SENES Oak Ridge Inc.
Center for Risk Analysis

Specialists in Energy, Nuclear And Environmental Sciences

Custom Applications in Human Health And Ecological Assessment

December 17, 2003

Alison Wolfe
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Arlington, VA 22206

Dear Ms. Wolfe:

Please find enclosed my review of the HHRA of the GE/Housatonic River Site Rest of River. My attached comments are analyzed and discussed according to the standards for peer review established for reviewers of the HHRA.

I also include the results and documentation of an independent uncertainty analysis for the health risk from the ingestion of fish. In this analysis, I used probabilistic uncertainty analysis (with Monte Carlo simulation) to address epistemic uncertainty. I produced 95% credibility intervals for exposures and risks that are conditioned on reference individuals belonging to two distinctly different exposure categories, RME and CTE. These results (given in Appendix 2) were generated in Microsoft Excel, using the software add-on package Crystal Ball® (Crystal Ball® 2000 Professional Edition, Decisioneering, Inc., Denver, CO, USA).

Overall, I conclude that the HHRA for the Housatonic River is a very detailed, comprehensive and extensively documented analysis. Clear attempts have been made to ensure conformity to EPA guidance. Nevertheless, discrepancies are evident in the calculation of 95% UCLs for the exposure point concentration (EPC) for designated exposure areas. There is a definite need for quantitative uncertainty analysis to be extended to the Phase II direct exposure pathway and to the analysis of exposure and risks from the consumption of agricultural products. The uncertainty analysis performed for consumption of fish and waterfowl is deficient and biased toward extreme values. The values produced by the probability bounds analysis (PBA) approach to describe epistemic uncertainty at the high and low end of the distribution of exposures are implausibly high for describing realistic exposures received by avid recreational anglers. I recommend a more rigorous probabilistic approach be used to address epistemic uncertainty in characterizing RME and CTE exposures and that all sources of known bias be removed from the uncertainty analysis. The quantitative uncertainty analysis should

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include an evaluation of uncertainty in the EPA cancer slope factors and reference doses for non-cancer health effects.

Suggestions are given at the end of my review for using information in addition to EPA's risk range of 10^{-4} – 10^{-6} lifetime cancer risk and the HI of 1.0, in order to properly put exposures and risks from the Housatonic River into perspective.

Please let me know if there are any further questions or additional comments required of me at this time. I have enjoyed the opportunity to serve as a peer reviewer on this project.

Sincerely,

F. Owen Hoffman, Ph.D.
President and Director

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Review of GE/ Housatonic River Site Rest of River HHRA

by

F. Owen Hoffman, Ph.D.
President and Director
SENES Oak Ridge, Inc. Center for Risk Analysis

December 17, 2003

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 general, the approach is consistent with EPA guidance for initial screening. There should be a reference to the EPA Guidance for Soil Screening, however.

A more transparent discussion is needed to clearly demonstrate that the degree of conservatism included in the Phase I screening approach is sufficient to minimize false negative conclusions, without producing an extreme number of false positive cases requiring more in-depth evaluation. It is also necessary to clearly state that screening in Phase I is only for direct contact with contaminated surface soil and sediment. Phase I screening is not intended for nor is it applicable to land that could be used for agricultural purposes in which contamination of food products would be an issue.

It would help reviewers and other readers of the HHRA if a discussion could be included that presents the degree of conservatism associated with each parameter and assumption applied for screening, so that an overall impression can be given as to the robustness of the Phase I approach.

The justification of a target risk level of 5×10^{-6} needs to be strengthened.

The selection of a cancer risk level substantially higher than 1×10^{-6} appears to have been an arbitrary decision made by EPA to avoid including too many exposure areas for more detailed analysis in Phase II.

The use of a six-year exposure duration for the non-cancer risk evaluation for children exposed to PCB's should be discussed further. The maximum ratio between body weight and soil intake would be for a child in the first two years of life, and this time period could result in a higher estimate of a PCB HI per unit soil concentration than produced using an averaging time and exposure duration of 6 years.

The validity of the procedures used to estimate an upper confidence limit of the mean should be re-examined. I have a concern with the reliability of the statistical procedures

used to determine a 95% UCL of the mean when samples are not taken at random and when less than detected data are assumed to be at a PCB concentration that is one-half the detection limit. The non-randomized sample design and the mixing of non-detects with detected values to determine the underlying shape of the true distribution of soil and sediment concentrations could produce a misleading result, albeit the direction of bias is probably still towards overestimating the true mean concentration.

The extent to which the current procedures produce a reliable over-estimate of the upper 95% confidence limit of the mean PCB concentration in soil and sediment should be discussed.

If the variability of the observations is very large, or the number of samples is very small, I anticipate that it will be difficult to exclude the likelihood that the underlying distribution is lognormal, unless the majority of samples are below the limit of detection and assumed to be at a concentration that is just one-half the limit. As stated above, this assumption will distort the shape of the underlying distribution.

The substitution of the maximum value observed for the 95% UCL to determine the exposure point concentration when the maximum value is lower than the 95% UCL is consistent with EPA guidance for baseline risk assessment. For Phase I screening, however, I would prefer that the upper 95% confidence limit of the mean still be used for comparison with the SRBC, even in those cases when it is higher than the maximum value observed. As stated by the authors of the HHRA in Attachment 4 of Vol. I, the maximum observed concentration may be lower than the upper 95% confidence limit of the mean simply because the number of samples taken is few and because of the fact that the initial sample obtained was not randomized.

In those cases where the upper 95% confidence limit of the mean is greater than the SRBC, but the maximum value observed is below the SRBC, additional sampling should be considered in Phase II to obtain a more reliable estimate of the mean concentration. The substitution of the maximum value observed for the EPC when the maximum value is less than the upper 95% confidence limit of the mean would be appropriate in a Phase II evaluation when the number of samples taken is considered to be of a sufficient size and sufficiently randomized to characterize the extent of contamination within a given exposure area.

B. Phase 2 – Direct Contact Exposure Assessment

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

- *The exposure scenarios which were evaluated.*

The exposure scenarios appear appropriate for the assessment of direct contact with PCB's, although the procedure for estimating the exposure and risk to dioxin-like PCB congeners is in need of further scrutiny.

- *The exposed populations which were selected for each scenario.*

The exposed populations appear appropriate, but the potential sizes of these populations should be discussed. The averaging time and exposure durations associated with age categories used for non-cancer risk assessment may be too large for children.

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

The exposure areas appear appropriate. The assumption of complete random access to an exposure area requires additional discussion. Individuals with preferred access to a subset of areas within a defined exposure area could receive exposures markedly different from that specified by the assumed EPC. The issue has been addressed in a qualitative discussion but not in a quantitative manner.

- *The routes of exposure for each scenario.*

The routes of exposure appear appropriate.

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

Yes, in general. For non-cancer risks, the relationship between annual intake and body weight should be addressed further. The highest ratio between intake and body weight is anticipated for the youngest age groups, which would be larger than what is currently assumed for an average extended over the ages of 1 to 6 years of age.

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?

The statistical procedures and spatial weighting methods used to determine the 95% UCL for the exposure point concentration (EPC) understate the overall uncertainty associated with determination of the true mean concentration for an exposure area. Uncertainty is due to limited and non-representative sampling and the use of spatial and accessibility

weighting to interpolate estimated concentrations for areas without direct measurements. Uncertainty associated with interpolation is presently ignored in the HHRA determination of the EPC.

The present procedures for estimating an EPC for a given exposure area should be further evaluated to determine the extent to which the true mean concentration is under- or over-estimated by the EPC and the likelihood that the estimated 95% UCL properly accounts for uncertainty in the estimate of the mean, without being an implausible over-estimate of the true mean.

Interpolation is used to project PCB concentrations as point estimates for a very large number of 3 sq. meter sub-areas that make up the overall exposure area. However, the uncertainty associated with spatial interpolation is not addressed. The projected PCB concentration for each sub-area is given as single value, not a range or a probability distribution of possibly true values. Interpolation uncertainty will affect the accuracy of the point estimate projected for a given sub-area. Interpolation uncertainty will also affect the accuracy and shape of the underlying frequency distribution of projected PCB concentrations that represents the population of projected sub-areas within the exposure area.

The authors of the HHRA discuss the fact that the original samples of PCB concentrations in sediment and soil are not obtained from a randomized design. They clearly recognize and express the concern that the sampled data must be representative of the true distribution of contamination within the exposure area before classical statistical procedures can be used reliably to test the underlying distributional shape of true values and to estimate the 95% UCL of the mean. Interpolation is employed to reduce the bias associated with a non-randomized sample design.

The procedure of inverse distance weighting is used to interpolate from the few locations where samples have been taken to the many sub-areas that are without a sample. The interpolated values are point estimates, without error. Thus, the 95% UCL for an exposure area EPC does not account for uncertainty due to interpolation. The use of statistical tests on interpolated point estimates to test for the shape of the underlying frequency distribution of true PCB concentrations is questionable, given the non-random nature of the original sample and uncertainty associated with spatial interpolation.

The uncertainty associated with spatial interpolation should be included in the analysis so that the 95% UCL will be inclusive of all identifiable sources of uncertainty, not just the frequency distribution of interpolated data and the degrees of freedom determined by the size of the original sample.

When re-evaluating the procedure used to obtain the EPC, the following questions should be addressed:

(a) What difference in the estimate of the 95% UCL would occur if Kriging were used for interpolation instead of inverse distance weighting?

(b) What difference in the estimate of the 95% UCL would occur if Kriging or inverse distance weighting were to be based on the logarithms of the original data as opposed to the untransformed values?

(c) What differences in results would occur if the 95% UCL were to be based on a full probabilistic uncertainty analysis composed of numerous alternative realizations of the true but unknown spatial distribution of PCB concentrations within the entire exposure area?

In the procedure proposed in (c) above, each alternative realization of the spatial distribution of concentrations would have a unique arithmetic mean (assuming randomized access to the exposure area by a potentially exposed person). Each realized mean concentration would be a representation of the true mean for the exposure area. The variation in mean concentrations would represent all quantifiable sources of uncertainty, including uncertainty due to limited sample size, imperfect sample representativeness, approximations associated with the mathematical models and weighting coefficients used for interpolation, as well as the chance that some subareas may have true concentrations that extend beyond the observed range defined by the minimum and maximum concentrations observed.

