

Peer Review of the Hudson River PCBs Human Health & Ecological Risk Assessments

Premeeting Comments

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**PEER REVIEW FOR THE HUDSON RIVER PCBs
HUMAN HEALTH RISK ASSESSMENT**

OVERALL RECOMMENDATIONS

The present EPA risk assessment provides sufficient information to conclude that PCBs released from the General Electric facility into the Upper Hudson River are a regulatory concern. The human health risk assessment does not provide sufficient information to enable evaluation of the potential health risk to humans under baseline conditions. Base-line conditions imply that no restrictions are in place to prevent people from utilizing the Upper Hudson River for sport fishing, harvesting of other aquatic organisms for food, or as a commercial fishery for striped bass.

In general, the toxicity coefficients obtained from the IRIS database are intended by EPA to be conservatively biased, i.e., in the presence of uncertainty their use will err on the safe side to ensure that real people exposed to PCBs are unlikely to suffer harm. The EPA human health risk assessment, however, does not indicate at what concentrations or exposure levels increased levels of harm might be expected to occur. Therefore, although information is adequate to conclude that PCBs are of a regulatory concern, information is inadequate to evaluate the uncertainty associated with anticipated health impacts. To properly assess the effectiveness of risk proposed reduction alternatives during the analysis of the feasibility of remediation, information is needed on the uncertainty in the toxicity coefficients for PCBs, the concentrations of PCBs in fish harvested at future dates, and concentrations of other cancer causing and non-carcinogenic substances in various environmental media of the Upper Hudson River.

The EPA human health risk assessment does not adequately address uncertainty in quantifying health risk. Areas where the uncertainty analysis is deficient are as follows:

- (1) *Individuals who would be exposed to contaminated environmental media in the Upper Hudson River are exposed to much more than just PCBs. They are also exposed to agricultural chemicals and to radionuclides introduced by various facilities and by atmospheric testing of nuclear weapons. All of these add to the overall health burden, and it is this cumulative burden that should be assessed. The current risk assessment focuses only on health the impact of exposure to PCBs.*

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- (2) *The assessment is focused on projected PCB concentrations in fish averaged for the entire 40-mile reach of the Upper Hudson River. Uncertainties on these projected average concentrations are not presented in the report.* In the March 2000 Responsiveness Summary, the projected average concentrations in the various species of fish are too narrow to be plausible. The assessment of uncertainty is limited to a comparison of model predictions with past observed concentrations in various fish species. The uncertainty associated with forecasting PCB concentrations in fish over time is not considered.

The comments in the EPA human health risk assessment about the differences between the uncertainty in the mean concentration of a sample versus the uncertainty in the mean concentration in a model prediction is technically incorrect. Both measured and modeled mean concentrations have associated uncertainty, and this uncertainty should be quantified and reported.

- (3) *Averaging concentrations and exposures over the entire reach of the Upper Hudson River is inappropriate.* Clean-up options will be designated for various subreaches of the river, and the HHRA should target those subreaches. In addition, the population exposed to the entire Upper Hudson River would likely be quite large, much larger than the 10,000 anglers referred to in the HHRA documents. It is easy to imagine that the number of people consuming fish out of the Upper Hudson River would include the families of anglers, families of those who harvest fish but who are not licensed, and those who would purchase fish from commercial fisheries, if such fisheries were to go into operation under baseline conditions. Therefore, targeting the upper 95th percentile of a very large population has the potential to substantially underestimate exposure to a significant subpopulation of that group. For example, assume that the total number of people who consume fish from the Upper Hudson River is on the order of 100,000 individuals. The top 5% of the distribution of that population would still entail a population of 5,000 individuals. The top 1% would include a population of 1,000 individuals. Therefore, I believe it is more appropriate to focus on subpopulations that would utilize subreaches of the Upper Hudson River, rather than the entire 40-mile reach. In addition, I would separately assess the exposure and risk for reference individuals characterized as casual, average, and maximal users of the Upper Hudson River, as opposed to treating inter-individual variability as a random process.

- (4) *The Monte Carlo analysis is not used to address uncertainty.* Interindividual variability among licensed anglers is simulated as a stochastic process. Instead of subdividing the population into those who would be most likely to consume moderate to average amounts of fish, and those likely to consume maximum amounts of fish, the Monte Carlo analysis simply draws directly on empirical results from slightly more than 200 respondents to a 1991 angler survey conducted for upstate New York. This survey was conducted over a broad region of the state. Sites included many that were subjected to fishing advisories. Thus, the database used to drive the Monte Carlo analysis is not directly relevant to the population of concern who would be consuming fish out of the Upper Hudson River. The degree to which the empirical database is relevant to the Upper Hudson River has been discussed, but the potential for bias is not included in the Monte Carlo analysis.

Many other sources of information that would lead to an expression of interindividual variability have not been included in the Monte Carlo analysis, including

- (a) the size of an average meal per person,
- (b) the amount of fish that would be caught from other locations besides the Upper Hudson River,
- (c) the likelihood that there would be variability in food preparation losses from one meal to another, and
- (d) the likelihood that food consumption patterns would change over a period of 7 to 40 years.

The 1991 angler survey itself was a recall study for a single fishing season, and its applicability for an average person over periods of 7 to 40 years is not discussed. I anticipate that the extent of interindividual variability as well as the amount of fish consumed at the upper percentiles of the true frequency distribution has been substantially overestimated.

The sensitivity analysis that is performed to indicate the uncertainty in the Monte Carlo analysis does not fully capture uncertainty. Many of the assumptions made are inappropriate. For example, fish concentrations are assumed to occur entirely at one segment of the river or another. This would be appropriate if one were to assess the interindividual variability in exposure for a subreach. However, the uncertainty analysis should include an estimate of the limits of credibility on the average concentrations in various fish species caught from each subreach of concern.

Food losses of PCBs as a result of cooking and preparation of fish is treated in the present EPA assessment as an uncertain variable. In actuality, losses due to food preparation should be treated as both a frequency distribution representing individual variability in food preparation, and as a probability distribution representing the state of knowledge in the average amount of loss for the population as a whole. One would expect to have differences in losses of PCBs from meal to meal and from year to year. The assumption of 0% loss for all meals over all years is implausible.

- (5) *Uncertainty in cancer and non-cancer health endpoints should be included explicitly.* Although there is EPA policy guidance that discourages risk assessors from explicitly considering the uncertainty in cancer slope factors (CSFs) and Reference Doses (RfDs), the risk calculation cannot be considered to be scientifically defensible until uncertainty in the toxicity coefficients is properly accounted for in the human health assessment.

It is EPA policy to allow for the expression of uncertainty about toxicity in ecological risk assessment. It is thus inconsistent to exclude the evaluation of uncertainty in the toxicity coefficients from the human health risk assessment of PCBs.

If EPA policy mandates the exclusion of the evaluation of uncertainty in the toxicity coefficients, then the human health risk assessment ceases to become a true risk assessment, and instead is restricted to a regulatory compliance calculation.

A detailed evaluation of potential uncertainty in the PCB toxicity coefficients is important because of the potential exposure of very large numbers of individuals. This is the case for the Upper Hudson River. Under baseline conditions, there would be no restrictions to the access and harvesting of fish. Baseline conditions should include sport fishing, unlicensed fishing, harvesting of other biota, and commercial fishing.

I thus conclude that the present EPA HHRA, although adequate for identifying a situation of regulatory concern, is inadequate as a scientifically defensible risk assessment. The present risk assessment may either over- or understate the true risk depending upon what information is and what information is not included in the analysis.

The following sections give my answers to specific questions that have been posed by EPA:

EPA Question 1, Hazard Identification and Dose Response

The human health risk assessment uses the most recent values of the cancer slope factors and noncancer risk RfDs listed in the Integrated Risk Information System (IRIS). This is the agency's database of consensus toxicity values. However, IRIS clearly states that risk assessors may use more recent data when such data are available.

The use of IRIS values of PCB toxicity is appropriate for indicating the presence of contamination that warrants regulatory concern. The cancer slope factors and RfDs, however, do not indicate the actual risk of cancer or of noncancer endpoints, because the uncertainties associated with these quantities are not included as a part of the risk assessment. Although RfDs are derived from NOAEL's or LOAEL's by a factor called an "uncertainty factor (UF)," these UF values do not disclose uncertainty. Values of UF are much like safety factors. Their use in the presence of uncertainty leads to a Reference Dose (RfD) to result in an adverse health effect. However, a noncancer Hazard Quotient for PCBs that exceeds 1.0 does not necessarily indicate that there is a significant health threat.

