current land use only. If this interpretation is correct, clarification of why these EAs were eliminated is needed.

B. Phase 2 – Direct Contact Exposure Assessment

Although the Charge questions do not specifically address the selection of COPCs, my comments on Section 2, Hazard Identification, follow.

The Phase II soil/sediment screening process focused on PCBs, PCDDs/PCDFs, and Appendix IX compounds. Screening of chemicals was based on comparisons to EPA Region IX PRGs as well as site-specific and Massachusetts (MDEP) background data. Use of established PRGs is acceptable, but the text needs to clarify why Region IX PRGs were used versus PRGs from other EPA regions or site-specific SRBCs (e.g., PRGs may be more conservative than site-specific SRBCs).

Also, in Phase II, PRGs based on exposure to multiple chemicals (instead of just PCBs) across multiple pathways (versus just direct contact with sediment and soil) would have been more representative and accurate. Had SRBCs been based on exposure to multiple chemicals across multiple pathways, additional chemicals may have been retained as COPCs. Although the affect on risk estimates is likely to small given that dominance of PCBs at the site, the potential to underestimate risks should be discussed in greater detail.

Table 2.2: The text needs to clarify why the PRG for naphthalene was used as a surrogate for four select PAHs. It would have been more conservative to use the PRG for BaP. If the PRG for BaP had been used, measured concentrations of acenapthylene, benzo(ghi)perylene, and 2-methylnapthalene would have exceeded their PRG and would not have been eliminated as COPCs at this point.

Table 2-3: I recommend adding a statement to footnote b stating that samples were shown to be normally distributed; hence the arithmetic mean is reported.

Table 2-4: I disagree with the elimination of aluminum and manganese as COPCs. Six of the seven samples exceeded the PRG for both chemicals. Given the small sample size (n=7) and the high exceedance rate, aluminum and manganese should be retained as COPCs. Since background concentrations were not provided for these two chemicals (since they were eliminated from the process), it is impossible to determine if measured levels exceed site-specific and MDEP background levels. Inclusion of aluminum and manganese as soil COPCs is likely to have

minimal impact on risk estimates, however.

Section 2.5.2.2.3: I disagree with the deletion of chromium as a COPC for soil based on comparison with background concentrations. Mean chromium concentrations in site soils exceed both MDEP and site-specific background levels. Inclusion of chromium as a COPC is likely to have minimal impact on risk estimates, however. I do agree with eliminating the five remaining PAHs since mean site-related concentrations were less than site and MDEP background levels.

Table 2-13 & Section 2.5.3.2.3: I disagree with the deletion of chromium and thallium as COPCs for sediment based on comparison with background concentrations. Mean concentrations in site sediment exceed MDEP and site-specific background levels. Inclusion of chromium and thallium as sediment COPCs is likely to have minimal impact on risk estimates, however.

1. Were exposure scenarios, exposed populations, land use areas, and routes of exposure appropriate, consider the following when addressing this question:
   – Current and reasonably anticipated future land uses, physical conditions, and accessibility;
   – Locations, concentrations, and distribution of COPCs in the sediment, bank soil, and floodplain soil; and
   – Ages of the selected exposed populations.

There is definitely disagreement between GE, EPA, and the public about possible future use of the area. On the one hand, the claim that increased commercial agricultural activity is not likely because it's not financially viable seems reasonable. Conversely, it does seem reasonable that if the floodplain were not contaminated, more non-commercial, small-scale agricultural activity would probably occur. Similarly, on one hand, the RA argues that since 75% of the land is state owned, future land use in those areas won't change. Conversely, just because the land is state owned doesn't mean land use wouldn't vary. New trails and fishing areas could be opened up by the state to encourage higher use of the area, for example (especially if the area was not contaminated). The main issue at stake here is that the RA is supposed to evaluate potential risks under current *and reasonable future land use scenarios*. The RA does seem biased toward little or no change in the future relative to current land use. I recommend that at a minimum, local government planning information be consulted to verify EPA's current position or a broader definition of future land use be adopted (i.e., that the RA acknowledge that in the absence of contamination, more areas may be used for agricultural and recreational purposes in the future than are currently considered).

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

Elimination of the Housatonic River as a source of drinking water now and in the future is appropriate. Although incidental ingestion of and dermal contact with surface water could occur, these pathways were appropriately eliminated by comparison of maximum measured surface water concentrations with site-specific SRBCs.

All other complete exposure pathways were evaluated with the exception of the breast milk pathway. One reason given for not calculating potential exposures to infants from consumption of breast milk was lack of EPA guidance. However, methodologies for quantifying the breast milk pathway are available in EPA's (1988) Hazardous Waste Combustion Guidance. Unless EPA can provide new information as to why methodologies available in other EPA documents and the scientific literature are not appropriate, potential risks from consumption of breast milk should be quantified. While the contribution to overall risk from the breast milk pathway is likely

to be small relative to other pathways, risks for the breast milk pathway should be evaluated to verify their contribution to overall risk.

3. Were the approaches and methods used to calculate and apply exposure point concentrations (EPCs) appropriate?

EPCs were defined as the 95% UCL or maximum value, whichever was lower, which is consistent with EPA guidance and appropriate.

A spatial weighting approach was used to calculate EPCs for Reaches 5 & 6. While I understand the purpose of the spatial weighting approach (to estimate concentrations over a large area where collecting samples from the entire area would be prohibitive), and I don't have a problem with the approach used, the discussion of the methodology used (Section 4.4.4.1, page 4-7 and Attachment 3 of the HHRA) is difficult to follow. Development of EPCs is a critical step in the RA process, since many subsequent calculations (for Reaches 5 & 6 only) rely on the accuracy and reliability of the spatial weighting results. The approach and assumptions used to generate EPC for Reaches 5 & 6 need to be much more transparent and verifiable. Detailed information on the extent that spatial weighting affects EPCs (i.e., would EPCs probably be higher or lower without spatial weighting) should be discussed as well. For example, if spatial weighting hadn't been used, would the authors expect soil concentrations to be higher by a factor of 2 or 10? I recommend the information provided to Panel members by EPA in response to a reviewer's questions be added to the text along with the example calculations provided.

The Panel was provided with the measured and interpolated data for five EAs selected by chance, so that Panel members could better evaluate the influence of IDW on EPCs. Dr. Ryan made an interesting observation that the maximum interpolated value exceeds the maximum measured value for EA32, which indicates that some of the interpolated data used to calculate the EPC for EA32 were taken from areas outside of EA32. While this approach seems odds, I'm not sure that it is wrong or significant. I had assumed that only values from within an EA would be used to calculate an EPC for that EA. If the number of interpolated values taken from outside an EA is small relative to the total number of interpolated values used to calculate the EPC for that EA, the effect on risk estimates would be small. I suggest EPA elaborate on why interpolated values were taken from outside an EA to clarify this issue.