The examples given on pages 22 to 28 of Attachment 4 to Volume 1 clearly show a wide variation in the 95% UCL when a restricted sample of size 30 is repeatedly taken at random from a data set of 1024 interpolated values. The authors of the HHRA seem to imply that the reliability of the approach used to obtain the 95% UCL from the mean and variance obtained from the entire 1024 interpolated subareas (with the degrees of freedom restricted to n=30) has been established through demonstration that agreement occurs with the average 95% UCL obtained from several thousand randomly repeated estimates of the 95% UCL (each derived from a simple random sample restricted to size n=30). I do not concur. Anticipated agreement between these two calculational approaches should be obvious, but such agreement does not establish the reliability of the result.

The variation in the repeated estimates of the 95% UCL provides some information on the overall reliability of the EPC, but it still does not account for interpolation uncertainty. Additional work needs to be undertaken to address the extent to which the present scheme used for interpolation from a non-random and somewhat biased sampling design may result in a misrepresentation of the true heterogeneity of subarea

concentrations within an exposure area and the extent to which there is an overall bias in the estimate of the exposure area mean and its 95% UCL.

The role of accessibility weighting in defining the EPC for each exposure scenario is also not entirely transparent. Intuitively, for areas that are "difficult to access" or "merely wadable," an accessibility weight of 0.5 seems high and biased towards overestimation of true exposure.

For Reach 7, the direct use of the non-random sample without interpolation to determine the 95% UCL on the mean for the EPC is most likely biased towards overestimation of the actual exposure received by an RME or CTE. The use of classical statistical tests to determine the underlying shape of the frequency distribution of contamination in soil and sediment based on samples that were not taken from a randomized design is questionable at best.

I recommend that EPA convene a separate panel of experts in uncertainty analysis of spatially distributed data to more thoroughly evaluate the adequacy of the procedures used for the estimation of the EPC before the present results are accepted for use 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 for each scenario;*

My concern here is with respect to the assumptions for children for estimation of the non-cancer HI. It is evident that exposure durations and averaging times less than 1 to 6 years could lead to a higher ratio of intake to body weight than would be produced with the current set of exposure assumptions. This is especially true for children ages 0.5 to 2 years of age who are toddlers and likely to play near the soil surface during the summer months.

An additional analysis of the appropriateness of the baseline risk values used in Phase II would require a full quantitative uncertainty analysis to reveal the effect of compounded conservative assumptions on the overall result.

- *Exposure frequency and area use factors for each scenario and exposure area;*

I will defer to my other colleagues on this issue. However, I do feel that quantitative uncertainty analysis would be useful as Phase II is a step beyond conservative screening, and should produce more realistic estimates of exposure and risk.

- *Soil ingestion rates;*

Again it appears as if the baseline exposure assumptions are standard, but a quantitative uncertainty analysis will reveal the extent to which compounded conservative assumptions lead to extreme conclusions. A quantitative uncertainty analysis will also reveal which assumptions and inputs will dominate the overall expression of uncertainty in exposure. Rather than treat inter-individual variability in the population as a stochastic process, I recommend approaching the RME exposure and the CTE as separate assessment endpoints or scenarios, each requiring their own unique set of assumptions.

Some panel members have mentioned recent studies by Calabrese and others to update assumptions used in the HHRA. I would also recommend a paper published in *Health Physics Journal* in 1998 by Dr. Steve Simon of the National Cancer Institute on the subject of soil ingestion rates (Simon S. Soil ingestion by humans: A review of history, data, and etiology with application to risk assessment of radioactively contaminated soils. *Health Physics* 74:6, 647-672. 1998).

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

I defer to my other colleagues on this issue.

- *Oral and dermal absorption factors.*

I defer to my other colleagues on this issue.

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 assumptions used for the deterministic estimate of baseline risk appear reasonable and consistent with EPA guidance. Results, however, that show merely a factor of two difference between the RME and CTE exposure are counterintuitive. I would expect a much wider margin of difference.

Again, evaluation of the effect of compounded conservative assumptions on the final results should be addressed using a quantitative uncertainty analysis, which has only been performed for the ingestion of fish and waterfowl.

6. Were the uncertainties adequately characterized and expressed?

No. The uncertainty analysis section for the Phase II Direct Contact Exposure Assessment is inadequate. All factors that could lead to an over- or under-estimate of exposure and risk should be identified and discussed. The extent to which over-or under-

estimation may occur should be quantified, at least in a general sense (i.e., less than a factor of 2, a factor of 2 to 5, on the order of a factor of 10, or greater than a factor of 10).

Preferably, a more formal quantitative uncertainty analysis should be performed. In so doing, I would recommend treating the RME and CTE as distinctly different scenarios of exposure. Probability distributions would be used that represent states of knowledge (given available evidence) about uncertain assumptions for estimating RME and CTE exposures and risks.

There is no mention of the degree to which uncertainty in the toxicity coefficients could lead to strongly biased results for either the RME or CTE. There is also a need for a quantitative uncertainty analysis associated with the use of TEQ's to estimate the cancer risk from the presence of dioxin-like PCB congeners and the use of regression analysis used to infer the quantitative presence of these congeners.

A quantitative uncertainty analysis would facilitate identification of results that contain a strong bias towards over-or under-estimation of exposure and risk. A quantitative uncertainty analysis would also disclose the relative importance of all assumptions affecting the estimate of exposure and risk and where improvements in the state of knowledge would be effective in reducing uncertainty and bias.

There needs to be more discussion about the potential for substantial bias associated with the assumption of random access to relatively large exposure areas, especially when true access for real persons may be non-random and restricted to a subsection of the overall exposure area.

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

Yes, for the deterministic estimate that leads to a baseline risk, but not from the standpoint of revealing the overall effect of compounded conservative assumptions. Therefore, I recommend that the Phase II direct contact scenario be subjected to a formal quantitative uncertainty analysis.

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 under the evaluation criteria?

Yes, for the purposes of a deterministic baseline risk assessment. The uncertainty analysis should include some probability that the RME and CTE catch freshwater fish in other locations besides the Housatonic River. There is also a need to evaluate the potential for the present concentrations to be reduced with time due to the continuous process of sedimentation with uncontaminated materials covering old contaminated sediment leading to reductions in future PCB concentrations in fish and waterfowl.

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

I believe the assumption for meal size for waterfowl ingestion is biased low. I see no logical reason why a meal size for fish consumption should be twice as large as a meal size for waterfowl. The apparent lower meal size for consumption of waterfowl is obtained from cited literature on dietary surveys on the consumption of poultry. The apparent discrepancy in the literature may be an artifact of the experimental design of the poultry dietary surveys. I question whether there are true differences in meal sizes for individuals likely to eat a meal of freshwater fish versus a meal of waterfowl. It seems intuitively reasonable that a generic meal size of about 8 oz. or 227 grams should be used for both and that meal size and body weight be correlated.

The meal sizes assumed for children ages 1 to 6 years of age appear high for the consumption of game fish. This may be due to the use of published dietary survey data for ages 3 to 5 years of age as a surrogate for the average daily consumption for the age group of ages 1 to 6 yrs. On the other hand, if non-cancer risks are relevant to an exposure duration of one to two years, then the highest ratio of intake to body weight would occur for the younger age groups of children (ages 1 to 2) even though the assumed daily dietary intake of fish for children of this age may be much less than assumed at present.

Some consideration should be given to the probability that the RME, and especially the CTE, catch freshwater fish from locations other than the Housatonic River. The assumption that 100% of freshwater game fish ingested are fish obtained from the Housatonic River appears extreme when other freshwater bodies are in the near vicinity.

Nevertheless, I would be reluctant to recommend a very high weight to the likelihood that some fraction of the total number of freshwater fish in the diet is from fish caught from other locations (as the HHRA should be focused on risks potentially caused by exposure to the ingestion of fish caught primarily from the Housatonic River). A weight of 15% to 20% could be given to the ingestion of fish from other locations for the RME who resides

continuously in the area for 70 years. A weight of up to 30% to 50% could be attributed to the CTE, but not more, especially considering that the CTE also is given an exposure duration that is substantially less than the RME. A range of plausible weights for the possibility that fish are caught from water bodies other than the Housatonic River could be considered within the quantitative uncertainty analysis.

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?

Yes, with the exception of issues already raised above.

To achieve a higher level of transparency, evaluation of fish concentration data for baseline risk EPC's need to be more explicit. It would therefore be useful to preserve information about EPC in results tables about exposures and risks for various river reaches.

Additional explanation would be useful as how EPC concentrations reflect the likelihood of harvesting migratory birds and birds taken from locations away from the Housatonic River.

Additional specific comments within Volume IV, Appendix C:

Pg. 4-30, Table 4-10: Why is the averaging time for non-cancer health effects taken to be 54 years for an adult and 6 years for a child? I would expect one year to be sufficient for estimating the non-carcinogenic effects of PCB's. For non-carcinogenic effects, the dose rate is more important than cumulative dose as is the case for carcinogens.

Pg. 4-34, Table 4-11: For the estimate of health risk, the central tendency estimate is more appropriately the arithmetic mean than the median for either an individual or a potentially exposed population.

Pg. 4-40 to 4-41: What is the justification for not using the 95th percentile ingestion rate for the assessment of non-cancer health effects? In any given year, it is likely that an angler could consume more fish than a value averaged over a prolonged time of residency, (say 50 to 60 years).

Page 4-42: I believe the ingestion rates of fish for ages 3 to 5 are biased high for the selected target group of children ages 1 to 6. I do not expect the average ingestion rate for children in this age group to be only a factor of 0.5 of that of adults.

Page 4-50, the assumption that all fish consumed are fish caught from the Housatonic River at the location of interest by the CTE is extremely pessimistic, especially for exposure durations that extend for 20 to 60 or more years.

Page 4-58, Table 4-28: Although the exposure duration for non-cancer health effects may be as long as that for cancer, the critical time of exposure could be as short as a single year. The for non-cancer health effects, the exposure rate is often more important than is the cumulative exposure over time and for young children the highest ratio between intake and body weight will occur in the first few years of life.