The most current RfDs used for PCBs include an uncertainty factor of 100 to 300. The potential for these values of UF to be overly protective should be assessed. For example, instead of multiple factors of 10 from subchronic to chronic exposure, and from animal studies to human studies, what if a factor of 3 had been used? Recent papers by Swartout et al. 1998, and Price et al. 1997a and 1997b, should be reviewed on this subject. Furthermore, it might be more appropriate to assess the combined amount of exposure to PCBs and other toxic substances, in order to look at the *total Hazard Index* and the *total cancer risk* from utilization of the Upper Hudson River, which would include exposure to water, sediment, and aquatic biota.

In conclusion, IRIS toxicity values of CSFs and RfDs are adequate for indicating levels of regulatory concern. They are in adequate for indicating levels above which human health risk will be significant or intolerable. The present risk assessment may either over- or understate the true risk depending upon what information is and what information is not included in the analysis.

EPA Question 2: EPA asked the reviewers to comment on whether the specification of central tendency and reasonably maximally exposed individual consumption rates of 4 and 31.9 grams per day, equivalent to approximately 6 and 51 half-pound meals per year, respectively, are reasonable to capture interindividual differences in exposure for point-estimate calculations.

My answer is yes, this is reasonable. Six fish meals per year from the Upper Hudson River can be attributed to any one of a large number of representative individuals that could utilize the Upper Hudson River. Fifty-one half-pound meals per year also appears to be reasonable as a maximum estimate. This maximum estimate could be achieved by someone eating much more than one fish meal per week during the fishing season, and relatively few fish meals during the rest of the year, or by someone consuming multiple fish meals per week, but only harvesting a few of those meals from the Upper Hudson River. I consider both numbers to be adequate for point-estimate calculations.

In fact, for the Monte Carlo calculation, if one is estimating the uncertainty in the exposure and risk to reference individuals, it would be appropriate to fix the dietary intake for the representative (or reference individual). The values of 4 and 31.9 grams per day would then be appropriate for use as fixed reference values. The uncertainty in risk would then be restricted to the uncertainty in the toxicity coefficients, the uncertainty in the concentration of the PCBs in the aquatic media, and the uncertainty associated with losses of PCBs due to food preparation.

EPA Question 3: Along the 40-mile reach of the Upper Hudson River, the EPA has assumed central tendency and reasonably maximal exposure durations of 12 and 40 years, respectively, for a cancer causing substance, and 7 years for exposure to noncancer causing substances.

I believe that these values for point-estimate calculations are appropriate; however, because of the very large population that could be affected along the 40-mile reach, it may be more appropriate to also consider individuals who would spend their entire lifetime accessing fish from this region. The size of the population that potentially could utilize fish from this region may exceed tens of thousands of individuals. For this reason it would be appropriate to consider individuals who could potentially be in residence for a period much longer than 40 years.

Much more important, however, is the fact that individuals won't necessarily harvest all of their fish from the Upper Hudson River. Some consideration should be given to the situation in which a residential angler living for more than 40 years along the Upper Hudson River actually consumes some amount of fish from other locations.

EPA Question 4: PCB concentrations in fish have declined in past decades, and the decline is expected to continue into the future. To evaluate noncancer effects for the maximally exposed individual, EPA used point concentrations in each medium (water, sediment, and fish), based on the average concentration forecast over the next 7 years from 1999 to 2006. For point concentrations for exposure, the central tendency exposure, EPA used the average of the concentrations forecast over 12 years, which is the 50th percentile of the residence duration developed from population mobility data. In addition, for completeness, EPA averaged the exposure concentration over 40 years to evaluate noncancer hazards for the same time period over which cancer risk was calculated. The review team has been asked to comment on whether this approach adequately addresses noncancer health hazards to the central tendency and reasonably maximally exposed individuals.

I believe it is appropriate to specify a reasonable midpoint for averaging concentrations in fish for a short-term time duration of a 7-year exposure. However, because the Hazard Quotients for PCBs are so large, it is also appropriate to look at subchronic exposures that may affect critical population subgroups. In this case, exposure durations as short as one year should be considered. During a one-year time period, a person could be at much lower body weight than is currently assumed in the risk assessment. The uncertainty in fish concentration at a given location, as mentioned previously, is not adequately expressed in the present EPA human health risk assessment.

My personal preference would be for the assessment to focus on subreaches of the Upper Hudson River and for the uncertainty in the average fish concentrations to be determined for those subreaches. To average the assessment over the entire 40-mile stretch of the river is inappropriate, and there is the possibility that critical subgroups of the population will be missed because of the very large number of people potentially exposed if no restrictions were placed on public utilization of this aquatic resource.

EPA Question 5, The Monte Carlo Analysis: We were asked to discuss whether the Monte Carlo analysis used in the HHRA makes appropriate use of the available data, uses credible assumptions, and adequately addresses variability and uncertainty associated with the fish ingestion pathway; the last item would include defining the angler population, PCB exposure concentrations, ingestion rates, exposure durations, cooking losses, etc.

Having reviewed the Monte Carlo calculations in detail, I find that they capture neither the interindividual variability of potential exposure in risk, nor the uncertainty about a reference average individual or a reference maximally exposed individual.

Monte Carlo calculations serve two distinctly different purposes. The first and perhaps most important purpose is to propagate uncertainty through risk assessment algorithms (when it is difficult or impossible to propagate such uncertainty using algebraic formulae). When Monte Carlo techniques are used to propagate uncertainty, the present state of knowledge is expressed as a subjective probability distribution given all of the evidence available (NCRP, 1996; IAEA, 1989; Cullen and Frey, 1999; National Research Council, 1994). A subjective probability distribution is specified for each variable that can be considered to be a true but unknown quantity.

In the present EPA human health risk assessment, the Monte Carlo calculation is not used to propagate uncertainty, but instead to simulate interindividual variability of exposure. Interindividual variability is assumed to be a stochastic, random process, which, of course, is not the case. There are distinct reasons why some individuals choose to eat more fish than do others.

A more practical approach would be to specify several reference receptors and use Monte Carlo techniques to quantify uncertainty about the health risk to those reference human receptors. At the very least, the Monte Carlo techniques should be used to quantify uncertainty for a reasonable average individual and for a reasonable maximally exposed individual.

In this particular assessment, the Monte Carlo calculation utilizes the average fish concentration of PCBs for a 40-mile reach. This average value is given without uncertainty. The Monte Carlo calculation also assumes that a dietary survey for upstate New York for free-flowing fresh water is directly applicable to the case of the Upper Hudson River. This fish survey is for licensed anglers, of whom 221 responded out of 1000 questionnaires that were distributed.

It is well known that fish surveys that are based on individual recall are biased. The tendency is to overestimate the amount of fish caught and consumed. The extent to which the distribution defined by 221 individuals may be biased has not been assessed. Instead, the 221 values are used verbatim, after unreasonable values at the low and high ends have been censored by the authors of the risk assessment. The censored values include those who reported more than 1,000 fish meals during the year and those who reported no fish meals during the year.

Several items are missing as the result of the empirical use of the 1991 fish survey results: (a) The extent to which the average value for this distribution is biased high, (b) the extent to which the upper end of the distribution is biased high, (c) the extent to which the relative variability reported from the distribution may be biased high, and (d) the extent to which a single-year recall survey is representative of a 7 to 40-year average also needs to be assessed.

The failure of the Monte Carlo calculation to consider uncertainty in fish concentrations, uncertainty in the amount of fish consumed that is actually caught or harvested from the Upper Hudson River, and the uncertainty associated with the use of empirical survey data to represent the dietary pattern of the population that might consume fish from the Upper Hudson River over a period of 7 to 40 years, renders the results of the Monte Carlo analysis uninterpretable.