I was surprised by the extremely low percentage of measured to extrapolated samples. For each of the five EAs listed in the memo from EPA, measured data comprised <1% to 1% of the total values (measured and interpolated) used to calculate the EPC for that parcel. The RA just doesn't give any indication of this fact and should. I recommend that EPA note this in the report and comment on the statistical reliability of that ratio. For example, when spatial weighting approaches are applied to other, large sites, is the ratio of measured to interpolated data similar, and is the IDW approach considered statistically reliable when the number of interpolated values is considerably larger than the number of measured values?

Finally, EPA calculated 95% UCLs for the measured data only and for interpolated data (for the five EAs mentioned above). For EA 40, the maximum measured value was used as the EPC, so a 95% UCL was not calculated. For the other four EAs, the 95% UCLs for measured and interpolated data varied by less than a factor of two. This suggests that the effect of spatial weighting on the calculation of 95% UCLs is probably small (within a factor of two) for most EAs. The EPC could be over- or underestimated, as EPA noted, depending on the sampling strategy used for different EAs. Clarification of this topic in the RA would be useful.

Since PCDD/PCDF and PCB congeners were only directly measured in 10% of site samples, regression analyses were used to estimate congener concentrations in the remaining samples. While I don't have a problem with the application of regression analyses *per se*, I found the discussion difficult to follow. Hence, it was difficult for me to evaluate the accuracy of the methods used. One example is the fact that the selection of regression models was based on the p value and sample size (when more than one regression equation had a p-value < 0.01). Why r^2 values were not considered is unclear. If a particular equation had a highly significant p-value but a relatively low r^2 value, it would have been selected under the methods used in the RA, but that equation may not be adequately predictive. A discussion of corresponding r^2 values and better justification for the equation selection process needs to be included.

Another potential problem associated with the use of regression equations is that the congener profiles were based on data collected in Reaches 5 & 6 only but applied to all downstream areas. In other words, the current approach does not account for the fact that congener profiles downstream could be different than those observed in Reaches 5 & 6. There needs to be some discussion on how representative the data from Reaches 5 & 6 are to the rest of the site and what impact the assumption of no change in the congener profiles downstream may have on risk estimates. In other words, what is the likelihood that congener profiles could be substantially different downstream than those observed in Reaches 5 & 6? If congener profiles might be different, would the assumption of no change in congener profiles likely to over- or underestimate EPCs?

In the calculation of EPCs for recreational activities, the authors apply a use-weighting factor to account for accessibility (which lowered EPCs), since the spatial weighting technique cannot account for accessibility. While this approach seems reasonable, the application of use weighting factors is very arbitrary and does not seem to accomplish its goal. I recommend that accessibility be accounted for by adjusting exposure frequencies or FI. In addition to the application of a use weighting factor, exposure frequency was also adjusted to represent the amount of time an individual spends in a given area performing a given activity. On the surface, this could appear to be two separate methods for accounting for the amount of time an individual would spend in a given area performing a given activity. Regardless of whether the authors decide to apply a use weighting factor or to adjust EF or FI, the method used to calculate EPCs for recreational exposures needs to be more fully explained to ensure that no "double counting" occurred (i.e., that EPCs were not lowered twice for the same reason).

Section 5.5: It would be useful to have a summary table of the EPCs used for each EA evaluated. I know these data are presented in Tables 5-2 through Tables 5-398, in Section 5 figures (in Appendix B), and in the text on a EA by EA basis, but it would be useful to have a table listing all of the EPCs for soil and sediment by EA, so the reader can easily see the variations in soil and sediment concentrations with location. Thus, I recommend adding the soil and sediment EPCs used to calculate HIs and risks to Table 5-1.

4. Were the values used to represent the exposure and absorption parameters used in the direct-contact exposure assessment (specifically exposure durations, exposure frequencies, use factors, soil ingestion rates, dermal contact factors, and oral and dermal absorption rates) appropriate?

The assumption that all exposures occur randomly across a tax parcel, EA, or subarea is troubling. I do agree that this approach is a logical starting point and is appropriate for areas where receptors are truly likely to traverse most or all of an exposure area (e.g., smaller areas less than five acres in size) or for areas where the EPCs do not differ significantly. However,

exposures could be underestimated for areas where it is possible that an individual may restrict his/her activities to a smaller area where concentrations are statistically higher than the EPC for the entire EA. My attached Table 1 shows the size of areas evaluated as well as the maximum and 95% UCL concentrations for all tax parcels whose designated land use is recreational and whose size is more than five acres. Table 1 shows that some of the tax parcels are quite large and that within many of the parcels, PCB concentrations are variable (as exemplified by the large difference between the maximum and 95% UCL concentrations). For these areas, the assumption of random exposure across an area may not be appropriate. For these areas, exposures to smaller subareas where PCBs have accumulated to a greater extent should be quantified (assuming that these areas are accessible for recreational use now or in the future).

Section 4.5.3.1.2, page 4-33: Exposure duration values were based on how long an individual lived at one address versus lived in the Housatonic River Area. Using the former could result in an underestimation of risks to an individual who lived at different locations but within the Housatonic River Area. RME and CTE exposures could be underestimated by a factor of two. I recommend basing ED values on duration of residency versus length lived at one residence, although this change will have minor impact on risk estimates.

Section 4.5.3.3, page 4-38. I disagree with the assumption that ATV and mountain/dirt bike users are limited to the older child receptor, since adults frequently participate in this type of activity. I do agree, however, the risks to the older child would be higher than those for the adult, so calculation of the adult receptor is not required. I recommend rewriting the text to clarify these issues.

Page 4-42, lines 7-11: I believe the older child is just as likely to go canoeing or boating at the same frequency as an adult. I suggest adjusting exposure frequency values accordingly.

Section 4.5.3.10.3, page 4-53, lines 21-24. Soil ingestion rates of 100 mg/day for the RME scenario and 50 mg/day for CTE scenario were used for the groundskeeper (individuals who mow lawns). These values are the same as those used for adult residential receptors. I believe that the soil ingestion rates for the groundskeeper should be consistent with those used for other contact-intensive activities (e.g., farming and riding ATVs/mountain bikes), since mowing can stir up a large amount of dust.