Pages 4-60 through 4-69, Tables 4-30 through 4-39: The ratio between the CTE and RME doses for non-cancer exposures appears too narrow to meet my sense of face validity. I would expect a difference much greater than a factor of 2, approaching a factor of almost 10 or greater. This difference is perhaps due to the assumption that both the RME and the CTE catch and consume all of their fish from a given location within the Housatonic River and that fishing occurs in a uniform manner throughout the year, with no difference occurring between the summer months (when most creel surveys are conducted) and the winter months. I believe the estimates for the RME are about right, but the CTE estimates are biased high.

Page 4-78, Tables 4-46 and 4-47: Again I believe that a bias is introduced with the direct application of the data on poultry consumption for children ages 3 to 5 as a surrogate for the age group 1 to 6. I anticipate that the age groups of 1 to 3 would be much less than the average for the age group 3 to 5. I also anticipate that the dietary survey is not appropriately age averaged. I anticipate more participants in the 4 to 5 year old range than in the 3 to 4 year old range.

If the non-cancer Hazard Index is averaged over 1 year instead of 6 years, the intake to body weight for a one to 2 year old is expected to be much higher than the value of 1.1 g/kg-d given for the age group 3 to 5 years.

Page 4-79: Why is the meal size for the consumption of poultry less than that for fish (i.e., 110 g per meal as compared with 227 g per meal)?

Page 4-81, Tables 4-48 and 4-49: the exposure duration and averaging times for non-cancer health effects seem very long. This would be appropriate if the non-cancer health impacts are the result of the cumulative lifetime exposure to PCB's as opposed to the maximum annual exposure rate.

Although, for the assessment of the non-cancer HI, the averaging time and exposure duration cancel, the ratio of intake-to-body weight will differ markedly for children of ages 1 to 3 than for children ages 3 to 6. The differences in the ratio of body weight-to-intake will be even more pronounced for children ages 1 to 3 than for adults. The ratio of body weight-to-intake will directly affect the magnitude of the Hazard Index.

Page 5-3, Figure 5-1, and Page 5-5, Figure 5-2: There are too many variables displayed and subsumed within the colored bars. At the very least, the risks and HI's for the RME should be kept separate from the CTE.

Figure 5-1 shows the importance of carefully investigating the affect of conservatism

associated with the use of one half the detection limit for dioxin-like congeners of PCB's and the assignment of specific TEQ's for each congener. Variation due to River Reach should be separated from variation due to the RME and CTE.

Pages 5-7 through 5-31, Tables 5-2 through 5-21: show in each table the value used as the Exposure Point Concentration. For example, it is apparent in Table 4-7 on page 4-25 that the EPC for Smallmouth Bass-West Cornwall/Bulls Bridge is more than one half that of Brown Trout-West Cornwall, yet the cancer risks and HI for Brown Trout are less than those calculated at the same location for Smallmouth Bass. This seems counterintuitive, until one remembers that different ingestion rates are assumed for Smallmouth Bass than for trout. This should be pointed out in a footnote to the tables.

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

The approaches were extensively described, but the appropriateness of the approaches used is questionable for a number of reasons.

The HHRA uncertainty analysis assumes that all inter-individual variability in exposure is due to natural stochasticity and erroneously states that such variability is irreducible. Uncertainty due to inter-individual variability can be reduced substantially by conditioning the assessment on the life-styles and other attributes of the target populations of interest (such as the average member of a population of casual recreational fishermen versus a representative member of a much smaller group of avid consumers of river fish, who are likely to utilize the river over extended periods of time).

Probabilistic approaches were restricted in the HHRA to the simulation of stochastic variability (aleatory uncertainty) of exposure within a relatively undefined population mixed with casual and avid recreational anglers. Epistemic uncertainty was analyzed using a non-probabilistic approach known as Probability Bounds Analysis (PBA).

Despite claims to the contrary in the HHRA, PBA is not well established and is rarely ever used in human health risk assessment. This reviewer found the description of the PBA approach extremely difficult to comprehend upon both first and subsequent readings. It took considerable effort to become familiar with the mathematical procedures and their limitations. The substitution of PBA in the Housatonic Rest of River HHRA for probabilistic methods appears to be have been a deliberate decision influenced by individuals who, because their conviction as frequentists, are averse to the use of Bayesian probability to represent the state of knowledge about true but uncertain quantities.

Contrary to what is stated in the HHRA concerning the advantages of PBA over Monte Carlo methods, probabilistic uncertainty analyses are:

- established and accepted procedures for addressing epistemic uncertainty,

- easier to implement and more transparent than PBA,

- provide probabilistic uncertainty information on the computed exposure and risk which is a quantitative representation of the analyst's state of knowledge,

- the resulting 90 or 95% credibility intervals for the RME and CTE are more suitable for decision making than the extreme limits produced by the PBA.

The discussion given in the HHRA (pages 1 through 69 of Attachment 5 to Volume 1) to justify the advantages of the probability bounds analysis over probabilistic uncertainty analysis reflects mostly points of view of the authors. They are not statements of scientific fact, nor are these statements widely endorsed by the majority of practitioners of probabilistic uncertainty analysis (Kaplan and Garrick 1981, Bogen and Spear 1987, IAEA 1989, Morgan and Henrion 1990, Hoffman and Hammonds 1994, MacIntosh 1994, NRC 1994, Burmaster and Rhodes 1996, Frey and Rhodes 1996, NCRP 1996, NCRP 1997, Pate-Cornell 1996, Frey 1998, Frey and Rhodes 1998, Cullen and Frey 1999, EPA 1999, Hoffman and Kaplan 1999. A fuller list of references is found at the end of this document.).

The local sensitivity information obtained from the PBA is inadequate to guide decisions as to where to improve the state of knowledge in order to effectively reduce epistemic uncertainty of the computed risk. This inadequacy is not just because of the local nature of the PBA sensitivity analysis that requires "pinching" of a p-box for an input variable at a specified percentile of the frequency distribution, but also because the PBA approach fails to address differences in the state of knowledge within the limits of its extreme values.

The upper bounds produced by the PBA at the upper percentiles of the frequency distribution of exposure are implausible extremes for a population of recreational anglers. The results at the upper end of the distribution of angler exposures are more indicative of subsistence fishermen, the existence of which has not been demonstrated to date.

The sensitivity of the risk estimates to uncertainty in the toxicity coefficients is not accounted for in the HHRA uncertainty analysis. The present HHRA uncertainty analysis has been restricted to the assumptions that determine individual exposure. The uncertainty in the cancer slope factor and RfD is not addressed, as a matter of EPA policy. This is an area where EPA policy and guidance should be reconsidered and improved.

I recommend that the HHRA uncertainty analysis be extended to include the uncertainty in the toxicity coefficients of the risk assessment. These coefficients are often the dominant source of uncertainty, once the attributes of exposure duration and exposure frequency have been defined for individuals who are representative of RME and CTE exposures.

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

The data sources for probability distributions were clearly described, but these data are not necessarily appropriate for defining stochastic variability of exposure without careful evaluation of the limitations of the data. The extent to which the various data sets are directly relevant for defining stochastic variability of exposure within a defined population has not been considered. It is evident that only minor amounts of epistemic uncertainty are assumed for exposure frequency while, for cancer risk, almost all the uncertainty is assumed to be associated with exposure duration, with a heavy weight assigned to minimum and maximum values. No probability distributions were assigned to represent sources of epistemic uncertainty.

No distributions are assigned to describe variability in the EPC for fish and waterfowl. Yet, the mean concentration in fish will be different for each person sampling a finite number of fish from the river. The uncertainty in the EPC is defined by a range of values arbitrarily restricted to the sample mean and the deterministic EPC. The uncertainty in the mean PCB concentration should be greater for an individual who consumes few fish from the river than for one who consumes many.

The application of data on fish consumption rates obtained from a relatively short-term dietary survey of sport anglers in Maine (from Ebert et al., 1993) to the population of recreational anglers who would use the Housatonic River is assumed to have only a 10% uncertainty in the estimates of the mean and spread of the distribution. No credit is given to the claim that some fish and waterfowl would be harvested by avid and casual recreational anglers from areas outside of the Housatonic River.

The p-boxes used for the PBA, however, assume that some individuals consume fish from the Housatonic River daily for the entire duration of their residence history, which is not a plausible assumption for recreational anglers. This assumption probably overstates the intake from representative members of subpopulations considered sustenance fishermen. However, there appears to be no recent record of the Housatonic River used by such persons.

The distribution of body weight is used to address stochastic variability but not epistemic uncertainty. The fact that cited information on short term observations of the variability of human body weights in a general population may only approximately describe the true variability of lifetime exposure among recreational anglers is not considered as a source of uncertainty. At a given percentile of the true frequency distribution of exposure, body weight is ignored as a factor that contributes to epistemic uncertainty.

The substitution of PBA for more established probabilistic approaches to address epistemic uncertainty is a major shortcoming of the quantitative uncertainty analysis in this HHRA. More detail on this issue is included in the following discussions.

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

No. In general, the rationales for choosing which variables describe stochastic inter-individual variability and which are associated with epistemic uncertainty are either missing or based on arbitrary assumptions. I have found the description of the probability bounds analysis and its implementation especially difficult to interpret. This difficulty has been compounded by an obvious upward bias introduced by ignoring the information contained in the lower confidence limit on the mean concentrations in fish and waterfowl and the upward bias introduced by using the EPA toxicity coefficients as point estimates, without including an estimate of uncertainty.

I have noted above that in the HHRA analysis of epistemic uncertainty, inter-individual variability of body mass is assumed to be known perfectly. On the other hand, the uncertainty in exposure duration for both the RME and the CTE is assumed to range from 1-64 years with heavy weights given to the extremes of this range (an assumption that I feel is unreasonable). Likewise, only a negligible 10% uncertainty is arbitrarily assigned to the distribution of the average number of fish meals consumed in a year (which I consider to be an underestimate of epistemic uncertainty). No variability is assumed to occur in the average concentration of PCBs in fish, nor for the size of the average fish meal (which leads to an underestimate of stochastic variability). The uncertainty in the mean concentration of PBC's in fish and waterfowl is too small and biased towards values that exceed the sample mean. No consideration is given to the chance that some fraction of the total freshwater fish consumed is taken from locations other than the Housatonic River.