Simulating individual variability in a large population is a daunting task. Relevant data must either be available or the uncertainty associated with partially relevant data must be explicitly considered. My present evaluation is that the high-end exposure is more than likely overestimated for the 95th percentile, but the 99th percentile and above are potentially underestimated because the dietary survey is truncated as a result of only 221 respondents. I believe the Monte Carlo calculation for interindividual variability should include the total number of people potentially exposed so that one can judge how many people could have a risk above a regulatory level of concern, and how many people could have risk extending into a region of a likely health threat.

Of course, the present Monte Carlo distribution does not include the variability or uncertainty in cancer slope factors and RfDs. Thus, the risk assessment is interpretable only from the standpoint of regulatory concerns, not from the standpoint of potential health risk. Because exposure to multiple contaminants in the aquatic medium has not been taken into account, it is virtually

impossible at present to make an overall assessment of health risk other than to state that if fishing restrictions were to be removed, it is very likely that the majority of the population who would consume fish from the Upper Hudson River would be exposed to PCBs at a level that would warrant regulatory concern.

EPA Question 6. The adequacy of EPA's evaluation and use of existing angler surveys in the Monte Carlo analysis of the fish ingestion pathway:

I touched upon much of this in my answer to the above question; however, I think that it is important that the potential sources of bias in any fish angler survey be considered explicitly. If the objective of the assessment is to estimate the exposure to the entire population who has consumed fish from the Upper Hudson River, then it is important to evaluate the extent to which angler survey data may be biased and might either under- or overstate the amount of actual fish consumption that occurs.

I believe the upper end of the distribution can be grossly overstated from the true values that would occur over a 10- to 40-year time period. I also believe that the interindividual variability, (or the geometric standard deviation) of the distribution, is overstated because the survey is based on individual recall for a relatively short time period.

For cancer-causing substances, I believe it is more important to look at the uncertainty on the *average* fish consumption than it is to look at the uncertainty on the median. The median in this case will understate the total cancer risk to this population. The total cancer risk (number of cases in the population), is a product of the *arithmetic mean* exposure in the population, the size of the population, and the cancer slope factor.

The sensitivity analysis, which uses (a) a range of variables of fish concentrations in the river, (b) a range of loss fractions due to cooking and preparing fish prior to human consumption, and (c) alternative databases for angler surveys, only partially captures the uncertainty that is present. The cooking loss variable is more appropriately expressed as a source of inter-individual variability of exposure. The uncertainty about the average loss due to cooking over a 10- to 40-year lifetime history of individuals capturing fish from the Upper Hudson River is much less than the range of 0 to 40% assumed in the HHRA.

EPA Question 7, Risk Characterization: Risk characterization in the human health risk assessment summarizes the cancer risk and noncancer hazards to individuals who may be exposed to PCBs in the Upper Hudson River. The reviewers were asked to comment on whether the risk characterization adequately estimates the relative cancer risks and noncancer hazards for each pathway and exposed population. Have major uncertainties been identified and adequately considered, and have the exposure assumptions been described sufficiently?

Again, the HHRA assessment adequately discloses that exposure to PCBs is of regulatory concern. However, to evaluate the health risk from exposure to PCBs and other contaminants existing in the Upper Hudson River, far more information is needed than is available at this time. The uncertainty associated with cancer slope factors and RfDs should be taken into account explicitly in order for the uncertainty in the risk estimates to be properly expressed. Without such uncertainty estimates, the risk of making the wrong decision when determining the feasibility of cleanup cannot be evaluated.

My conclusion is that the risk characterization, although adequate to indicate a situation of regulatory concern, is clearly inadequate for expressing the degree of health risk that may be present under baseline conditions. The true health risk may be grossly overstated in some aspects, and in other aspects, it may be understated. The extent to which it is either overstated or understated cannot be evaluated given the information at hand.

EPA GENERAL QUESTIONS

EPA General Question 1, Clarity and Transparency of the HHRA

This HHRA is consistent with other regulatory documents that I have reviewed; however, this and the other documents all suffer from being written for an audience of regulators, not for an audience of individuals who might be concerned with potential health hazards associated with consuming PCB-contaminated fish from the Upper Hudson River.

My recommendation is that the overall report be carefully edited, the use of regulatory acronyms and jargon eliminated, and the report re-written so that an interested individual can readily comprehend the content of this report.

Many of the figures are presented on a linear arithmetic scale. They should instead be plotted on a logarithmic scale (but retaining the arithmetic units). The use of the logarithmic scale is most appropriate for evaluating *relative differences* (as opposed to absolute differences) in trends over time. The probability plots that are presented in the present HHRA on fish consumption rates from various angler surveys are virtually unreadable to all but statisticians. These plots should be redrawn showing the number of fish meals on the y-axis (preferably using a log scale), and showing the relative probability or cumulative probability on the x-axis. Software is readily available that will allow a more transparent presentation of probability plots.

EPA General Question 2, Provide any other comments or concerns about strengths and weaknesses of the HHRA

I would like to understand how exposure to other contaminants, in combination with the ingestion of PCBs, affect the total cancer risk, as well as the noncancer health risk, to individuals consuming fish from the Upper Hudson River.

I would give a much lower priority to the use of Monte Carlo calculations to simulate interindividual variability, unless it is the objective of the assessment to indicate the fraction of the total population of exposed individuals that would be potentially at risk. If this is the case, then the empirical use of angler surveys are clearly not relevant without some correction for bias. The relevant population of concern are those individuals who would consume fish from the river

under baseline conditions, including the families of licensed anglers, unlicensed individuals who utilize the Hudson River as source of food, and those who eat fish from commercial operations. The latter would involve the consumption of fish shipped to restaurants, supermarkets, and so forth.

I believe the strength of the present human health risk assessment is to demonstrate that even under the most optimistic conditions, PCBs in the Upper Hudson River present a regulatory concern. I believe the most pronounced weakness is the failure to disclose uncertainty in individual exposures and health risk.

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Appendix of Detailed Comments
Volume 2F – Human Health Risk Assessment Hudson River PCBs Reassessment RI/FS

Page ES-2, Exposure Assessment

The RME and CTE capture differences between the high end and the averages, although CTE may be underestimated. Uncertainty about the CTE and RME needs to be quantified. Estimates of RME and CTE need to be made for each location of concern as opposed to the entire 40-mile stretch of river.

Pages ES 2-3, Ingestion of Fish

The extent to which a recall survey of 221 individuals (licensed anglers) is applicable to the population of individuals potentially consuming fish from the Upper Hudson is questionable. Also questionable is the representativeness of the data for exposure durations of 7 to 41 years.

Assumptions about PCB losses during cooking and the fraction of sport fish consumed that come from the Upper Hudson River is questionable. Especially considering a 7- to 40-year exposure duration.

Younger ages may be important if a sub-chronic exposure to PCB is considered important. For non-cancer risk, background PCBs in diet should be accounted for. Uncertainty in UF should be expressed in the risk analysis.

Specific Remarks

- 1) There is a need to simulate random variability of the high end of the distribution separately, accounting for all potential aspects of partial relevancy of data.
- 2) The population should be all those consuming fish from the Upper Hudson River, not just licensed anglers.
- 3) The 1-D Monte Carlo simulations do not consist of 10,000 simulated anglers in that an empirical distribution of New York anglers is used with over 221 data points.
- 4) The fraction of fish caught beyond the Upper Hudson River should be included as a variable.
- 5) None of the 72 alternative distributions address uncertainty; they are simply alternative data sources to extreme assumptions about fish concentrations at a fixed location as opposed to the average for the entire reach of 40 miles. Variability in losses due to food preparation are ignored and artificially treated as a source of uncertainty.

- 6) When the objective is to simulate the frequency of real exposures in a real population of individuals, the best estimate of central tendency is the arithmetic mean, not the 50th percentile.

- 7) When the objective is to simulate a true frequency distribution of risk, the target population must be rigorously defined and the size of the exposed population estimated. It is presently not known if the Monte Carlo simulation refers to a population size of a few hundred or 10's of thousands of individuals assuming that there would be no restrictions on the harvesting of fish.

To estimate uncertainty, I recommend a 1-D Monte Carlo simulation be performed for the RME and for the CTE at each subreach of the Upper Hudson River.

Page ES-4

Need to state what the results were from Kimbrough et al. (1999).