Section 4.5.3.9.1, page 4-51, lines 15-19: I disagree with the EF used for agricultural receptors. It is likely that farmers would work in their fields many more days than just at planting and harvesting time. EF certainly needs to be modified to a much higher value (five days a week?)

I agree with the use of an oral absorption factor of 100% for PCBs.

The specific EPA document that recommends a dermal absorption rate of 14% for PCBs need to be cited (along with the Wester et al., 1993 study).

The text notes that the EF of 60 days/year was based on professional judgment that an individual would fish two times a week over a seven-month period. Survey data presented in Maine, Connelly, and ChemRisk studies seem to suggest an EF of 30-40 days/year. I recommend an EF of 30-40 days per year be used, since it is based on empirical data versus professional judgment.

There was much discussion on soil ingestion rates during Panel deliberations. The authors did use EPA standard default values, which is appropriate in an assessment of this type. If the authors review newer, peer reviewed studies and choose to lower the rate that would be

acceptable as well. Since I have not reviewed the Staneck and Calabrese (1997, 2000) articles, I cannot comment on their accuracy.

It is appropriate to assume that 100% of soil ingested by recreational receptors comes from the floodplain (i.e., FI=1) if EF and ED accurately reflect the amount of time spent in the floodplain (versus outside of the floodplain).

Section 4.5.3.1, page 4-32, lines 11-12: As written, current language makes it sounds as if people over the age of 45 weren't considered in the RA. Suggest changing the text to state an exposure duration of 45 years was used.

5. Is the approach used to estimate a Reasonable Maximum Exposure (RME) and a Central Tendency Exposure (CTE) appropriate?

Yes, overall, the approach is reasonable and consistent with EPA guidance. There is some question about the approach used to calculate RME EPCs, with respect to application of the bootstrapping technique. While I agree that bootstrapping is commonly used, it may not be as robust (and possibly conservative) as the t-statistic and Land's method. Considering that 78 out of 90 EPCs for Reaches 5&6 (zero out of 30 for Reaches 7&8), were derived using bootstrapping, the authors need to discuss the influence bootstrapping may have on EPC calculations. Specifically, is bootstrapping expected to over- or underestimate EPCs and by what factor? One way to provide a perspective on this issue would be to use to calculate 95% UCLs assuming the data are normally and lognormally distributed (using the t-statistic and Land's method), and the comparing these UCLs to the value derived using bootstrapping.

6. Were the uncertainties adequately characterized and expressed?

Uncertainties were evaluated (for the most part) qualitatively (versus applying a formal quantitative analysis, such as a Monte Carlo simulation). The Uncertainty Analysis provides limited information as to whether a source of uncertainty is likely to over- or underestimate risks. I suggest the authors include a summary table including each and every source of uncertainty associated with the Phase 1 assessment, whether the source is likely to over- or underestimate risks, and quantify (where possible) the extent to which the source is likely to over- or underestimate risk. Then, major sources of uncertainty should be discussed in more detail as well. I note three issues below that warrant further evaluation/discussion. This approach should be repeated for all subsequent analysis (Phase II, Fish and Wildlife, Agricultural).

Random Exposure Within an Exposure Area.
The issue of assuming random exposure within a parcel is troublesome. The text admits that if individuals preferentially occupy one part of a parcel over another, exposures could be higher or lower than estimated. For the larger parcels with varying PCB concentrations, the potential to underestimate risks seems large enough that a more quantitative reporting of to what extent risks could be underestimated is warranted (or recalculation of potential exposures in smaller areas).

Current Versus Future Exposures
Section 7.2.2.5 states that only properties currently used for residential or agricultural purposes were evaluated. Thus, risks to potential future receptors who could reasonably use specific areas for residential or agricultural purposes in the future were not quantified. This approach is inconsistent with EPA policy and may underestimate risks if these areas experience different land uses in the future. I recommend that possible exposures to individuals who could live on one of the "several locations that are not currently developed but could be used for housing in the future"

be quantified. The same applies for those areas that are not currently used for agricultural purposes but could be in the future.

Uncertainty Associated with the Toxicity Assessment
Potential cancer risks were appropriately quantified using the approved dioxin cancer slope factor (CSF) of $1.5x10^{5}$ $(mg/kg\text{-}day)^{-1}$. Since EPA is reevaluating the potential cancer effects of dioxin-like compounds and may revise the CSF for dioxin, I recommend that cancer effects be quantified (versus just stating that risks would increase by a factor of six) using the revised EPA CSF of $1x10^{6}$ $(mg/kg\text{-}day)^{-1}$ for all pathways and receptors and results presented in the Uncertainty Analysis.

The quantification of potential noncancer effects from exposure to dioxin-like compounds remains controversial. While EPA has not formally established an RfD for dioxin-like compounds, there is a growing body of literature suggesting that dioxin-like compounds may cause noncancer effects in humans. At a minimum, I recommend that potential noncancer effects be discussed in the toxicity assessment. Although the "informal" RfD of 1.0 pg/kg-day for dioxin has not been approved by EPA, it has been used in other EPA assessments, thus giving use of the informal value some legitimacy. If the authors choose to provide a perspective on potential noncancer effects from exposure to dioxin-like compounds, the results should be included as part of the Uncertainty Analysis, not the formal RA.

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

Yes, except for the comments made previously.

C. Phase 2 - Fish and Waterfowl Exposure Assessment

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

The comment about the application of bootstrapping techniques to calculate EPCs made previously applies to the fish data as well.

Section 2.2.2.3 describes how total cancer risks were lowered slightly to account for the amount of dioxin-like PCBs in the CSF for PCBs. The text further states (page 2-11, lines 5-6, for example) that uncertainty associated with calculation of the expected TEQ could over- or underestimate cancer risk from exposure to PCBs. Since 1) the authors admit that there are no reports in the open literature (or EPA documents presumably) that address methods for avoiding double counting, and 2) Dr. Keenan presented a compelling argument that the CSF already accounts for the presence of dioxin-like PCBs, I recommend that this approach be eliminated. If the authors do keep the adjustment in the report, they need to quantify to what extent risks may be over- or underestimated using this approach (versus just qualitatively discussing this point). See additional comments on this topic made under "E. Phase II – Integrated Risk Evaluation."