The probability bounds analysis indicates that the HHRA point estimates of cancer risk and non-cancer risk calculated in Chapter 5 could be substantial understatements of the true risk. This impression is misleading. The very high values produced as upper bounds are partially an artifact of the PBA method itself and partly a function of the rather arbitrary assumptions made about which parameters were to be considered as determinants of stochastic variability and which were to be assigned a p-box to represent epistemic uncertainty in true but unknown quantities.

The extent of bias towards high values of exposure and risk would become apparent if the uncertainty analysis of the HHRA were to be carefully re-evaluated and the attributes of the CTE and RME targeted explicitly in a one dimensional Monte Carlo uncertainty analysis of epistemic uncertainty. The extent of this bias would become further apparent if the uncertainty of the cancer slope factors and RfD's would be taken into account.

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

No. The failure to address uncertainty in the toxicity coefficients used in the HHRA is a major shortcoming of the uncertainty analysis section of this report. In this analysis, probability distributions are used only to depict inter-individual variability in exposure as a stochastic process. Epistemic uncertainty in exposure is not addressed using probabilistic uncertainty analysis.

However, for the EPA toxicity coefficients, neither inter-individual variability nor epistemic uncertainty is addressed quantitatively (a practice that is unfortunately consistent with current EPA policy and guidance for Superfund risk assessment). In the absence of quantitative information on the uncertainty in the estimate of the cancer slope factors and the RfD's for PCB and PCB congeners, the quantitative uncertainty analysis of the HHRA for fish and waterfowl ingestion degrades into an evaluation of exposure only, not risk.

Because the PBA is based on the propagation of extreme values, the results of the HHRA PBA give the impression that the point estimates of cancer risk and HI's in Chapter 5 of Volume IV, Attachment C are potentially either over- or under-estimates of true exposures. I believe this result is an artifact of the PBA approach that propagates extreme values combined with a systematic bias of input assumptions that are skewed towards high values of exposure and risk.

A systematic bias towards over-statement of the risk is partly due to

(a) the treatment of the toxicity coefficients as having no uncertainty,

(b) the failure to account for the full uncertainty on the mean concentration of PCB's in fish, including the mean concentration of PCB like congeners,

(c) the assumption that 100% of the fish consumed are from the river, and

(d) the assumption that fish are caught from the river in every month out of the year.

Because the size of the population of recreational anglers is not rigorously defined, it is difficult to determine what the upper-end of the distribution of exposures represents that is simulated by Monte Carlo analysis. The Monte Carlo analysis used in the HHRA to simulate inter-individual variability as a stochastic process is truncated at the upper 99^{th} percentile of the frequency distribution of true individual exposures. If the population size were as large as 10,000 persons, the 99^{th} percentile would underestimate exposure for the top 100 persons in the distribution. The 95^{th} percentile would underestimate exposure for the top 500 persons. The 90^{th} percentile would underestimate exposure for the top 1000 persons.

I have made a preliminary probabilistic evaluation of epistemic uncertainty in cancer risk and HI for the RME and CTE, respectively, for a nominal PCB concentration in fish of 1 ppm (see attached MS Excel spreadsheet workbook). This PCB concentration is roughly comparable to the EPC concentrations used for bass caught from West Cornwall/Bulls Bridge. In making this comparison, I have assigned probability weights to a range of plausible values to quantify epistemic uncertainty in model inputs, including uncertainty in the cancer slope factor and RfD. Monte Carlo simulation was employed to propagate epistemic uncertainty from inputs to exposure and risk. The results are expressed as a 95% credibility interval for the RME and the CTE (see Appendix 1, including attached MS Excel/Crystal Ball spreadsheet).

Based on these calculations, I conclude that the HHRA point estimates of risk in Vol. IV, Chapter 5 are in reasonable agreement with the upper limit of a 95% credibility interval of cancer risk and non-cancer HI for both the RME and CTE. This conclusion was maintained even after the analysis was re-run with toxicity coefficients held constant at their specified EPA regulatory defaults of 2 $(\text{mg kg}^{-1}\ \text{d}^{-1})^{-1}$ for the cancer slope factor and 2×10^{-5} mg $\text{kg}^{-1}\ \text{d}^{-1}$ for the RfD.

The relative range of my 95% credibility intervals for the RME and CTE was about two orders of magnitude for either the RME or CTE. This was reduced to about a factor of about 20 when the toxicity coefficients are assumed to be fixed without uncertainty, although the upper bound of the 95% credibility interval did not change appreciably (a similar result was reported by Land 2002 [Land C. Uncertainty, low-dose extrapolation and the threshold hypothesis. *J. Radiol. Prot.* 2:1–7. 2002]). By comparison, the range of the HHRA probability bounds analysis often approaches three orders of magnitude.

The upper bounds of the PBA exceed the limits of my 95% credibility interval for the RME and CTE by an order of magnitude (Appendix 1, Table 1). These upper bounds produced by the PBA appear implausibly high for a realistic population of avid recreational anglers. On the other hand, the relative range of uncertainty at a given percentile that is produced by the PBA for non-cancer HI is merely a factor of about 2 around a central value. This result for non-cancer risk implies a level of epistemic uncertainty (at a given percentile of the frequency distribution that describes inter-individual variability of true exposure) that is intuitively implausible.

The expectation of uncertainty much larger than a factor of two, even at a given percentile of the frequency distribution of true exposures, is based on the use of disparate sets of partially relevant data sets to define the true but unknown frequency distribution of exposure, the use of restricted bounds to describe the uncertainty in the mean PCB concentration in fish and waterfowl, the need to consider the fact that realistic harvesting of fish and waterfowl will include locations other than the Housatonic River, and the fact that the target population for which stochastic variability in exposure is simulated is essentially undefined. The range of uncertainty in HI at a given percentile will be expanded still further if the HHRA analysis were to include uncertainty in the RfD for PCB's and the dioxin-like PCB congeners, as the dominant source of uncertainty in these variables is epistemic.

Additional comments on Volume IV, Appendix C, Chapter 6 of the HHRA:

1. Page 6-1: the CTE should approximate the mean, not the median of the population of recreational anglers.

2. Page 6-11: the HHRA states that the exposure point concentration (EPC) should be evaluated with a probability bounds analysis by substituting an interval for the point estimate. The interval "must be bounded below by a value that is known to be as low as the EPC could possibly be and above by a value that is known to be as high as the EPC could possibly be." But, in actuality the bounds are the sample mean and the EPC, whereby the EPC is the upper 95% confidence limit of the mean. The lower confidence limit on the mean is not used. This leads to an upward bias in the overall estimate of exposure and risk to the RME and CTE.

3. Pages 6-28 through 6-92, Figs. 6-17 through 6-103: all figures should be replotted using a logarithmic scale since the results span several orders of magnitude. The point estimates of the RME and CTE risk values in Chapter 5 should be included for comparison.

4. Pages 6-60 through 6-91, Tables 6-6 through 6-13: the depiction of the RME as belonging to a subgroup potentially spanning the 90^{th} to the 99^{th} percentile of the population is useful. Additionally useful would be information about the potential size of the population of anglers being simulated.

   For example, if the population of recreational anglers were to approach 10,000 persons, the upper 90^{th} percentile of the distribution would underestimate exposures and risks for the top 1000 individuals and the 99^{th} percentile would underestimate exposure and risks for the top 100 persons.

   For this reason, I believe it best to target the analysis on the RME and CTE as separate entities and not attempt to simulate inter-individual variability as a stochastic process. There are defined reasons (by number of fish meals and length of residency in the region) that can explain the major sources of individual variability of exposure. Variability need not be treated as a purely stochastic process. For the sake of transparency and ease of analysis, I would replace the one-dimensional Monte Carlo analysis of aleatoric variability with a one-dimensional analysis of epistemic uncertainty targeted at reference persons representing the attributes of the CTE and RME.

   Because the HHRA probabilistic analysis is supposed to investigate the uncertainty in true exposures and risk, I recommend that the probabilistic risk analysis include the uncertainty in the PCB and TEQ concentrations in fish and the uncertainty in the toxicity factors (i.e., the cancer slope factor for PCB's and Dioxin, the Toxicity Equivalent Factors for dioxin-like PCB's, and the RfD for PCB's consumed in fish). The uncertainty in EPA toxicity coefficients may need to be undertaken as an effort that is external to this particular assessment for the Housatonic River. Nevertheless, I feel that such information would be of value to

risk managers, especially given the magnitude, cost, and potential disruption to sensitive habitats created by remediation efforts.

5. General comments on Chapter 6:

   I believe it best to perform a probabilistic uncertainty analysis on the CTE and the RME separately and to include the uncertainty in the mean concentration of PCB's and dioxin-like congeners in fish and waterfowl and the uncertainty in the EPA toxicity coefficients for cancer and non-cancer health effects.

   If EPA insists on using Monte Carlo techniques to simulate variability and exposure within the general population of recreational anglers using the questionable assumption that all inter-individual variability is stochastic, then I would prefer the use of two dimensional (second order) Monte Carlo simulation over the present probability bounds analysis to address epistemic uncertainty. Second order Monte Carlo analysis is well established in the general risk assessment community. The inner loop simulates the unknown frequency distribution, the outer loop generates alternative realizations of this unknown distribution based on all quantities for which there is lack of knowledge. In this case, I would prefer for the size of the simulated population be specified and that the upper 99.9^{th} percentile of the distribution be quantified.

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?

The deterministic analysis of the baseline risk assessment appears to be appropriate with the exception of the items mentioned in response to earlier questions. The chance that fish and waterfowl are harvested from locations other than the Housatonic River should be considered as well as the chance that PCB concentrations will be reduced in future time. Numerous sources of bias need to be removed from the quantitative uncertainty analysis and the PBA approach should either be replaced, or at least augmented, by a probabilistic analysis of epistemic uncertainty. The upper bound estimates of the PBA are unrealistically high for a population of avid recreational fishermen and thus violate a common sense of face validity. The quantitative uncertainty analysis should be extended to include uncertainty in the toxicity coefficients.