Since the size of the population representative of the RME is less than 10,000 individuals, this would translate to a low probability of even one excess case of cancer. Again, the CTE for a total population of approximately 100,000 individuals would indicate less than one excess case of cancer. The CTE estimate should be based on the arithmetic mean, not the 50th percentile of the population.

Page ES-4, Risk Characterization

Risk estimates should include a < sign to reflect the fact that the CSF are upper bound estimates.

Uncertainty in RME and CTE risks need to be disclosed.

Cancer risk should be estimated for all chemicals and radionuclides contained in Upper Hudson River Fish, not just PCBs alone, unless it is certain and PCBs are dominant. General bans on fishing may be more health protective than attempts to remediate only for PCBs.

Page ES-5

The Hazard Index should be estimated for all chemicals in food having the potential to induce effects on the same organ or tissue. Background exposures to these chemicals should be added to what is measured in fish tissue from the Upper Hudson River.

Page ES-5, Monte Carlo Estimate

There is a need to show the size of the population that is potentially affected. This Monte Carlo summary is a mere mathematical exercise. There is no rigorous attempt to quantify uncertainty. The table is meaningless. A total revision is recommended.

Uncertainty about variability is not depicted. All data sets used have uncertainty. To say that all fish meals for all persons over 40 years will be taken from one location and subjected to losses of zero percent is unrealistic.

Page ES-6, Table

Need to show uncertainty in UF.

Page ES-6, Comparison of Point Estimate and Monte Carlo Analyses

"For non-cancer hazards, the point estimate RME for fish ingestion (116) falls between the 95th and 99th percentiles of the Monte Carlo base case" this is only because of the assumption of 20% loss for cooking.

Page ES-6, Major Finding of the HHRA

The following statement is not true: "Under the RME scenario for eating fish, the calculated risk is one additional case of cancer for every 1,000 people exposed. This excess cancer risk is 1,000 times higher than USEPA's goal of protection and ten times higher than the highest risk level allowed under Superfund law."

There is a need to assess uncertainty in UF for the following statement: For non-cancer health effects, the RME scenario for eating fish from the Upper Hudson results in a level of exposure to PCBs that is more than 100 times higher than USEPA's reference level (Hazard Index) of one.

Page 2, first paragraph

The baseline risk assessment should include the plausibility of a commercial fishery as existed prior to 1976.

Page 5, 4th paragraph

The following statement is not advised: "...such that the RME can be determined based on estimates from the high-end of the Monte Carlo exposure distributions."

Page 7, Section 2.1.2, Potential Receptors

Without restrictions, how many would consume fish from the Upper Hudson River?

Page 10, Section 2.3 Exposure Point Concentrations

Last two sentences in this section are not true.

Page 13, first paragraph

Variability versus uncertainty.

Page 13, Concentration Averaged Over Locations

Treat each location separately.

Page 35, last paragraph

This is not a risk assessment.

Page 36, second equation

Is not 100%.

Page 37, Section 3.2.1, Fish Ingestion Rate

The first paragraph is not based on the survey.

The last paragraph is not relevant to the assessment question.

Page 39, second paragraph

What is the relevancy?

Page 43, first paragraph

For cancer causing substances, the mean is more relevant than is the median.

Page 46, Single Versus Multiple Waterbodies

This is for the year but not for the 12 to 40 year duration.

Page 48, fourth paragraph

Table should be 3-5.

Page 49, second paragraph

Untrue.

Page 58, second paragraph

Should consider correlation with ingestion.

Table 3-1

There is no uncertainty given for the size of fish meal used.

Table 3-2

Show the mean value.

Table 3-5

Losses due to cooking are both uncertain and variable.

Table 3-16

The information presented is not consistent with the scale of time-averaging of the risk assessment. These distributions do not refer to the 12 to 40 year average. Instead it gives the distribution for the body weight at a given time of measurement. It is not even relevant to the uncertainty in an annual average body weight.

Figure 3-2

Many of the figures are presented on a linear arithmetic scale. They should instead be plotted on a logarithmic scale (but retaining the arithmetic units). The use of the logarithmic scale is most appropriate for evaluating *relative differences* (as opposed to absolute differences) in trends over time. The probability plots that are presented in the present HHRA on fish consumption rates from various angler surveys are virtually unreadable to all but statisticians. These plots should be redrawn showing the number of fish meals on the y-axis (preferably using a log scale), and showing the relative probability or cumulative probability on the x-axis. Software is readily available that will allow a more transparent presentation of probability plots.

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Clarification to Pre-Meeting Comments Upper Hudson River Human Health Risk Assessment Peer Review Harlee Strauss, Ph.D. May 22, 2000

I would like to clarify two statements I made regarding non-cancer dose response and the evaluation of the non-cancer effects of PCBs on young children in my pre-meeting comments (page 74 of the premeeting comment book).

1) In briefly mentioning some of the inadequacies of the toxicity profile (Appendix C), I stated that it did not give the reader as sense of the extent of the database with respect to neurodevelopmental and immunological effects on children. Unfortunately, the parenthetical comment that followed that statement could be misleading. I did not mean to imply there are three cohort studies examining immunological effects. In addition, the consistency of the neurodevelopmental findings is true only in a broad sense of the observations of adverse effects at young ages. Some of the studies yield different results with respect to adverse endpoints observed at a young age and the persistence of these deficits into school age years. The three cohorts I was referring to were the Lake Michigan cohort (c.f., Jacobson and Jacobson, 1996), the North Carolina cohort (c.f., Rogan and Gladen 1991), and the Dutch cohort briefly described in the Appendix C toxicity profile. Based on a recent literature survey, it appears that at least one additional cohort study is underway (c.f., Winneke et al. 1998, regarding a study conducted in Dusseldorf, Germany).

2) I suggested a margin of exposure approach be used to evaluate the potential effects of PCBs on children, and that *in utero*, breast milk and direct consumption exposures be included. I further suggested that the dose should be calculated using a short averaging time and high end concentrations of PCBs in fish because of the critical window of development is likely to be short. I would like to clarify that this method of calculating dose only applies to *in utero* exposure (Note: this approach assumes that transient elevations in blood PCBs due to recent PCB-contaminated fish ingestion is important with respect to toxicity, although the maternal body burden is probably the major determinant to *in utero* exposure if averaged throughout gestation). Breast milk exposures should be based on long term averages as PCB concentrations in breast milk reflect the mother's body burden of PCBs. In addition, it may be appropriate to consider the *in utero* exposure separately as well as in combination, as most (but not all) of the neuro-toxicological effects associated with PCBs in the cohort studies cited above appeared to be associated only with *in utero* exposures.

References

- Jacobson and Jacobson, 1996. New England J of Med 335:783-9.
Rogan and Gladen, 1991. Ann. Epidemiol 1:407-13.
Winneke et al., 1998. Toxicol Let. 103:423-8.

Peer Review of the Hudson River PCBs Human Health & Ecological Risk Assessments

Premeeting Comments

Saratoga Springs, New York
May 30–June 2, 2000

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Note: Premeeting comment materials have been reproduced as received.

Hudson River PCBs Site Reassessment RI/FS

Risk Assessments

Peer Review 4

Background for Peer Review 4

The peer review for the Human Health Risk Assessment and the Ecological Risk Assessment is the fourth and final peer review that the U.S. Environmental Protection Agency (USEPA) is convening for the major scientific and technical work products prepared for the Hudson River PCBs site Reassessment Remedial Investigation and Feasibility Study (RI/FS). USEPA previously has peer reviewed the modeling approach (Peer Review 1) and the geochemistry studies (Peer Review 2). The peer review for the computer models of fate, transport, and bioaccumulation of PCBs (Peer Review 3) will conclude on March 28, 2000.

This peer review is comprised of two panels of independent experts: one for the Human Health Risk Assessment and one for the Ecological Risk Assessment. The reviewers are asked to determine whether the risk assessment they review is technically adequate, competently performed, properly documented, satisfies established quality requirements, and yields scientifically valid and credible conclusions. The reviewers are not being asked to determine whether they would have conducted the work in a similar manner.

In making its remedial decision for the PCB-contaminated sediments in the Upper Hudson River, USEPA will answer the three principal study questions that are a focus of the Reassessment RI/FS:

1. When will PCB levels in fish meet human health and ecological risk criteria under continued No Action?
2. Can remedies other than No Action significantly shorten the time required to achieve acceptable risk levels?
3. Could a flood scour sediment, exposing and redistributing buried contamination?