For Reaches 5 & 6, fish data used were "skinned and trimmed fillets." Use of these data may underestimate risks for individuals who cook and consume fish with the skin on. Did any of the three studies cited in Table 4-12 provide insight into whether individuals prepare and consume fish with the skin on? If not, the cooking loss data for PCBs presented in Tables 4-18 and 4-19 for skin-on and skin-off fillets might provide a basis for estimating the concentration of PCBs in fish with skin on. Table 4-19 shows that loss of PCBs during cooking is higher for skin-on fillets

versus skin-off fillets, which suggests that the concentration of PCBs in fish consumed by anglers would be higher than that measured in "skinned and trimmed fillets."

It is appropriate to combine the four fish species into two discernable groups, to calculate one EPC for fish, and to use those EPCs to calculate doses for a general angler. This approach, however, does not account for the angler who may consume only one group of fish (bass/bullhead or sunfish/perch). Since the concentrations in these two groups of fish were statistically different, risks to individuals who consume fish from one group only could be over- or underestimated. I suggest calculating risks to individuals who may consume only one group of fish to resolve this issue.

Furthermore, the analysis does not take into account the fact that some individuals may fish repeatedly in the same small area (i.e., a favorite spot), particularly when evaluating data for Reaches 5&6, which cover a relatively large area. The text should discuss how fish concentrations vary within EAs and the potential to underestimate risks if individuals fish in one area versus randomly within and EA (as was assumed). I would prefer to see actual quantification of risks to individuals who consume fish from one area only, especially for areas within Reaches 5 & 6.

To account for the fact that some of the waterfowl that could be consumed from the study area are migratory, the EPC was modified. I recommend that FI be adjusted versus the EPC as a more technically-correct approach. FI should be set equal to the percentage of waterfowl in the area that are resident (non-migratory) birds.

2. Were the exposure assumptions and parameters used in both the assessments of fish and waterfowl consumption appropriate?
3. Was the basis for the selection of point estimate RME and CTE exposure parameter values appropriate, and were they clearly described and referenced?

Tables 4-42 and 4-43: It is not clear why different consumption rates (g/day) were used to calculate noncancer and cancer doses from consumption of waterfowl. Table 4-42 list an adult RME consumption rate of 5 g/day, while Table 4-43 lists an adult RME consumption rate of 20 g/day. CTE consumption rates for adults and children are consistent between the two tables. Noncancer doses reported in Table 4-51 are not consistent with the ingestion rates of 20 and 10 g/day listed in Table 4-43, so I suspect the RME consumption rates listed in Table 4-43 are erroneous.

The authors appropriately elected to use fish ingestion rates from the Maine Angler Survey. Given the information presented by Ms. Ebert, however, it seems clear that the ingestion rates selected by EPA were incorrect (too high). The consumption rate of 32 g/day used in the RA was based on a sensitivity analysis. Using the empirical data reported by Ebert et al., a 95th percentile fish ingestion rate of 12 to 16 g/day would be more appropriate.

I agree that assuming an individual consumes one fish meal a day from the Housatonic River, 365 days a year, for 60 years seems overly conservative. On page 4-31, lines 6-8, the report notes that according to the MDPH survey, 32% of residents claimed to consume freshwater fish one to four times a month, 26% one to two times a week, and 1% at least three times a week. This survey information should be used to adopt an upper-bound number of meals likely to be consumed by adults (e.g., an EF of two to three fish meals per week from the Housatonic River seems more reasonable).

The waterfowl scenario assumes individuals consume only breast tissue with skin on. The RA needs to clarify that this approach may underestimate risk for individuals who may consume other parts of the bird (e.g., legs and other dark meat).

A summary table on the concentration of PCBs in ducks taken from a reference area was provided to the Panel and PCB levels in reference ducks varied by three orders of magnitude. It would be prudent to statistically evaluate if PCB levels in waterfowl taken from the affected area are statistically higher than PCB levels in ducks taken from the reference area.

4. Were the probabilistic approaches used clearly described, and were they appropriate?
5. Were the distributions used in the probabilistic assessments clearly described, and were they appropriate?
6. Were the uncertainties in the data and models adequately characterized and expressed?

Since probabilistic risk assessment (PRA) is not my area of expertise, I have only a few comments to make here. I leave the detailed review of the PRA to my colleagues.

I strongly disagree with using point estimates for fish concentrations in the PRA. Fish concentrations were allowed to vary from the mean to the 95% UCL value only instead of using the entire range of measured data. What is the value of having so much measured data if it isn't used in the PRA? Regardless of the methodology dictated in EPA's Uncertainty Guidance, I recommend that fish concentrations be allowed to vary over the range of measured data in the PRA.

Secondly, I concede that it EPA guidance recommends not including uncertainty associated with the toxicity constants in the PRA, and I understand the logic. On the one hand, the uncertainty associated with the toxicity values can overwhelm uncertainty associated with other parameters. On the other hand, there IS a great deal of uncertainty associated with the toxicity data, and that source of uncertainty should be accounted for in some way. I recommend performing the PRA both ways (one run with toxicity constant held steady, another run with toxicity constants allowed to vary over a reasonable range).

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

I suggest the authors include a table summarizing each and every source of uncertainty associated with the Phase II assessment, whether the source is likely to over- or underestimate risks, and quantify (where possible) the extent to which the source is likely to over- or underestimate risk. Then, major sources of uncertainty should be discussed in more detail as well. I note three issues below that warrant further evaluation/discussion.

Section 7 does a reasonably good job of qualitatively discussing sources of uncertainty and variability associated with the point estimates. It fails, however, to aggregate these sources to provide the reader with a revised risk estimate reflecting uncertainty and variability. For example, if individuals consumed skin-on fillets (point estimates for fish taken from the Massachusetts portion of the study area are based on skin-off fillets), risks could be increased by a factor of two to four. PCDDs/PCDFs were not analyzed for in Connecticut fish samples. The addition of PCDD/PCDFs could increase risk for CT consumers by a factor of two. If an individual ate one species of fish (versus a mixture as assumed in RA), risks could increased by another factor of 2.5. If an individual consumed fish from one location (versus random access

within an exposure area), risks could increase or decrease, depending on COPC concentrations. (This point needs to be discussed quantitatively in Section 7.) I recommend that at least two additional scenarios be discussed in Section 7, one worst-case and one best case. For example, what if an individual ate skin-on fillets only, ate the most highly contaminated species only, and consumed fish from one, highly contaminated area only, how much would risk estimates change? Conversely, what if an individual ate skin-off fillets only, consumed the least contaminated species only, and consumed fish from the least contaminated area only, how much would risk estimates change? This type of cumulative analysis would be more useful than simply listing all the types of uncertainty and their effect on risk estimates.

Table 7-1, page 7-3: Data presented in Table 7-1 would be much more meaningful if actual concentration numbers were presented as well as percent change in the EPC.