D. Phase II – Agricultural Exposures

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

The approaches appear conceptually reasonable, but the analysis should be based on actual measured concentrations of PCB's in soil.

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

There is so much uncertainty associated with the estimation of soil-to-plant transfer, that a formal quantitative uncertainty analysis should be performed.

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

There is so much uncertainty associated with the estimation of the transfer of PCB's to animal tissue, milk and eggs, that a formal quantitative uncertainty analysis should be performed.

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

There is a need to evaluate the potential for compounded conservative assumptions leading to a strong bias in the over-all result.

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

Mostly. But there is a need for an evaluation of the effect of uncertainty on the final result of exposure and risk. This is an area that would benefit from directed research to increase the base of knowledge about agricultural transfer coefficients for PCB's and dioxin-like congeners.

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

Yes, with the possible exception of the consumption of milk. The milk pathway usually affects infants and very young children more than adults. The consideration of childhood exposure, especially early in life, is potentially important for the evaluation of non-cancer risks.

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

No. There is a need for a formal uncertainty analysis to be performed for the agricultural pathway.

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?

The evaluation of the overall approach would be facilitated if a full quantitative uncertainty analysis were to be performed. This analysis should be extended to include the uncertainty in the toxicity coefficients as well. The uncertainty analysis need not attempt to simulate inter-individual variability of exposure in a large population. It would be sufficient for the analysis to address epistemic uncertainty in exposure and risk to the RME and CTE as separate exposure scenarios. Careful consideration should be given to the appropriate age groups and ratios of intake to body weights of children when estimating the non-cancer HI.

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?

The assessment would be markedly improved if the uncertainty in slope factors, reference doses, and calculations of TEQ were to be included in a quantitative uncertainty analysis. The bases for the chosen slope factors and reference doses should include a discussion about the fact that PCB's have long residence times in the human body and thus the daily average intakes normalized to body weight might not be as pertinent in determining the actual risk as would the accumulated body burden over time.

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

Yes, but I would prefer that information about the results for the RME and CTE risk be kept separate, as well as information about the risks for each specific reach of the Rest of River.

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?

Not with respect to uncertainty. A full quantitative uncertainty analysis is recommended for the direct contact and agricultural product pathways. Probabilistic approaches are preferred for addressing epistemic uncertainty as opposed to the non-probabilistic PBA.

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

No. See above comments. I do feel, however, that the baseline deterministic risk estimates approach the upper bounds of a 95% credibility interval when a probabilistic uncertainty analysis was conditionally focused on the RME and CTE as separate exposure scenarios. This means that the deterministic risk estimates are reasonably protective without being a gross overestimate of true risk. The extent to which the deterministic estimates may be an overestimate of true risk would require that the uncertainty in the cancer slope factors and the RFI be accounted for quantitatively.

F. General

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

Yes.

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

Yes, with the exception of the numerous cautions mentioned above about the need to formally assess uncertainty. This is also an area that would benefit from additional research.

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

Yes, but EPA guidance is unclear with respect to the use of probabilistic uncertainty analysis for quantifying epistemic sources of uncertainty. EPA guidance remains to be developed to address quantifying uncertainty in toxicity coefficients.

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.

No. The failure to quantify uncertainty in the toxicity coefficients used and the TEQ's calculated is a major shortcoming of the present analysis. Uncertainty in the EPC for sediment, soil, fish, and waterfowl should be further evaluated and inappropriate bias removed from the uncertainty analysis.

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?

See "Soil ingestion by humans: A review of history, data, and etiology with application to risk assessment of radioactively contaminated soils."(S. Simon. *Health Physics* 74:6, 647-672. 1998.) and "Uncertainty, low-dose extrapolation and the threshold hypothesis" (C. Land. *J. Radiol. Prot.* 2:1–7. 2002.) concerning the influence of threshold values of risk within a probability distribution defining epistemic uncertainty in the cancer slope factor for radiation. See the attached references documenting the use of probabilistic approaches for addressing epistemic uncertainty.

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.

The HHRA has not considered the possibility that future concentrations of PCB's will be lower in the future due to natural processes of sedimentation. Note that the HHRA for the Hudson River did include the effect of long-term sedimentation on reducing future lifetime exposures.

The use of Kriging should be explored for estimating exposure area EPC's, and formal uncertainty analysis used to obtain the 95% UCL. The harvesting of uncontaminated fish and waterfowl from locations other than the Housatonic River need to be considered.

A discussion needs to be given about the relatively long residence time of PCB's in the human body and the build-up of PCB's in human tissue over time that could result from many years of chronic exposure. The question remains about how the cumulative body burden of PCB's relate to the estimate of cancer and non-cancer health risk (as opposed to a daily averaged dose). To the extent that biokinetics of long term exposure indicate a potential underestimate of life-time health risk, this source of uncertainty should be taken into account explicitly.

7. 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?*

In general, this is true. In fact, the critical details of earlier discussions probably are not sufficient to conclude that PCB exposure in the Housatonic River is not a health risk of concern, especially for the ingestion of fish and waterfowl. The fact that human epidemiological information in worker populations is inconclusive cannot be used as evidence of no risk, especially since workers are not exposed to the same pathways and congeners of PCB's as are recreational anglers and other members of the public. Nevertheless, the EPA policy not to engage in a quantitative uncertainty analysis for the toxicity coefficients prevents evaluation of the likelihood that the deterministic estimates of risk could be overly pessimistic due to the effects of multiple compounded conservative assumptions. Although this uncertainty is undoubtedly large and, will probably dominate over the uncertainty in exposure, I doubt (based on my own independent analysis of uncertainty) that the uncertainty in the risk coefficients will be so large as to negate the conclusion that the present levels of PCB's in the Housatonic River represent an important source of environmental contamination.

- *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?*

Yes, with the exception of numerous comments made above. I feel that the PBA analysis performed to address epistemic uncertainty for the ingestion of fish and waterfowl is misleading and biased towards high values of exposure and risk. I recommend that the PBA be replaced or augmented by a probabilistic assessment of epistemic uncertainty about the quantification of RME and CTE exposures and risks and that the rationales for the choice of probability distributions to address state of knowledge uncertainty be strengthened.

The potential future risks have not been adequately addressed. There is no procedure for incorporating the results of sediment modeling into the HHRA. Quantitative uncertainty analysis remains to be performed for the Phase II Direct Contact Exposure Scenarios and exposures from the agricultural pathway.

The presentation of the context of results should include more than the EPA target risk range. I recommend that risks be placed into perspective with the background risk of cancer and anticipated non-cancer diseases for a non-exposed population. Risks can be given as lifetime absolute risk (background + exposure), excess lifetime absolute risk above background, and excess relative risk above background. These estimates would help the affected local population evaluate the extent to which exposures to PCB contamination are likely to affect the background incidence of disease and the extent to which such exposures are at or above epidemiological limits of detection for a large cohort size. In putting risks into perspective, the size of the potentially exposed population should be addressed.

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Appendix 1

Table 1. A comparison of an independent uncertainty analysis targeted at the RME and CTE angler with the HHRA results at West Cornwell/Bulls Bridge for consumption of bass. Created using Crystal Ball.

Uncertainty Analysis Targeted at the RME and CTE		HHRA West Cornwall/Bulls Bridge, Bass Consumption		
95% credibility interval [risk coefficients uncertain]	95% credibility interval [risk coefficients constant]	HHRA point estimates	PBA (90th, 99th percentile) lowest lower to highest upper bound	PBA (95th percentile)
RISK RME	RISK RME			
1.1 E-5 to 1.3 E-3	8.6 E-5, 1.4 E-3	1 E-3	3 E-5, 1.8E-2	4.1 E-5, 4.2 E-3
			PBA (25th, 75th percentile)	PBA (50th percentile)
RISK CTE	RISK CTE			
8.7 E-7 to 1.3 E-4	5.5 E-6, 1.7 E-4	9 E-5	2.2 E-6, 6.8 E-4	5.3 E-6, 2.9 E-4
Non-Cancer Hazard Index		Non-Cancer Hazard Index		
			PBA (90th, 99th percentile)	PBA (95th percentile)
HI RME	HI RME			
0.24 to 36	4.4, 54	26	8.2, 370	13, 72
			PBA (25th, 75th percentile)	PBA (50th percentile)
HI CTE	HI CTE			
0.035 to 7.0	0.62, 11	12	0.45, 13	1.4, 5.1

Appendix 2
PCB Risk Assessment
Developed using Crystal Ball

Table 2										
Cancer risk (adult)										
RME										
fish conc	food prep loss	meal size	meals per month	mo/yr fish taken from river	ave. daily consumption	number of years consuming fish	averaging time	body weight	slope factor	**risk**
mg/kg		kg			kg/d	yr	yr	kg	(mg/kg/d)^-1	
1	0	0.227	4	12	0.0299	64	70	70	2	**7.8E-04**
CTE										
fish conc	food prep loss	meal size	meals per month	mo/yr fish taken from river	ave. daily consumption	number of years consuming fish	averaging time	body weight	slope factor	**risk**
mg/kg		kg			kg/d	yr	yr	kg	(mg/kg/d)^-1	
1	0.24	0.227	2	6	0.0075	25	70	70	1	**2.9E-05**
Cancer risk = C_{fish} x (1-food loss) x meal size x meals per month x months per year fish taken from river x number of years fish consumed from river x cancer slope factor / averaging time / body weight										
Non-cancer risk (adult)										
RME										
fish conc	food prep loss	meal size	meals per month	mo/yr fish taken from river	ave. daily consumption	number of years consuming fish	averaging time	body weight	RfD	**HI**
mg/kg		kg			kg/d	yr	yr	kg	(mg/kg/d)	
1	0	0.227	4	12	0.0299	1	1	70	2.00E-05	**21.3**
CTE										
fish conc	food prep loss	meal size	meals per month	mo/yr fish taken from river	ave. daily consumption	number of years consuming fish	averaging time	body weight	RfD	**HI**
mg/kg		kg			kg/d	yr	yr	kg	(mg/kg/d)	
1	0.24	0.227	2	8	0.0100	1	1	70	2.00E-05	**5.4**
Non-cancer HI = C_{fish} x (1-food loss) x meal size x meals per month x months per year fish token from river / body weight / RfD										