The risk assessments will be used to help address the first two questions. Specifically, the risk assessments will be used in the Feasibility Study to back-calculate to appropriate levels of PCBs in fish to compare various remedial alternatives, including the No Action alternative (i.e., baseline conditions) required by federal Superfund law.

Human Health Risk Assessment Review

Human Health Risk Assessment Charge

The goal of the Human Health Risk Assessment (HHRA) is to evaluate the cancer risks and non-cancer hazards associated with human exposure to PCBs in the Upper Hudson River in the absence of remediation of the PCB-contaminated sediments and any institutional controls, such as the fish consumption advisories that are currently in place (i.e., under baseline conditions). The following documents will be provided to the peer reviewers:

Primary

- Human Health Risk Assessment, Upper Hudson River, August 1999
- Responsiveness Summary for Human Health Risk Assessment, Upper Hudson River, March 2000

References

- Human Health Risk Assessment Scope of Work, July 1998
- Responsiveness Summary for Human Health Risk Assessment Scope of Work, April 1999
- Executive Summary for the Human Health Risk Assessment, Mid-Hudson River, December 1999
- Executive Summary for the Baseline Ecological Risk Assessment, August 1999
- Executive Summary for the Baseline Ecological Risk Assessment for Future Risks in the Lower Hudson River, December 1999
- Executive Summary for the Revised Baseline Modeling Report, January 2000
- Suggested charge questions from the public for the HHRA, February & March 2000

The reference documents listed above are being provided to the reviewers as background information, and may be read at the discretion of the reviewers as time allows. The reviewers are not being asked to conduct a review of any of the background information.

Additional Reassessment RI/FS documents are available on USEPA's website (www.epa.gov/hudson) and/or by request. Additional documents include the following:

- Hudson River Reassessment RI/FS Database, August 1998
- Executive Summaries for other USEPA Reassessment RI/FS Reports
- Peer Review Reports from first two peer reviews
- Responsiveness Summary for first peer review
- New York State Department of Health advisories for chemicals in game and sportfish (www.health.state.ny.us/nysdoh/enviro/fish.htm)

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Specific Questions

Hazard Identification/Dose Response

1. Consistent with its risk assessment guidance (USEPA, 1993), USEPA considered scientific literature on PCB toxicity, both as to cancer and non-cancer health effects, published since the 1993 and 1994 development of the non-cancer reference doses (RfDs) for Aroclor 1016 and Aroclor 1254, respectively, and since the 1996 reassessment of the cancer slope factors (CSFs). Based on the weight of evidence of PCB toxicity and due to the Agency's ongoing reassessment of the RfDs, USEPA used the most current RfDs and CSFs provided in the Integrated Risk Information System (IRIS), which is the Agency's database of consensus toxicity values. The new toxicity studies published since the development of the RfDs and CSFs in IRIS were addressed in the context of uncertainty associated with the use of the IRIS values (see, HHRA, pp. 76-77 and Appendix C). Please comment on the reasonableness of this approach for the Upper Hudson River.

Exposure Assessment

2. Since 1976, the New York State Department of Health has issued fish consumption advisories that recommend "eat none" for fish caught in the Upper Hudson River. To generate a fish ingestion rate for anglers consuming fish from the Upper Hudson River under baseline conditions (i.e., in the absence of the fish consumption advisories), USEPA used data on flowing water bodies in New York State (1991 New York Angler survey, Connelly et al., 1992) to derive a fish ingestion rate distribution. The 50th and 90th percentiles were used for the fish ingestion rates for the central tendency (average) and reasonably maximally exposed (RME) individuals (i.e., 4.0 and 31.9 grams per day, equivalent to approximately 6 and 51 half-pound meals per year, respectively) (see, HHRA, pp. 24 and 37). Please comment on whether this approach provides reasonable estimates of fish consumption for the central tendency and RME individuals for use in the point estimate calculations.
3. Superfund risk assessments often assume a 30-year exposure duration, based on national data for residence duration. However, because an angler could move from one residence to another and still continue to fish the 40 mile-long Upper Hudson River, USEPA developed a site-specific exposure duration distribution based on the minimum of residence duration and fishing duration. The residence duration was based on population mobility data from the U.S. Bureau of Census (1990) for the five counties that border the Upper Hudson. The fishing duration was developed from the 1991 New York Angler survey (Connelly et al., 1992). The 50th and 95th percentiles of the distribution were used for the central tendency (average) and RME exposure durations (i.e., 12 and 40 years, respectively). Please comment on the adequacy of this approach in deriving site-specific exposure durations for the fish ingestion pathway (see, HHRA, pp. 23 and 49-57).

4. PCB concentrations in Upper Hudson River fish generally have declined in past decades and the decline is expected to continue into the future. Therefore, to evaluate non-cancer effects for the RME individual, USEPA used exposure point concentration in each medium (water, sediment, and fish) based on the average of the concentrations forecast over the next 7 years (1999 to 2006), which gives the highest chronic dose considered in the HHRA. For the central tendency exposure point concentrations, USEPA used the average of the concentrations forecast over 12 years (1999 to 2011), which is the 50th percentile of the residence duration developed from the population mobility data (U.S. Bureau of Census, 1990). In addition, for completeness, USEPA averaged the exposure concentration over 40 years (1999 to 2039) to evaluate non-cancer hazards for the same time period over which cancer risk was calculated. Please comment on whether this approach adequately addresses non-cancer health hazards to the central tendency and RME individuals (see, HHRA, pp. 67-68).

Monte Carlo Analysis/Uncertainty Analysis

5. USEPA policy states that probabilistic analysis techniques such as Monte Carlo analysis, given adequate supporting data and credible assumptions, can be viable statistical tools for analyzing variability and uncertainty in risk assessment (USEPA, 1997a). Consistent with this policy, USEPA used a tiered approach to progress from a deterministic (i.e., point estimate) analysis to an enhanced one-dimensional Monte Carlo analysis of the fish ingestion pathway (see, HHRA, Chapter 3, pp. 33-59). Please discuss whether this Monte Carlo analysis makes appropriate use of the available data, uses credible assumptions, and adequately addresses variability and uncertainty associated with the fish ingestion pathway (e.g., defining the angler population, PCB exposure concentrations, ingestion rates, exposure durations, cooking losses) qualitatively or quantitatively, as appropriate, in the analysis (see, HHRA, pp. 72-74).
6. For the Monte Carlo analysis, USEPA evaluated a number of angler surveys, but excluded local angler surveys, such as the 1996 and 1991-1992 Hudson Angler surveys (NYSDOH, 1999; Barclay, 1993), due to the fish consumption advisories. The 1991 New York Angler survey (Connelly et al., 1992) was used as the base case and other surveys were used to address sensitivity/uncertainty in fish ingestion rates (see, HHRA, pp. 37-46). Please comment on the adequacy of USEPA's evaluation and use of existing angler surveys in the Monte Carlo analysis of the fish ingestion pathway.

Risk Characterization

7. The risk characterization section of the HHRA (Chapter 5, pp. 67-80) summarizes cancer risks and non-cancer hazards to individuals who may be exposed to PCBs in the Upper Hudson River. Please comment on whether the risk characterization adequately estimates the relative cancer risks and non-cancer hazards for each pathway and exposed population. Have major uncertainties been identified and adequately considered? Have the exposure assumptions been described sufficiently?

General Questions

- 1) A goal for risk assessments is that they be clear, consistent, reasonable and transparent and adequately characterize cancer risks and non-cancer hazards to the exposed population, including children (USEPA, 1995b, 1995d). Based on your review, how adequate are the HHRA and Responsiveness Summary when measured against these criteria?
- 2) Please provide any other comments or concerns, both strengths and weaknesses, with the HHRA not covered by the charge questions, above.

Recommendations

Based on your review of the information provided, please select your overall recommendation for the HHRA and explain why.

1. Acceptable as is
2. Acceptable with minor revision (as indicated)
3. Acceptable with major revision (as outlined)
4. Not acceptable (under any circumstance).