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?

Yes, with the exception of other comments made.

General Comments of the Fish and Waterfowl Exposure Assessment

This volume is the most organized and well-written. Presentation is clear, thorough, and easy to comprehend.

The issue of potential risks to individuals who may participate in subsistence fishing needs to be evaluated in more detail. There is clearly contention among members of the public that some local Indians do participate in subsistence fishing and risks to these individuals have not be quantified. EPA needs to provide evidence supporting a claim of no subsistence fishing or quantify risks to these individuals.

Just because waterfowl were not sampled directly in CT does not mean that risks for consumption of waterfowl by CT residents can't and shouldn't be calculated. In the absence of actual site-specific data, the HHRA should adopt a conservative method for quantitatively evaluating human exposure to waterfowl by CT residents. The most conservative approach would be to assume that waterfowl in CT are contaminated at the same level as waterfowl in MA. Or since tPCB concentrations decrease with increasing distance from the source, tPCB concentrations in CT waterfowl could be adjusted to reflect this decline and risks quantified.

Section 3.2.4.2, page 3-10, lines 3-14. It is not clear how the expected TEQ concentration was calculated using the data listed in Table 3-3. Table 3-3 provides a listing of the data and results but does not provide a sample calculation (as the text states). Please provide a sample calculation. Also, line 10 states that expected the TEQ concentration was subtracted from the predicted TEQ concentration. Please clarify which value in Table 3-3 is the predicted value?

It would be useful to provide information on the number of waterfowl that actually reside in the affected area, so that consumption rates and exposure frequencies assumed in the HHRA can be balanced against reasonable hunting practices.

Sections 4.3.4.1 and 4.3.4.2 are redundant and should be combined.

Fig 5-1: The incorrect figure is included. Fig 5-1 lists cancer risks associated with consumption of agricultural produce, not fish consumption.

Page 8-2, lines 27-30 & Table 8-1: The text states that data presented in Table 8-1 show a steady decline in cancer risk estimates from Reaches 5 & 6 downstream to Lakes Lillinonah and Zoar. While this is true, it would be helpful if the text clarified to what extent the reduction in cancer risk could be attributable to the fact the only bass and trout were sampled in lower reaches (versus just declining concentrations in general as one progresses further away from the source).

D. Phase II – Agricultural Exposures

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

I strongly disagree with using the "assumed" soil concentrations of 2 mg/kg and 0.5 mg/kg in lieu of measured or modeled values for non-parcel specific exposure scenarios. One reason given for adopting this approach was that it illustrates how risks would change with decreasing PCB concentrations. "For example, 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 for corn silage was contaminated with 5 mg/kg and the remaining 90% of the cultivated land was not contaminated with tPCBs." While this is true, it is not representative of current site exposures, and associated results are not very meaningful. Furthermore, the approach represents a significant departure from EPA protocols. I strongly urge that the final HHRA use site-specific data to calculate a range of actual soil concentrations versus hypothetical values. While this could be done without calculating parcel-specific risk estimates, measured data from areas where agricultural practices do or could occur should be used.

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

Soil-to-grass transfer factors for tPCBs and dioxin-like PCB congeners were mean concentration ratios based on measured, site-specific data (n = 10). Soil-to-corn transfer factors for tPCBs were mean concentration ratios based on measured, site-specific data (n = 5). Soil-to-corn transfer factors for dioxin-like PCB congeners were based on soil-to-grass ratios.

For exposed vegetables, the soil-to-plant transfer factor was defined as the maximum transfer factor for corn. The maximum value reported in Table 4-6 is $6x10^{-3}$, while the transfer coefficient for exposed vegetables listed in Table 4-5 is $6x10^{-4}$. Table 4-5 appears to contain a typo, which should be corrected.

For root vegetables, the higher of the transfer factors reported for beets and turnips based on site-specific data for Aroclor 1260 was used. This value is likely to underestimate exposures since beets and turnips were washed before analysis. Beet and turnip values reported for Aroclor 1248 are 75 and 275 times higher than values for Aroclor 1260, while values for total PCBs are similar to values for Aroclor 1260.

For exposed fruits, the soil-to-plant transfer factor was set equal to the transfer factor for exposed vegetables ($6x10^{-4}$). Thus, the discrepancy noted above (with respect to the correct exponent) for exposed vegetables applies here.

While weak correlations between site-specific plant and soil concentrations were blamed on several factors (e.g., influence of background levels and contaminant transfer from one area to another), the fact that several plant species were washed and scrubbed before analysis, which

could potentially remove contamination, and small sample size were minimized. Data from washed and scrubbed plants can not be used to reliably estimate plant concentration factors.

Given the uncertainty associated with using site-specific data (e.g., some samples were scrubbed before analysis, small sample size), I strongly recommend that more site-specific data be collected to provide more accurate biotransfer factors for plants and animals. In the absence of such data and in the presence of such high levels of uncertainty, I must recommend that the maximum (versus mean or best estimate values) be used, at least for the RME scenario.

Given the uncertainty associated with the site-specific data (and resulting transfer factors), I strongly recommend that the authors gather information on soil-to-plant transfer factors available in the literature or those generated from predictive equations available in the literature and compare the range of literature and predicted values with site-specific values. My guess is that this comparison will show that the site-specific values are conservative and will lend credibility to the risk calculations.

I agree with Mr. Washburn's assessment that given the uncertainty and limitations associated with the site-specific data available, the regression analyses used to estimate congener-specific plant concentrations for PCBs are highly dubious and unreliable. Therefore, risks for the agricultural scenario should be limited to exposure to total PCBs only. The lack of congener-specific biotransfer data for PCBs precludes a reliable calculation of congener-specific uptake by plants and animals.

Section 4.3.3.1: Since PCDDs/PCDFs were not detected in the 10 grass samples analyzed from the site (despite elevated detection limits), it was assumed that "PCDD/PCDF concentrations are likely to be small contributors to TEQ concentrations compared with PCB concentrations." The small sample size and relative high detection limits do not warrant exclusion of PCDDs and PCDFs from quantitative calculation when literature values are available. This approach is likely to underestimate risks associated with exposure to ingestion of cow beef and milk. Again, before relying solely on weak site data, I suggest a review of the literature (e.g., the Dioxin Reassessment documents). Unless additional site-specific data are gathered (as recommended), literature values may need to be adopted or incorporated, since site values are tenuous as best.