Appendix 2.1

PCB Risk from fish ingestion

All risk coefficients assumed to be uncertain

Crystal Ball Report

Simulation started on 11/16/03 at 14:44:35

Simulation stopped on 11/16/03 at 14:44:37

Forecast: RME risk **Cell: K5**

Summary:

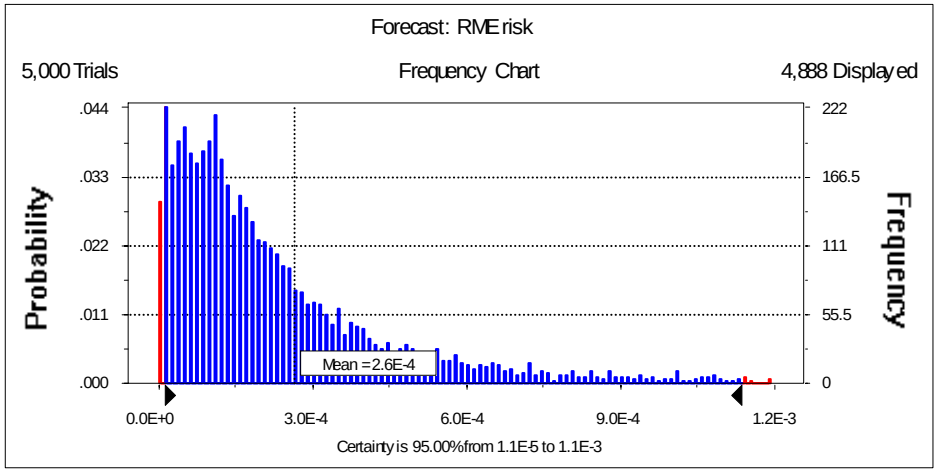
Certainty Level is 95.00%

Certainty Range is from 1.1E-5 to 1.1E-3

Display Range is from 0.0E+0 to 1.2E-3

Entire Range is from 1.9E-6 to 7.7E-3

After 5,000 Trials, the Std. Error of the Mean is 4.8E-6



Percentiles:

Percentile	Value
0.0%	1.9E-06
2.5%	1.1E-05
5.0%	1.7E-05
50.0%	1.6E-04
95.0%	8.4E-04
97.5%	1.1E-03
100.0%	7.7E-03

End of Forecast

Appendix 2.1

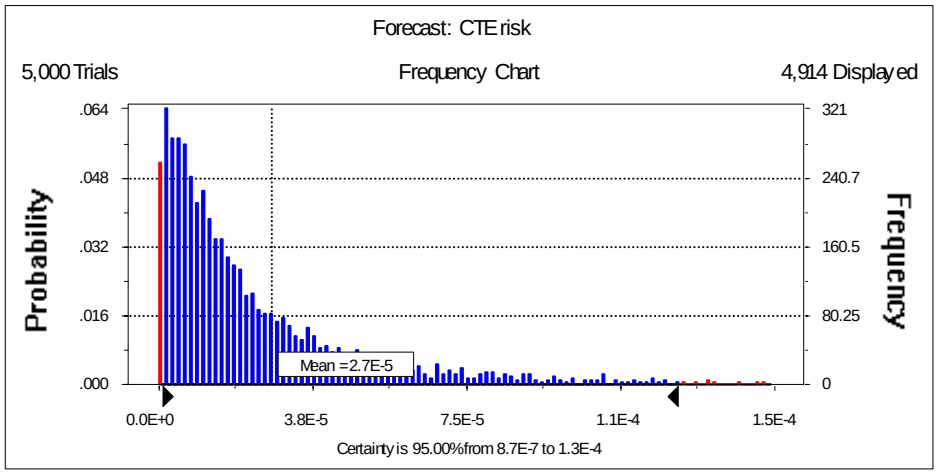
PCB Risk from fish ingestion

All risk coefficients assumed to be uncertain

Forecast: CTE risk **Cell: K10**

Summary:

Certainty Level is 95.00%
Certainty Range is from 8.7E-7 to 1.3E-4
Display Range is from 0.0E+0 to 1.5E-4
Entire Range is from 9.6E-8 to 4.8E-4
After 5,000 Trials, the Std. Error of the Mean is 5.4E-7



Percentiles:

Percentile	Value
0.0%	9.6E-08
2.5%	8.7E-07
5.0%	1.4E-06
50.0%	1.5E-05
95.0%	9.1E-05
97.5%	1.3E-04
100.0%	4.8E-04

End of Forecast

Appendix 2.1

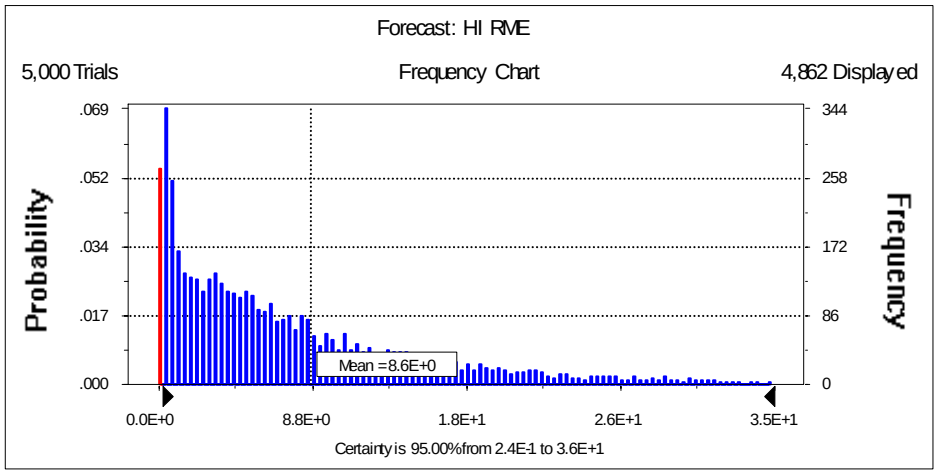
PCB Risk from fish ingestion

All risk coefficients assumed to be uncertain

Forecast: HI RME **Cell: K17**

Summary:

Certainty Level is 95.00%
Certainty Range is from 2.4E-1 to 3.6E+1
Display Range is from 0.0E+0 to 3.5E+1
Entire Range is from 3.2E-2 to 1.1E+2
After 5,000 Trials, the Std. Error of the Mean is 1.4E-1



Percentiles:

Percentile	Value
0.0%	3.2E-02
2.5%	2.4E-01
5.0%	3.3E-01
50.0%	5.6E+00
95.0%	2.8E+01
97.5%	3.6E+01
100.0%	1.1E+02

End of Forecast

Appendix 2.1

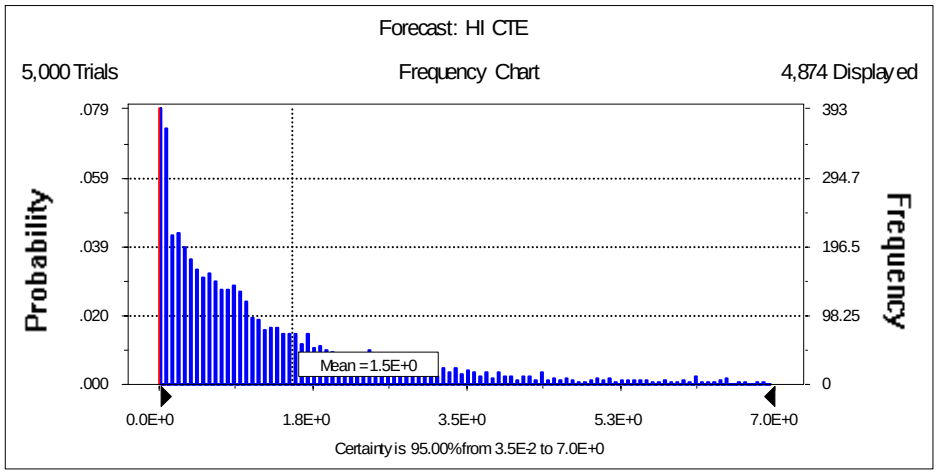
PCB Risk from fish ingestion

All risk coefficients assumed to be uncertain

Forecast: HI CTE **Cell: K22**

Summary:

Certainty Level is 95.00%
Certainty Range is from 3.5E-2 to 7.0E+0
Display Range is from 0.0E+0 to 7.0E+0
Entire Range is from 4.7E-3 to 3.7E+1
After 5,000 Trials, the Std. Error of the Mean is 2.9E-2



Percentiles:

Percentile	Value
0.0%	4.7E-03
2.5%	3.5E-02
5.0%	5.3E-02
50.0%	8.6E-01
95.0%	5.3E+00
97.5%	7.0E+00
100.0%	3.7E+01

End of Forecast

Appendix 2.1

PCB Risk from fish ingestion

All risk coefficients assumed to be uncertain

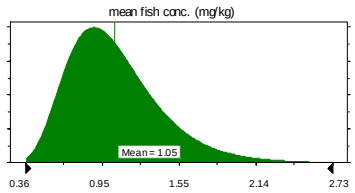
Assumptions

Assumption: mean fish conc. (mg/kg) **Cell: A5**

Lognormal distribution with parameters:

Geometric Mean	1.00
Geometric Std. Dev.	1.40

Selected range is from 0.00 to +Infinity

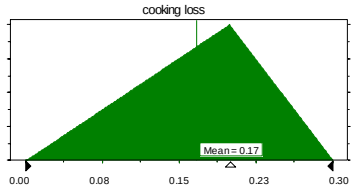


Assumption: cooking loss **Cell: B5**

Triangular distribution with parameters:

Minimum	0.00
Likeliest	0.20
Maximum	0.30

Selected range is from 0.00 to 0.30

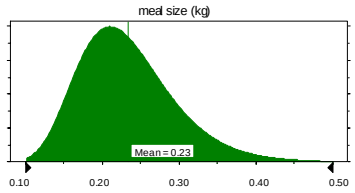


Assumption: meal size (kg) **Cell: C5**

Lognormal distribution with parameters:

Geometric Mean	0.23
Geometric Std. Dev.	1.30

Selected range is from 0.00 to +Infinity



Correlated with:

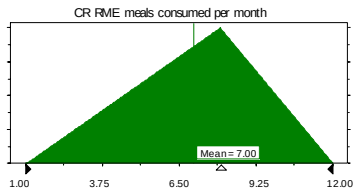
Body Weight (kg) (I5)	0.70

Assumption: CR RME meals consumed per month **Cell: D5**

Triangular distribution with parameters:

Minimum	1.00
Likeliest	8.00
Maximum	12.00

Selected range is from 1.00 to 12.00



Appendix 2.1

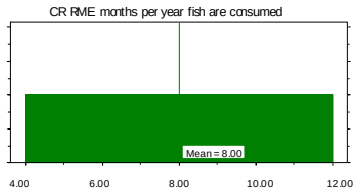
PCB Risk from fish ingestion

All risk coefficients assumed to be uncertain

Assumption: CR RME months per year fish are consumed **Cell: E5**

Uniform distribution with parameters:

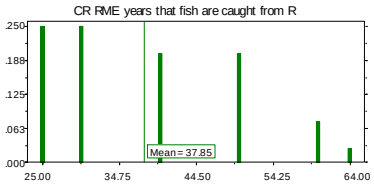
Minimum	4.00
Maximum	12.00



Assumption: CR RME years that fish are caught from R **Cell: G5**

Custom distribution with parameters:

		Relative Prob.
Single point	25.00	0.250000
Single point	30.00	0.250000
Single point	40.00	0.200000
Single point	50.00	0.200000
Single point	60.00	0.075000
Single point	64.00	0.025000
Total Relative Probability		1.000000

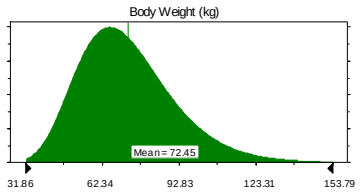


Assumption: Body Weight (kg) **Cell: I5**

Lognormal distribution with parameters:

Geometric Mean	70.00
Geometric Std. Dev.	1.30

Selected range is from 0.00 to +Infinity



Correlated with:

meal size (kg) (C5)	0.70

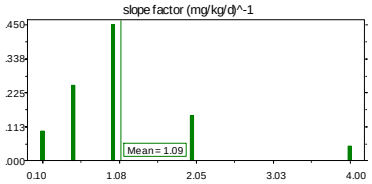
Appendix 2.1

PCB Risk from fish ingestion

All risk coefficients assumed to be uncertain

Assumption: slope factor (mg/kg/d)^-1 **Cell: J5**

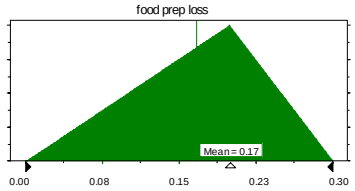
Custom distribution with parameters:		Relative Prob.
Single point	0.10	0.100000
Single point	0.50	0.250000
Single point	1.00	0.450000
Single point	2.00	0.150000
Single point	4.00	0.050000
Total Relative Probability		1.000000



Assumption: food prep loss **Cell: B10**

Triangular distribution with parameters:	
Minimum	0.00
Likeliest	0.20
Maximum	0.30

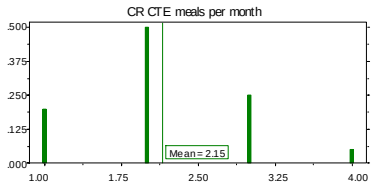
Selected range is from 0.00 to 0.30



Assumption: CR CTE meals per month **Cell: D10**

Custom distribution with parameters:		Relative Prob.
Single point	1.00	0.200000
Single point	2.00	0.500000
Single point	3.00	0.250000
Single point	4.00	0.050000
Total Relative Probability		1.000000

Assumption: CR CTE meals per month (cont'd) **Cell: D10**

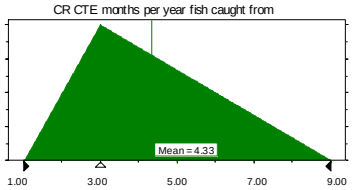


Assumption: CR CTE months per year fish caught from **Cell: E10**

Triangular distribution with parameters:

Minimum	1.00
Likeliest	3.00
Maximum	9.00

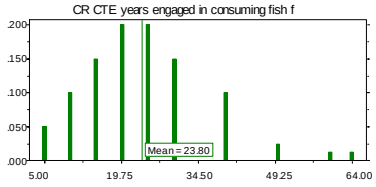
Selected range is from 1.00 to 9.00



Assumption: CR CTE years engaged in consuming fish f **Cell: G10**

Custom distribution with parameters:

		Relative Prob.
Single point	5.00	0.050000
Single point	10.00	0.100000
Single point	15.00	0.150000
Single point	20.00	0.200000
Single point	25.00	0.200000
Single point	30.00	0.150000
Single point	40.00	0.100000
Single point	50.00	0.025000
Single point	60.00	0.012500
Single point	64.00	0.012500
Total Relative Probability		1.000000



Appendix 2.1

PCB Risk from fish ingestion

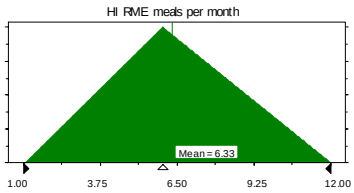
All risk coefficients assumed to be uncertain

Assumption: HI RME meals per month **Cell: D17**

Triangular distribution with parameters:

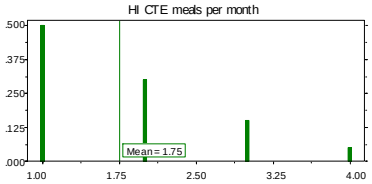
Minimum	1.00
Likeliest	6.00
Maximum	12.00

Selected range is from 1.00 to 12.00



Assumption: HI CTE meals per month **Cell: D22**

Custom distribution with parameters:		Relative Prob.
Single point	1.00	0.500000
Single point	2.00	0.300000
Single point	3.00	0.150000
Single point	4.00	0.050000
Total Relative Probability		1.000000



Assumption: RfD (mg/kg/d) **Cell: J17**

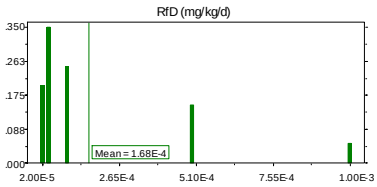
Custom distribution with parameters:		Relative Prob.
Single point	2.00E-05	0.200000
Single point	4.00E-05	0.350000
Single point	1.00E-04	0.250000
Single point	5.00E-04	0.150000
Single point	1.00E-03	0.050000
Total Relative Probability		1.000000

Appendix 2.1

PCB Risk from fish ingestion

All risk coefficients assumed to be uncertain

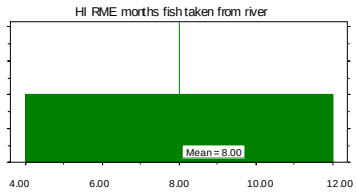
Assumption: RfD (mg/kg/d) (cont'd) **Cell: J17**



Assumption: HI RME months fish taken from river **Cell: E17**

Uniform distribution with parameters:

Minimum	4.00
Maximum	12.00

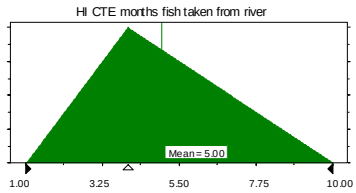


Assumption: HI CTE months fish taken from river **Cell: E22**

Triangular distribution with parameters:

Minimum	1.00
Likeliest	4.00
Maximum	10.00

Selected range is from 1.00 to 10.00



End of Assumptions

Appendix 2.2

PCB Risk from fish ingestion

Risk coefficients held constant at EPA defaults

Crystal Ball Report

Simulation started on 11/17/03 at 9:13:03

Simulation stopped on 11/17/03 at 9:13:06

Forecast: RME risk **Cell: K5**

Summary:

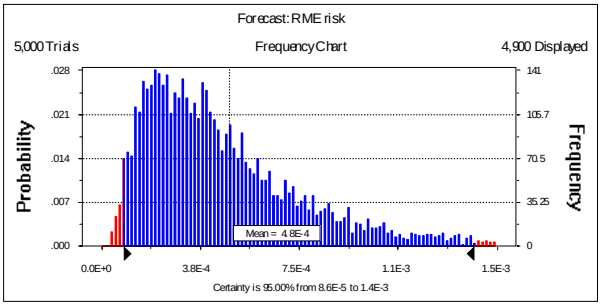
Certainty Level is 95.00%

Certainty Range is from 8.6E-5 to 1.4E-3

Display Range is from 0.0E+0 to 1.5E-3

Entire Range is from 2.7E-5 to 3.7E-3

After 5,000 Trials, the Std. Error of the Mean is 5.1E-6



Percentiles:

Percentile	Value
0.0%	2.7E-05
2.5%	8.6E-05
5.0%	1.1E-04
50.0%	3.9E-04
95.0%	1.2E-03
97.5%	1.4E-03
100.0%	3.7E-03

End of Forecast

Appendix 2.2

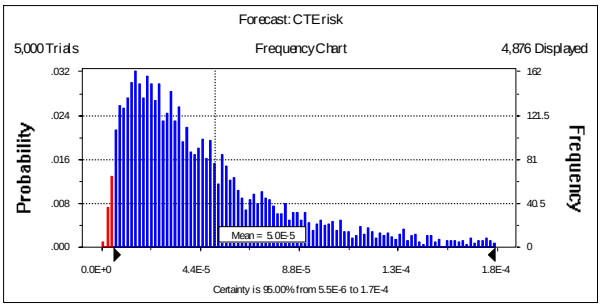
PCB Risk from fish ingestion

Risk coefficients held constant at EPA defaults

Forecast: CTE risk **Cell: K10**

Summary:

Certainty Level is 95.00%
Certainty Range is from 5.5E-6 to 1.7E-4
Display Range is from 0.0E+0 to 1.8E-4
Entire Range is from 1.0E-6 to 4.8E-4
After 5,000 Trials, the Std. Error of the Mean is 6.6E-7



Percentiles:

Percentile	Value
0.0%	1.0E-06
2.5%	5.5E-06
5.0%	7.4E-06
50.0%	3.6E-05
95.0%	1.4E-04
97.5%	1.7E-04
100.0%	4.8E-04

End of Forecast

Appendix 2.2

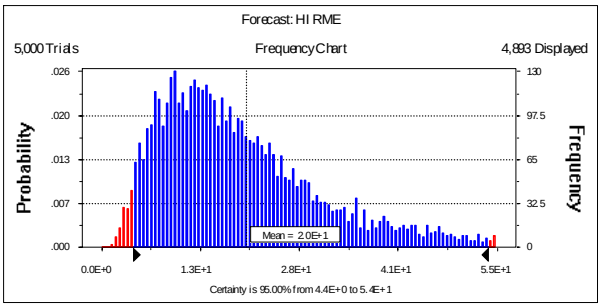
PCB Risk from fish ingestion

Risk coefficients held constant at EPA defaults

Forecast: HI RME **Cell: K17**

Summary:

Certainty Level is 95.00%
Certainty Range is from 4.4E+0 to 5.4E+1
Display Range is from 0.0E+0 to 5.5E+1
Entire Range is from 1.1E+0 to 1.3E+2
After 5,000 Trials, the Std. Error of the Mean is 1.9E-1



Percentiles:

Percentile	Value
0.0%	1.1E+00
2.5%	4.4E+00
5.0%	5.4E+00
50.0%	1.7E+01
95.0%	4.6E+01
97.5%	5.4E+01
100.0%	1.3E+02

End of Forecast

Appendix 2.2

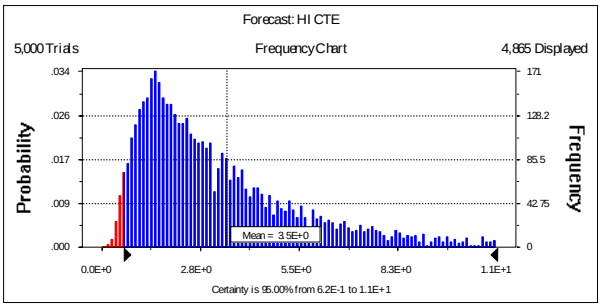
PCB Risk from fish ingestion

Risk coefficients held constant at EPA defaults

Forecast: HI CTE **Cell: K22**

Summary:

Certainty Level is 95.00%
Certainty Range is from 6.2E-1 to 1.1E+1
Display Range is from 0.0E+0 to 1.1E+1
Entire Range is from 1.7E-1 to 3.0E+1
After 5,000 Trials, the Std. Error of the Mean is 4.0E-2



Percentiles:

Percentile	Value
0.0%	1.7E-01
2.5%	6.2E-01
5.0%	7.8E-01
50.0%	2.6E+00
95.0%	9.0E+00
97.5%	1.1E+01
100.0%	3.0E+01

End of Forecast

Appendix 2.2

PCB Risk from fish ingestion

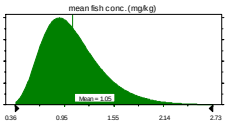
Risk coefficients held constant at EPA defaults

Assumptions

Assumption: mean fish conc. (mg/kg) **Cell: A5**

Lognormal distribution with parameters:

Geometric Mean	1.00
Geometric Std. Dev.	1.40

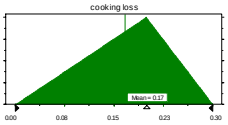


Selected range is from 0.00 to +Infinity

Assumption: cooking loss **Cell: B5**

Triangular distribution with parameters:

Minimum	0.00
Likeliest	0.20
Maximum	0.30

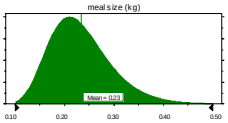


Selected range is from 0.00 to 0.30

Assumption: meal size (kg) **Cell: C5**

Lognormal distribution with parameters:

Geometric Mean	0.23
Geometric Std. Dev.	1.30



Selected range is from 0.00 to +Infinity

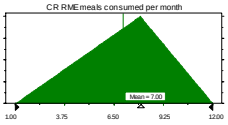
Correlated with:

Body Weight (kg) (I5)	0.70

Assumption: CR RME meals consumed per month **Cell: D5**

Triangular distribution with parameters:

Minimum	1.00
Likeliest	8.00
Maximum	12.00



Selected range is from 1.00 to 12.00

Appendix 2.2

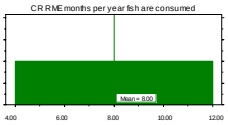
PCB Risk from fish ingestion

Risk coefficients held constant at EPA defaults

Assumption: CR RME months per year fish are consumed **Cell: E5**

Uniform distribution with parameters:

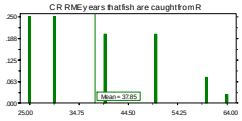
Minimum	4.00
Maximum	12.00



Assumption: CR RME years that fish are caught from R **Cell: G5**

Custom distribution with parameters:

		Relative Prob.
Single point	25.00	0.250000
Single point	30.00	0.250000
Single point	40.00	0.200000
Single point	50.00	0.200000
Single point	60.00	0.075000
Single point	64.00	0.025000
Total Relative Probability		1.000000



Assumption: Body Weight (kg) **Cell: I5**

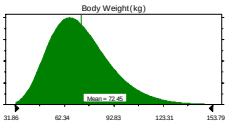
Lognormal distribution with parameters:

Geometric Mean	70.00
Geometric Std. Dev.	1.30

Selected range is from 0.00 to +Infinity

Correlated with:

meal size (kg) (C5)	0.70



Appendix 2.2

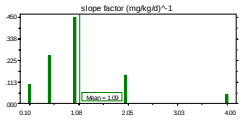
PCB Risk from fish ingestion

Risk coefficients held constant at EPA defaults

Assumption: slope factor (mg/kg/d)^-1 **Cell: J5**

Custom distribution with parameters:		Relative Prob.
Single point	0.10	0.100000
Single point	0.50	0.250000
Single point	1.00	0.450000
Single point	2.00	0.150000
Single point	4.00	0.050000
Total Relative Probability		1.000000

**** Frozen Assumption ** value used was 2.00**



Assumption: food prep loss **Cell: B10**

Triangular distribution with parameters:	
Minimum	0.00
Likeliest	0.20
Maximum	0.30

Selected range is from 0.00 to 0.30

Assumption: CR CTE meals per month **Cell: D10**

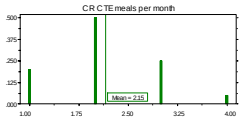
Custom distribution with parameters:		Relative Prob.
Single point	1.00	0.200000
Single point	2.00	0.500000
Single point	3.00	0.250000
Single point	4.00	0.050000
Total Relative Probability		1.000000

Appendix 2.2

PCB Risk from fish ingestion

Risk coefficients held constant at EPA defaults

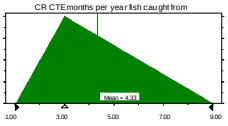
Assumption: CR CTE meals per month (cont'd) **Cell: D10**



Assumption: CR CTE months per year fish caught from **Cell: E10**

Triangular distribution with parameters:

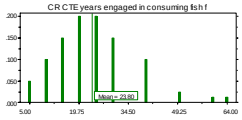
Minimum	1.00
Likeliest	3.00
Maximum	9.00



Selected range is from 1.00 to 9.00

Assumption: CR CTE years engaged in consuming fish f **Cell: G10**

Custom distribution with parameters:		Relative Prob.
Single point	5.00	0.050000
Single point	10.00	0.100000
Single point	15.00	0.150000
Single point	20.00	0.200000
Single point	25.00	0.200000
Single point	30.00	0.150000
Single point	40.00	0.100000
Single point	50.00	0.025000
Single point	60.00	0.012500
Single point	64.00	0.012500
Total Relative Probability		1.000000



Appendix 2.2

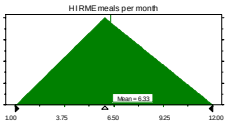
PCB Risk from fish ingestion

Risk coefficients held constant at EPA defaults

Assumption: HI RME meals per month **Cell: D17**

Triangular distribution with parameters:

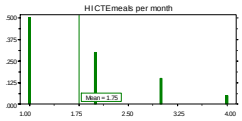
Minimum	1.00
Likeliest	6.00
Maximum	12.00



Selected range is from 1.00 to 12.00

Assumption: HI CTE meals per month **Cell: D22**

Custom distribution with parameters:		Relative Prob.
Single point	1.00	0.500000
Single point	2.00	0.300000
Single point	3.00	0.150000
Single point	4.00	0.050000
Total Relative Probability		1.000000



Assumption: RfD (mg/kg/d) **Cell: J17**

Custom distribution with parameters:		Relative Prob.
Single point	2.00E-05	0.200000
Single point	4.00E-05	0.350000
Single point	1.00E-04	0.250000
Single point	5.00E-04	0.150000
Single point	1.00E-03	0.050000
Total Relative Probability		1.000000

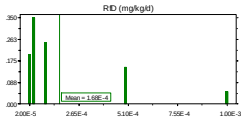
**** Frozen Assumption ** value used was 2.00E-5**

Appendix 2.2

PCB Risk from fish ingestion

Risk coefficients held constant at EPA defaults

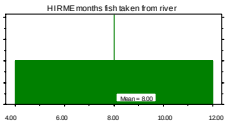
Assumption: RfD (mg/kg/d) (cont'd) **Cell: J17**



Assumption: HI RME months fish taken from river **Cell: E17**

Uniform distribution with parameters:

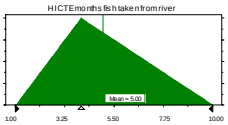
Minimum	4.00
Maximum	12.00



Assumption: HI CTE months fish taken from river **Cell: E22**

Triangular distribution with parameters:

Minimum	1.00
Likeliest	4.00
Maximum	10.00



Selected range is from 1.00 to 10.00

End of Assumptions