Holly Hattemer-Frey

**REVIEW OF THE HUMAN HEALTH RISK ASSESSMENT
HUDSON RIVER PCBs REASSESSMENT RI/FS**

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COMMENTS IN RESPONSE TO CHARGE QUESTIONS

1. Chapter 4, Toxicity Assessment: I agree that the toxicity data currently available in IRIS for PCBs should be used in the assessment. This approach is consistent with EPA policy and allows for easy comparison of risks between hazardous waste sites. On the other hand, it is reasonable to discuss the more recently-available data on the potential toxicity of PCBs (e.g., Kimbrough *et al.*, 1999) and the effect using these data would have on risk estimates. For example, if the more recently-available toxicity data were used, would risks increase or decrease and by what factor? Relying exclusively on the more recently-available data is not appropriate, however, since a full peer review of the data has not yet been completed.
2. Fish Ingestion Rates (p. 43, bottom): The reason for selecting the 90th percentile fish ingestion rate of 31.9 g/day versus the 95th percentile value (63.4 g/day) seems arbitrary. The 90th percentile value was selected because it is more consistent with 95th percentile values reported in other studies. While this is true, adopting the 90th percentile value discounts the fact that NY anglers may actually consume more fish than anglers from other states. Since the authors didn't have any *a priori* reason to disbelieve the 91 Angler survey results, the arbitrary selection of the 90th percentile value may underestimate angler fish consumption. This is a minor point, however, since doubling the fish ingestion rate would not substantially increase risk estimates.
3. Section 3.2.4.3, Exposure Duration: Calculation of a site-specific exposure duration using census and mobility data is appropriate. If I understand the approach correctly, a one-year probability that an individual would move out of the region is estimated for a given number of years, and then those 1-year probabilities are summed to determine the probability that an individual would move out in a specified time period. This approach does not seem to account for the fact that individuals who moved out in a given year (e.g., the first year) would not be available to move in subsequent years. If this is true, then residence duration is likely to be overestimated, and the approach used should be modified or the extent to which results may be overestimated should be discussed.
4. Page 23, para 2 notes that the 50th and 95th percentile values for fishing duration are 12 and 40 years, respectively. Since PCB concentrations in fish will decline over time, adopting an exposure duration (ED) of 7 and 12 years for the RME and CT scenarios,

respectively, will yield the highest chronic dose to receptors. Since the HHRA has acknowledged the conservativeness of this approach and calculated a hazard quotient assuming a 40-year exposure duration for comparison, I believe that this approach adequately addresses noncancer hazards.

5. Monte Carlo Analysis: See general and specific comments below.
6. Monte Carlo results associated with using the Maine fish ingestion rates, which were the lowest rates of the studies evaluated, are presented on p. 78-79. For completeness sake, the text should include a discussion of Monte Carlo results using the range of fish ingestion rates reported in West *et al.*, 1989 and Connelly *et al.*, 1996 as well. This is a minor point, as I agree that adopting a different fish ingestion rate in the base case (or point-estimate calculations) will not substantially alter risk results.
7. Overall, the risk characterization adequately estimates cancer and noncancer risks to exposed individuals. Deficiencies in the exposure assessment and other aspects of the risk assessment that affect risk results are detailed below.

GENERAL COMMENTS

1. The method by which PCB concentrations in fish were calculated (p. 11-14, Section 2.3.1 and p. 23-24) is not well presented. While I understand that the details on how fish concentrations were calculated are presented in the Baseline Modeling Report, which has been separately peer reviewed, it is crucial that individuals reading the HHRA have a clear understanding of the process. The description of how fish concentrations were derived is not transparent or adequately summarized. I found it confusing and difficult to follow. A more detailed, step-by-step explanation would enhance the report. Perhaps including a sample calculation and/or a flow diagram of the process would be useful. Furthermore, any significant changes concerning how fish concentrations were calculated raised by the ecological peer reviewers should also be addressed in the HHRA.
2. The Phase 2 assessment did not evaluate potential risks associated with the consumption of home-grown fruits and vegetables and soil for individuals living in floodplain areas where residential soils may have been contaminated during flood events. Information on when and where the Upper Hudson River (UHR) may have flooded during the last 20 to 30 years should be available. If flooding has occurred, information on where floodplain soils may have been contaminated with PCBs should be summarized in the risk assessment. A crude, conservative calculation of potential risks associated with soil and produce ingestion could be completed by assuming that the current soil concentration equals the current sediment concentration. Although exposures via contact with floodplain soils are likely to be a minor relative to fish ingestion, they should still be address in the HHRA.

3. Agree that limiting the focus of the Phase 2 investigation to PCBs is appropriate in this case.
4. Chapter 2 is poorly organized and confusing. The text consistently refers to details presented in Chapter 3. For example, specific information on why the 90th percentile fish ingestion rate (versus the 95th percentile value) was used in point-estimate calculations is described in Section 3.2.1.3 (p. 42)--not in Chapter 2 as it should be. Details affecting point-estimate calculations should be incorporated into Chapter 2 to facilitate better comprehension of how baseline exposures were calculated.
5. Chapter 2 summarizes the exposure parameters used to calculate intakes but does not present the results of the exposure assessment. Tables documenting calculated intakes for each pathway and receptor group should be included in Chapter 2.
6. Chapter 3 does not provide sufficient detail on the range of values used in the Monte Carlo (MC) analysis for each parameter. Tables summarizing the specific range over which each parameter was allowed to vary should be included.
7. The start date for the HHRA is 1999 (p. 72, para 2), which does not consider individuals who may have been fishing in the UHR before 1999. In many risk assessments, exposure-point concentrations are based on measured data only, and extensive modeling to predict future concentrations is not done. In this case, however, extensive modeling has been done, including a short-term hindcast calibration test covering the period 1991 to 1997 (see Baseline Modeling Report Executive Summary p. ES-4, para 1). It may be possible to estimate fish concentrations for periods before 1999 with great difficulty. If it is, then the magnitude of exposures before 1999 should be evaluated (or at least discussed in the uncertainty section).

SPECIFIC COMMENTS

p. ES-3 to ES-4: The actual RfDs and CSFs used to calculate risks and HI values should be reported here instead of stating that the "most current values" were used.

Chapter 2

Table 2-1 and Section 2.1: Both should be revised to include evaluation of exposure to floodplain soils by residential and recreational receptors (e.g., picnickers) via direct ingestion.

page 8, Section 2.1.3, para 1: The text states that ingestion of river water as drinking water was not evaluated since PCB levels in the UHR were less than the Maximum Contaminant Level (MCL). For accuracy and clarity, the text should compare the upper-bound or maximum concentration of PCBs in the river water with the actual MCL.

p. 11, last para: The text states that PCB concentrations were determined for six fish species.

These six species were chosen because they were representative of bottom feeders, top predators, and semi-piscivorous fish. In other words, they were apparently chosen to fulfill the data needs for the ecological risk assessment. A complete list of fish species likely to occur in the UHR as well as those species likely to be consumed by humans should be included to allow the reader to confirm that the fish species selected are representative of species consumed by humans.

p 11, bottom to 12, top: The reason why PCB concentrations in fish were based on a Tri+ concentration (i.e., only data for PCB congeners with three or more chlorine molecules were used) needs to be more clearly explained. The reasons why a Tri+ concentration was used are outlined in paragraph 2, p. ES-4 of the Executive Summary for the Baseline Modeling Report. That information needs to be added here for clarity. Moreover, information on how Tri+ concentrations compare to total PCB concentration and how using Tri+ concentrations (versus total concentrations) has affected risks estimates should be discussed.

p 12, para 2: The text states that fish PCB concentrations were assumed to be lognormally distributed. A test to confirm the distribution of these data (e.g., a Shapiro-Wilk or W goodness of fit test) should have been done and results reported here.

p 23, Averaging Time: The text states that "to avoid confusion" a 70-year life expectancy was used to calculate cancer risk averaging times. The more current estimate of 75 years based on more recent EPA guidance (cited as USEPA, 1997f in the document) should have been used.