Section 4.4.4, page 4-34, lines 1-6: Predicted home garden vegetable concentrations are based on an assumed soil concentration of 2 mg/kg, the residential soil cleanup level for this site. The HHRA is supposed to evaluate potential risks to individuals under current and future scenarios in the absence of remediation. Therefore, using the soil cleanup level as the basis for baseline risk assessment calculations is inappropriate.

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

Section 4 provides a well-researched, detailed summary of the difficulties and uncertainties associated with deriving BCFs for animal tissues.

A BCF for Aroclor 1254 of 3.6 was adopted as the BCF for tPCBs. EPA (1994) reported that BCFs for PCBs in beef and dairy cattle ranged from 2.1 to 5.9, with most values reported for Aroclor 1254. While use of the slightly higher BCF (e.g., 5.9) is recommend, it would not substantially alter risk estimates.

There are little or no data on the transfer of dioxin-like PCBs to animal products. BCFs for

dioxin-like PCBs in beef and dairy cattle were (after reviewing data available in the literature) estimated using the predictive equation presented on the top of page 4-18. While I do not have a problem with using predictive equations to estimate biotransfer factors in the absence of measured data, results obtained for the PCB congeners do not seem defensible. The BCF used for PCB 126 is 10 times higher than the BCF used for other PCB congeners. As a result, PCB 126 accounts for 70-90% of total risk. This may be due (as Mr. Washburn noted), to the lack of reliable fate and transport data for the individual PCB congeners. Regardless, given the uncertainty associated with deriving reliable BCFs for PCB congeners, it seems more prudent to focus risk calculations for the agricultural scenario on total PCBs only and PCDDs/PCDFs but not dioxin-like PCB congeners.

Mammalian BCFs for PCDDs and PCDFs were the mean of three studies whose results are reported in Table 4-4c. Results of these three studies are in good agreement and use of the maximum versus mean value (while recommended) will not substantially alter risk estimates.

The text states that non-commercial beef and dairy cattle are likely to graze more and have a higher soil ingestion rate relative to commercially-raised cattle. Table 4-3 indicates that the percent soil in the diet for home-raised and commercial beef cattle to be identical (2%), which seems contradictory to the previous statement. Minor point.

4. Were the exposure assumptions and parameter values appropriate?
5. Was the basis for selection of values clearly described and referenced?

Yes, except for the following comments.

Page 4-37, line 22: Use of 75^{th} percentile (versus 90^{th} or 95^{th} percentile) consumption rates for RME scenario is not consistent with EPA guidance. Upper-bound ingestion values should be used for the RME scenario.

Use of an FI=1 is overly conservative. It is not likely that 100% of the fruits and vegetables consumed by residents would originate from the study area. I recommend that FI be adjusted to account for seasonal versus year-round consumption rather than modifying ingestion rates.

Table 4-3 shows that FI was set to 1 for agricultural animals (i.e., "100% of the cultivated and grazing areas are within the 1-ppm isopleth.") This assumption applies to both commercial and non-commercial farmers. While this number seems high to me, I did not find evidence in the document to dispute that assumption, nor did I find evidence to support it. Section 4.2.2.1 needs to provide more information about the reasonable availability of cultivated and grazing areas within the affected area both now and in the future (in the absence of remediation) to clarify this point.

Table 4-8: F_{GI}, fraction absorbed in the GI tract is listed in Table 4-8 but not in the equation on page 4-35, nor is it discussed in the text.

Table 4-8: No loss during cooking is assumed for the RME scenario but a cooking loss factor is applied to the CTE scenario, which seems reasonable.

Given the small number of fiddlehead ferns analyzed, I recommend that the maximum measured concentration (versus site-specific mean) be used to calculate risks.

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

No, since hypothetical versus site-specific soil concentrations were used to model exposures. Otherwise, the approach used is appropriate with the exception of the other comments made.

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

Given the high degree of uncertainty associated with the calculation of risks from ingestion of home grown produce and agricultural products, I strongly recommend that 1) a formal, quantitative uncertainty analysis be performed for the agricultural analysis, and 2) that site-specific vegetable/plant, milk, and beef tissues be analyzed if possible to yield more reliable BCFs.

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?

I strongly recommend that more site-specific data on the biotransfer of PCBs to plants and agricultural animals be collected to reduce uncertainties in the agricultural RA. I agree that given the limited site-specific data available, risks associated with agricultural pathways are speculative. Even if there are no beef or dairy cows grazing on floodplain soils, an experiment could be done where site soil was fed to cows and site-specific, BCFs calculated. Similarly, various agricultural and home-grown crops could be grown in site soils, and BCFs quantified.

Instead of summarizing risks in Section 5 as "exceeded or within EPA acceptable range," listing the actual risk level would be much more informative. Classifying risks as within the acceptable range means that they can vary from $1x10^{-6}$ to $1x10^{-4}$. Risks for consumption of commercial and farm-raised animal products (milk, beef, poultry, and eggs) were at the high end of EPA's acceptable risk range ($4x10^{-4}$ for commercial milk to $8x10^{-4}$ for home-raised poultry) or exceeded the acceptable range ($1x10^{-3}$ to $6x10^{-3}$) for the remaining agricultural pathways. All of these risk levels are associated with a soil concentration of 2 mg/kg.

E. Phase II – Integrated Risk Evaluation

1. Were the bases for the toxicity assessment adequately described including the cancer slope factors, reference doses, and calculations of TEQ?

Toxicity constants used in the risk assessment were adequately described with the exception of the proposed (new) reference dose and cancer slope factor for dioxin-like compounds. This issue has been discussed earlier in these comments. Discussion of the potential noncancer effects of dioxin-like compounds was sparse, probably since the authors chose not to quantify noncancer effects from dioxin-like compounds. Even if noncancer effects from exposure to dioxin-like compounds are not quantified, information on possible noncancer health effects should included. Furthermore, some discussion of mechanism of action and target endpoints for PCBs and PCDDs/PCDFs should be included. Even if noncancer risks for PCDDs/PCDFs were quantified, the mechanism of action and target endpoints for PCDDs/PCDFs may be sufficiently different than that for PCBs, that HQs for these two groups of chemicals probably shouldn't be summed.