Tables 2-6 thru 2-8: These tables are very confusing. Data included in these tables is not defined adequately in the text or footnotes. A more detailed discussion of the difference between columns 3, 8, and 11 and which values were used as the exposure-point concentration needs to be included in the text. Some acronyms/abbreviations cited in the first line of the footnotes don't seem applicable and should be deleted (i.e, Max, UCL-N, 95% UCL-T, and Mean-T). In columns 10 and 13, I recommend including the actual number of years over which data were averaged to clarify (e.g., 40, 7, or 12 years). Column 5 should note that the value listed is the maximum *modeled* concentration. Delete column 6 since it's not relevant. Line 4 should state "species weighted for cancer exposure." Footnotes 1 and 2 should refer to the appropriate column for clarity.

Chapter 3

p. 33, para 1: The text needs to explain more clearly that a Monte Carlo analysis was done on the fish ingestion pathway only because risks associated with the other exposure pathways quantified were minor relative to fish consumption.

p. 35, para 3: The text states that "for reasons describes later" a 2-D Monte Carlo analysis could not be done. Even after reading all of Chapter 3, it is not clear why a 2-D analysis wasn't (or couldn't be) done.

p 51, Adjustment 1: Acknowledging that my area of expertise is not probabilistic analyses, could s and c be rounded to the nearest of 5 (instead of 10) without compromising the robustness

of P(s,c)? Since rounding to 10 is likely to underestimate child exposures, rounding to the nearest five might afford better representation of child exposures.

p 51, Adjustment 2: The assessors opted to include the data for non-respondents even though information on the age at which these individuals started fishing was not reported and had to be estimated. Estimating the age at which non-respondents starting fishing introduces error into the assessment. Discuss the effect on model outcome of adopting these adjustments. Are the assumptions and adjustments adopted likely to over- or underestimate exposures?

p. 52, Discussion of Assumptions: A number of the assumptions associated with the Monte Carlo analysis assume that the angler population is at steady-state, meaning that the age profile remains consistent over time. Do the 1980 and 1990 Census data support this assumption, or do they indicate the population living near the UHR is getting older or younger?

p. 54: Again, more adjustments are made to the raw data before using it in the Monte Carlo Analysis without discussing the error or bias introduced by making these adjustments. Discuss the effect on model outcome of adopting these adjustments. Are the assumptions and adjustments adopted likely to over- or underestimate exposures?

p 66, last sent: Recommend adding text that the magnitude of uncertainty associated with possible endocrine disruption cannot be determined at this time.

Sect. 5.1.2, p 69, top: Point-estimate risks for children consuming fish should be formally calculated and reported here and in the Executive Summary, and child-specific exposure factors be included in Section 2.4.1. Even though 1988 and 1991 New York Angler Surveys (Connelly et al., 1990; 1992) reported that the average individual didn't start fishing in the UHR until age 13 to 14, it is possible that children of anglers were fed fish taken from the UHR. The assumption that children consume portions 1/3 the size of an adult portion would yield RME and CT child ingestion rates of 10.5 g/day and 1.3 g/day, respectively. These values seems reasonable and are fairly consistent with those reported in USEPA, 1990), which reports RME and CT child ingestion rates of 7.5 g/day and 2.8 g/day respectively.

Section 5.3.1: This section should include uncertainty associated with not evaluating potential exposures and risks from direct ingestion of soil and produce.

Monte Carlo Analysis: One possible scenario that was not evaluated (and perhaps should be) is an individual who preferentially consumes fish from the same species and location (e.g., someone who only eats bass from the Thompson Pool area). This scenario will probably not substantially alter risk estimates, since PCB fish concentrations did not vary dramatically within a given species taken from the same location (as exemplified in Table 5-34), but for completeness sake it

Holly Hattemer-Frey

should be included.

RECOMMENDATION

Based on my review of the HHRA, I find the report acceptable with the major revisions outlined above.

REFERENCES

U.S. Environmental Protection Agency (1990). Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions, PB90-187055, EPA /600/6-90/003, Environmental Criteria and Assessment Office, Cincinnati, OH.

Owen Hoffman

**PEER REVIEW FOR THE HUDSON RIVER PCBs
HUMAN HEALTH RISK ASSESSMENT**

The following is the report of my review of the Human Health Risk Assessment on Polychlorinated Biphenyls (PCBs) in the Upper Hudson River conducted for the U.S. Environmental Protection Agency. My review concentrated on two documents: Volume 2F - Human Health Risk Assessment Hudson River PCBs Reassessment RI/FS (August 1999), produced by TAMS Consultants, Inc., and Gradient Corporation; and Hudson River PCBs Reassessment RI/FS Responsiveness Summary for Volume 2F - Human Health Risk Assessment (March 2000), produced by TAMS Consultants, Inc., and Gradient Corporation.

OVERALL RECOMMENDATIONS

The present EPA risk assessment provides sufficient information to clearly indicate that PCBs released from the General Electric facility into the Upper Hudson River are a regulatory concern. The human health risk assessment does not provide sufficient information to enable evaluation of the potential health risk to humans under baseline conditions. Base-line conditions imply that no restrictions are in place to prevent people from utilizing the Upper Hudson River for sport fishing, harvesting of other aquatic organisms for food, or as a commercial fishery for striped bass.

In general, the toxicity coefficients obtained from the IRIS database are intended by EPA to be conservatively biased, i.e., in the presence of uncertainty their use will err on the safe side to assure that real exposed persons are unlikely to suffer harm. The EPA human health risk assessment, however, does not indicate at what concentrations or exposure levels increased levels of harm might be expected to occur. Therefore, although information is adequate to conclude that PCBs are of a regulatory concern, information is inadequate to evaluate the uncertainty associated with anticipated health impacts.

To properly assess the cost-effectiveness of risk reduction during the analysis of the possibility of remediation, information is needed on the uncertainty in the toxicity coefficients for PCBs, the concentrations of PCBs in fish harvested at future dates, and concentrations of other cancer causing and non-carcinogenic substances in various environmental media of the Upper Hudson River.

The EPA human health risk assessment does not adequately address uncertainty in quantifying health risk. Areas where the uncertainty analysis is deficient are as follows:

- (1) *Individuals who would be exposed to contaminated environmental media in the Upper Hudson River are exposed to much more than just PCBs.* They are also exposed to agricultural chemicals and to radionuclides introduced by various facilities and by atmospheric testing of nuclear weapons. All of these add to the overall health burden, and it is this cumulative burden that should be assessed. The current risk assessment focuses only on health the impact of exposure to PCBs.
- (2) *The assessment is focused on projected PCB concentrations in fish averaged for the entire 40-mile reach of the Upper Hudson River. Uncertainties on these projected average concentrations are not presented in the report.* In the March 2000 Responsiveness Summary, the projected average concentrations in the various species of fish are too narrow to be plausible. The assessment of uncertainty is limited to a comparison of model predictions with past observed concentrations in various fish species. The uncertainty associated with forecasting PCB concentrations in fish over time is not considered.

The comments in the EPA human health risk assessment about the differences between the uncertainty in the mean concentration of a sample versus the uncertainty in the mean concentration in a model prediction is technically incorrect. Both measured and modeled mean concentrations have associated uncertainty, and this uncertainty should be quantified and reported.

- (3) *Averaging concentrations and exposures over the entire reach of the Upper Hudson River is inappropriate.* Clean-up options will be designated for various subreaches of the river, and the HHRA should target those subreaches. In addition, the population exposed to the entire Upper Hudson River would likely be quite large, much larger than the 10,000 anglers referred to in the HHRA documents. It is easy to imagine that the number of people consuming fish out of the Upper Hudson River would include the families of anglers, families of those who fish but who are not licensed, and those who would purchase fish from commercial fisheries, if such fisheries were to go into operation under

baseline conditions. Therefore, targeting the upper 95th percentile of a very large population has the potential to substantially underestimate exposure to a significant subpopulation of that group. For example, assume that the total number of people who consume fish from the Upper Hudson River is on the order of 100,000 individuals. The top 5% of the distribution of that population would still entail a population of 5,000 individuals. The top 1% would still include a population of 1,000 individuals. Therefore, I believe it is more appropriate for the assessment to target subpopulations that would utilize subreaches of the Upper Hudson River, rather than the entire 40-mile reach. In addition, I would target separately individuals characterized as casual, average, and maximal users of the Upper Hudson River as opposed to treating inter-individual variability as a random process.