Calculation of TEQs is an acceptable method of integrating risks associated with exposure to a mixture of PCDDs, PCDFs, and PCBs that have dioxin-like properties. The TEFs used to calculate TEQs were appropriate and consistent with the current literature. The appropriateness of calculating "excess" PCB TEQ concentrations was confusing, hard to follow, and may not be technically accurate. Section 2.2.2.3 describes how total cancer risks were lowered slightly to

account for the amount of dioxin-like PCBs in the CSF for PCBs. Dr. Keenan presented a compelling argument contradicting the accuracy of this approach. EPA needs to clearly refute Dr. Keenan's arguments or eliminate the adjustment for excess PCBs. I vote for eliminating the calculation, as incorporating this adjustment seems to add a lot of confusion to the process for something that (I'm guessing) has a minimal effect on risk estimates. If the adjustment is left in, it would be helpful if this section also included information on how much risks would change if this adjustment had not been used. Furthermore, page 2-11, lines 5-6 states that uncertainty associated with calculation of the expected TEQ could over- or underestimate cancer risk from exposure to PCBs. I recommend including here a quantitative estimate of the extent to which risks could be over- or underestimated using this method.

2. Did the risk characterization describe the methods and risk summary at an adequate and appropriate level of detail?

No. The risk characterization section of several volumes of the report was frustratingly vague. Instead of summarizing risks as "exceeded, were within, or were below EPA's acceptable range," listing the actual risk level would be much more informative.

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?

No, there was virtually no calculation of cumulative risks (summing of risks across all pathways for a given receptor group), and I strongly disagree with this omission. The only two aggregate risks calculated are an angler and hunter who may have contact with contaminated soil while fishing or hunting, and who eat what they catch. Risk to individuals who could who live in the floodplain, participate in recreational activities in floodplain soils, and consume fish and/or agricultural products taken from affected areas are not quantified. Granted, risks from fish ingestion and consumption of agricultural products are substantially higher than risks from other pathways, it is still appropriate and beneficial to sum risk across pathways so that 1) the contribution from all pathways can be evaluated and 2) a total (cumulative) is risk quantified.

I do strongly agree, however, the background risks from exposure to PCBs should NOT be added to site risks. The purpose of the RA is to evaluate risks from exposure to site-related contamination. Because background risks are typically much higher than site risks, adding in background levels dwarfs site risks to the point that it makes it extremely difficult to determine where action within the affected area needs to be taken (i.e., where existing site levels pose a threat to human health).

4. Were the uncertainties associated with both cancer and non-cancer health effects adequately characterized and expressed?

No, the Toxicity Assessment needs to describe in much greater detail the uncertainties associated with the cancer and noncancer toxicity constants used in the RA.

F. General

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

Yes, the toxicity data were applied appropriately.

2. Were the important assumptions for estimation of dose (i.e., toxicity and exposure) and risk identified?

Yes, with exceptions noted in responses to other comments.

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

Yes, the calculation of dose and risk were performed correctly and are consistent with EPA guidance.

4. 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.

One major data gap is the lack of a formal, quantitative uncertainty analysis for the agricultural analysis. Many of the exposure parameters, particularly chemical-specific transfer factors, are highly uncertain. Performing a quantitative uncertainty analysis for the agricultural analysis would be useful.

5. 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?

The recent publication by Simon on soil ingestion rates should be considered as well as the recent study by Kimbrough reporting the epidemiological effects of worker exposure to PCBs.

6. 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.

Potential risks to infants from consuming breast milk were not quantified. Guidance for performing this type of assessment can be found in the following references. I am sure other sources are available as well.

U.S. Environmental Protection Agency, *Guidance for Performing Screening Level Risk Analyses at Combustion Facilities Burning Hazardous Waste*, Office of Emergency and Remedial Response, Office of Solid Waste, April, October, and December, 1994.

Smith, A.H., 1987. Infant Exposure Assessment for Mother's Milk Dioxins and Furans Derived from Waste Incinerator Emissions, Risk Analysis, 7:347.

The serum data available from two MDEP studies should be incorporated into the risk assessment. First, the authors need to report if levels measured in local residents are consistent with the range of "background" levels currently reported in the literature. This discussion needs to include information on different age groups (versus making generalities about the entire population as a whole). Secondly, the RA can use the serum data to provide a limited reality check on the RA results. The RA should be careful to note that blood levels are not necessarily

indicative of actual exposures since individuals sampled may not be representative of the entire range of potentially exposed individuals. I agree with Mr. Washburn's comments on this topic.

I also recommend that more information on the susceptibilities of different ages (particularly young children and fetuses) be included in the toxicity assessment. Information contained in the Schantz paper might be useful.

7a. 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?

I agree that risks presented in the HHRA report (Section 7) are supported by information presented in earlier sections, but I dislike the method of presenting risks. Figures 7-1 through 7-4 present CTE and RME risks combined (the lowest CTE risk to the highest RME risk). I would prefer to see CTE and RME risks presented separately, so that I could readily differentiate between the two exposure scenarios.

7b. With respect to the conclusions in the HHRA report, 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?

I do have some concerns about the delineation of future land uses. The assessment seems to focus primarily on current land use conditions, because future land use conditions are not expected to change substantially. While this may be true, arguments for this position are weak. The text (Section 7.2.2.5) states that only properties currently used for residential or agricultural purposes were evaluated. Phase 1 screenings were based on current land use (and zonings) only, which seems to discount the possibility that some areas could be converted to agricultural or residential use in the future. Section 7 of the Phase II assessment (page 7-4) states that only Reaches 7 & 8 have areas where land use could change. Reaches 5 & 6 have "no properties that could have their screening result changed based on realistic future land use." The text should elaborate on what land uses are currently designated for Reaches 5 thru 8, and document how these land uses may change (Reaches 7 & 8) or provide justification for why the land use would not change (Reaches 5 & 6). Although this information may be summarized in Table 7-1, it is not clear to the reader as written.

I also believe that the lack of modeling data substantially hinders the RA process. Without modeling results, it is impossible to estimate how future concentrations and congener profiles may change over time. Without such information, the reliability of future risk estimates is questionable. PCB concentrations are likely to decrease over time but congener profiles could change such that the PCB congeners present in river soil and sediment could be more or less toxic. Some discussion on the lack of modeling data may have on reliably estimating future concentrations should be included in the RA.

It is very risky (and inappropriate in my opinion) to relate cancer incidence to PCB exposure. Just because ATSDR/MDPH results may not show an increase in the rate of any cancer type does not mean that the RA is overly conservative. The latency period between exposure and cancer can be several years, so it is inappropriate to use a lack of cancer incidence as verification no adverse health effects.

ADDITONAL COMMENTS ON THE HHRA NOT COVERED IN CHARGE QUESTIONS

General Comments on Vol. I, HHRA

The HHRA does not address historical exposures to residents living in or near the affected area but appropriately focuses on current and potential exposures only. While addressing past exposures will not affect the calculation of clean up goals, it is an important aspect of evaluating total risk to exposed individuals and should be addressed in some fashion in the HHRA. Measured fish concentrations used to calculate current and future exposures could underestimate risks to individuals who may have consumed fish from the Rest of River area over past decades. I recommend that some discussion of past exposures via fish consumption be included as part of the Uncertainty Analysis.

I recommend that population risk estimates be calculated to provide risk managers with additional information. The purpose of population risk estimates is not to discount individual risks, nor am I saying that remediation decisions should be based on population risk estimates. For a large, complicated site such as this one, however, population risk estimates may provide useful information for risk managers. EPA made a comment during one of the meetings that it was difficult to get an idea as to the number of people living in or area the affected area. I believe census data could be useful here.

The issue of whether or not subsistence fishing occurs in the Rest of River area needs to be finalized. Claims that subsistence fishing does not occur are weak given the many rebuttal arguments offered in other public comments on the HHRA.

Overall, I find Vol 1 (the volume summarizing the HHRA) too sparse. While I appreciate the effort to summarize the risk assessment process in the HHRA and leave the technical details to the appendices, there are several areas lacking in detail. For example, there needs to be some discussion of how COPCs were identified in the HHRA.

The Site History section of Vol IV, Appendix C (Consumption of Fish) is superior to the site history information included in the HHRA. I recommend that this section replace the current Site History section of the HHRA (Vol I).

Specific Comments on Vol. I, HHRA

Page 1, lines 24-25: The text does not present clear evidence that all PCBs present in the Rest of River Study Area originate from the GE facility. The report needs to clarify that there are no other PCBs sources upstream.

Page 1-5 to 1-6, Appendices A & B: The text states that the Phase I screening-level evaluation was based on direct contact to PCB-contaminated soil and sediments only, while Phase II evaluated PCBs, PCDDs, PCDFs. It is not clear at this point why other COPCs were not included in the Phase I and II screening assessments.

Page 1-7, lines 4-6: The text states that this report was prepared according to EPA policies and procedures using guidance documents listed in Table 1-1. Table 1-1 also lists MA Dept of Environmental Protection (MDEP) guidance documents. Suggest adding MDEP to the first sentence or deleting reference to it from Table 1-1.

Page 2-3, lines 22-23: "… where as toxicity values for noncancer effects associated with oral exposures are known as reference doses (RfDs)." This statement is misleading since there are

inhalation references doses as well.

Page 2-4, line 23: Recommend changing the "likelihood that an individual *will* develop cancer" to *may* develop cancer. CSFs are not used to predict a certainty of cancer but a probability.

Table 2-3: It would be helpful if the common names were listed for the PCBs congeners as well as the chemical formula. Also, it is not clear what the number before the colon means.

Section 2.2.2.1: It is inferred that there were no PCB congeners present in any media for which a TEQ value was not available. If this is true, it needs to be stated. If not, how these PCB congeners were handled in the TEQ calculations needs to be explained.

Page 2-8, lines 20-21: "TEQ concentration estimates … were based on measured congener data. Please clarify if congener-specific data were measured for PCDDs, PCDFs, and PCBs. Lines 23-25 seem to imply that congener-specific data were collected for PCBs only. This section needs a clearer and more extensive discussion of how concentrations for PCDDs, PCDFs, and dioxin-like PCBs were estimated using regressions analyses.

Page 2-20, lines 1-2: Agree with the use of an oral absorption factor of 100% for PCBs.

Page 2-20, lines 10-12. The specific EPA document that recommends a dermal absorption rate of 14% for PCBs need to be cited here (along with the Wester et al., 1993 study).

Section 2.4.2 is very repetitive to Section 2.2.3.2 and can be deleted.

Section 3.3: Inherent to the discussion of the calculation of the EPC (95% UCL) based on whether the data were lognormally or normally distributed is the assumption that the distribution of the data has been determined. The text should include information on how the distribution of different data sets was determined and what the results were.

Table 3-1: Please give more details either here or in the text as to how the exposure frequencies of 84 and 56 days were developed (e.g., days/week times weeks/year).

Tables 3-1 through 3-7: References for the sources of the parameters used to calculate SRBCs (e.g., skin absorption factors, soil and sediment ingestion factors, etc) should be cited here. Also, it is not clear if the values used for the various exposure parameters were upper-bound or 50^{th} percentile values. For example, Table 3-3 notes that the soil adherence factors used were 50^{th} percentile values. Similar information needs to be provided for the other exposure parameters used to calculate SRBCs.

Section 5.4.1, page 5-11, lines 23-26. Please provide a reference for the statement that EPA found no evidence of subsistence fishing in MA and CT reaches of the Housatonic River.

Sections 5.5.1.1, 5.6.1.1, Table 5-7, and Table 5-15: Cancer risks are reported for tPCBs and TEQ risks from excess dioxin-like PCBs and dioxin/furan congeners only in Sections 5.5.1.1, 5.6.1.1, Table 5-7, and Table 5-15 despite the fact that Section 5.2.1.3 (pages 5-7 to 5-8) lists 14 COPCs for fish? It needs to be clarified that tPCB risks only are reported because the relative contribution from other COPCs was very low (about 1% or less).

Table 6-4: I disagree with setting BCFs for various compounds to zero because the compound was not detected in soil.

References

U.S. Environmental Protection Agency (EPA), 1994. Estimating Exposure to Dioxin-Like Compounds, Volume III, Site-Specific Assessment Procedures. EPA/600/6-88/005C.

Table 1. Recreational Land Use Categories – Phase 1 Screening

Tax Parcel Area Evaluated	Acres Within the Floodplain	Maximum / 95% UCL Concentrations (mg/kg)
H6-4-5	14.4	151 / 84
J6-4-2	49	77 / 48
I6-1-41	32	154 / 58
J5-2-105	5.9	46 / 26
J4-3-13	35	874 / 100
J4-3-12	7.5	141 / 106
J6-1-3	8.2	117 / 190
K3-1-19	6.6	12 / 10
J2-2-2	58	78 / 17
K2-1-1	7.4	65 / 829
33-40	30	83 / 200
29-3	21	97 / 43
29-9	15	126 / 111
29-2	102	249 / 44
29-1	29	88 / 58
24-7	14.5	20 / 751
19-3	32	77 / 24
19-5	9.4	50 / 81
18-84	51	0.03
19-1	70	94 / 12
14-4	87	80 / 18
13-2	68	0.02
1-4	13	334 / 142
1-3	18	94 / 10
1-1	14	101 / 40