- (4) *The Monte Carlo analysis is not used to address uncertainty.* It is used only to simulate interindividual variability among licensed anglers as a stochastic process. Instead of subdividing the population into those who would be most likely to consume moderate to average amounts of fish, and those likely to consume maximum amounts of fish, the Monte Carlo analysis simply draws directly on empirical results from slightly more than 200 respondents to a 1991 angler survey conducted for upstate New York. This survey was conducted over a broad region of the state. Sites included many that were subjected to fishing advisories. Thus, the database used to drive the Monte Carlo analysis is not directly relevant to the population of concern who would be consuming fish out of the Upper Hudson River. The degree to which the empirical database is relevant to the Upper Hudson River has been discussed, but the potential for bias is not included in the Monte Carlo analysis.

Many other sources of information that would lead to an expression of interindividual variation have not been included in the Monte Carlo analysis, including

- (a) the size of an average meal per person,
- (b) the amount of fish that would be caught from other locations besides the Upper Hudson River,
- (c) the likelihood that there would be variability in food preparation losses from one meal to another, and
- (d) the likelihood that food consumption patterns would change over a period of 7 to 40 years.

The 1991 angler survey itself was a recall study for a single fishing season, and its applicability for an average person over periods of 7 to 40 years is not discussed. I anticipate that the extent of interindividual variability as well as the amount of fish consumed at the upper percentiles of the true frequency distribution has been substantially overestimated.

To summarize, the Monte Carlo analysis fails to capture the inter-individual variability of exposure averaged over a time period of 7 to 40 years and the interindividual variability and uncertainty of risk to these individuals. The sensitivity analysis that is performed to indicate the uncertainty in the Monte Carlo analysis does not fully capture uncertainty. Many of the assumptions made are inappropriate. For example, fish concentrations are assumed to occur entirely at one segment of the river or another. This would be appropriate if one were to assess the interindividual variability for a sub-reach, but what is needed here is for the uncertainty analysis to include an estimate of the uncertainty on the average concentrations in various fish species caught from the subreach of concern.

Food losses as a result of cooking and preparation of fish is treated as an uncertain variable. In actuality, food loss should be treated as a frequency distribution, as one would expect to have differences in food losses from meal to meal and from year to year. The assumption of 0% food loss for all meals over all years is implausible.

- (5) *Uncertainty in cancer and non-cancer health endpoints should be included explicitly.* Although there is EPA policy guidance that discourages risk assessors from explicitly considering the uncertainty in cancer slope factors (CSFs) and Reference Doses (RfDs), the risk calculation cannot be considered to be scientifically defensible until uncertainty in the toxicity coefficients is properly accounted for in the human health assessment.

It is EPA policy to allow for the expression of uncertainty about toxicity in ecological risk assessment. It is thus inconsistent to exclude the evaluation of uncertainty in the toxicity coefficients from the human health risk assessment of PCBs.

If EPA mandates the exclusion of the evaluation of uncertainty in the toxicity coefficients, then the task ceases to become a true risk assessment. Instead, the task is restricted to an EPA mandated regulatory compliance calculation.

A detailed evaluation of potential uncertainty in the PCB toxicity coefficients is important because of the potential exposure of very large numbers of individuals. This is the case for the Upper Hudson River. Under baseline conditions, there would be no restrictions to the access and harvesting of fish. Baseline conditions should include sport fishing, unlicensed fishing, harvesting of other biota, and commercial fishing.

I thus conclude that the present EPA HHRA, although adequate for identifying a situation of clear regulatory concern, is inadequate as a scientifically defensible risk assessment. The present risk assessment may be either overly pessimistic or not protective enough depending upon what information is and what information is not included in the analysis.

The following sections give my answers to specific questions that have been posed by EPA:

EPA Question 1, Hazard Identification and Dose Response

The human health risk assessment uses the most recent values of the cancer slope factors and noncancer risk RfDs listed in the Integrated Risk Information System (IRIS). This is the agency's database of consensus toxicity values. However, IRIS clearly states that risk assessors may use more recent data when such data are available.

The use of IRIS values of PCB toxicity is appropriate for indicating the presence of contamination that warrants regulatory concern. The cancer slope factors and RfDs, however, do not indicate the actual risk of cancer or of noncancer endpoints, because the uncertainties associated with these quantities are not included as a part of the risk assessment. Although RfDs are derived by a factor known as an "uncertainty factor (UF)," then UF values do not disclose uncertainty. Values of UF are equivalent to safety factors that are used to account for the presence of uncertainty in order for the exposure assessment to indicate levels that are appropriately protective.

Simply because a noncancer hazard quotient for PCBs exceeds 1.0 does not necessarily indicate that there is a significant health threat. The most current RfDs used for PCBs include an uncertainty factor of 100 to 300. The potential for these values of UF to be overly protective should be assessed. For example, instead of multiple factors of 10 from subchronic to chronic

exposure, and from animal studies to human studies, what if a factor of 3 had been used? Recent papers by Swartout et al. 1998, and Price et al. 1997a and 1997b, should be reviewed on this subject. Furthermore, it might be more revealing to show the combined amount of exposure to PCBs and other toxic substances, in order to look at the total Hazard Index and the total cancer risk from utilization of the Upper Hudson River, which would include exposure to water, sediment, and aquatic biota.

In conclusion, IRIS toxicity values of CSFs and RfDs are adequate for indicating levels of regulatory concern. They are not adequate for indicating potential human health risk. The present risk assessment may be either overly pessimistic or not protective enough depending upon what information is and what information is not included in the analysis.

EPA Question 2: EPA asked the reviewers to comment on whether the specification of central tendency and reasonably maximally exposed individual consumption rates of 4 and 31.9 grams per day, equivalent to approximately 6 and 51 half-pound meals per year, respectively, are reasonable to capture interindividual differences in exposure for point-estimate calculations.

My answer is yes, this is reasonable. Six fish meals per year from the Upper Hudson River can be attributed to any one of a large number of representative individuals that could utilize the Upper Hudson River. Fifty-one half-pound meals per year also appears to be reasonable as a maximum estimate. This maximum estimate could be achieved by someone eating much more than one fish meal per week during the fishing season, and relatively few fish meals during the rest of the year, or by someone consuming multiple fish meals per week, but only harvesting a few of those meals from the Upper Hudson River. I consider both numbers to be adequate for point-estimate calculations.

In fact, for the Monte Carlo calculation, if one is estimating the uncertainty in the exposure and risk to reference individuals, it would be appropriate to fix the dietary intake for the representative (or reference individual), and then the values of 4 and 31.9 grams per day would then be appropriate for use as fixed reference values. The uncertainty in risk would then not be a function of diet, but a function of the uncertainty in the toxicity coefficient, the uncertainty in the concentration of the PCBs in the aquatic media, and the uncertainty associated with losses of PCBs due to food preparation.

EPA Question 3: Along the 40-mile reach of the Upper Hudson River, the EPA has assumed central tendency and reasonably maximal exposure durations of 12 and 40 years, respectively, for a cancer causing substance, and 7 years for exposure to noncancer causing substances.

I believe that these values for point-estimate calculations are appropriate; however, because of the very large population that could be affected along the 40-mile reach, it may be more appropriate to also consider individuals who would spend their entire lifetime accessing fish from this region. The size of the population that potentially could utilize fish from this region may exceed tens of thousands of individuals. For this reason it would be appropriate to consider individuals who could potentially be in residence for a period much longer than 40 years.

Much more important, however, is the fact that individuals won't necessarily harvest all of their fish from the Upper Hudson River. Some consideration should be given to the situation in which a residential angler living for more than 40 years along the Upper Hudson River actually consumes some amount of fish from other locations.

EPA Question 4: PCB concentrations in fish have declined in past decades, and the decline is expected to continue into the future. To evaluate noncancer effects for the maximally exposed individual, EPA used point concentrations in each medium (water, sediment, and fish), based on the average concentration forecast over the next 7 years from 1999 to 2006. For point concentrations for exposure, the central tendency exposure, EPA used the average of the concentrations forecast over 12 years, which is the 50th percentile of the residence duration developed from population mobility data. In addition, for completeness, EPA averaged the exposure concentration over 40 years to evaluate noncancer hazards for the same time period over which cancer risk was calculated. The review team has been asked to comment on whether this approach adequately addresses noncancer health hazards to the central tendency and reasonably maximally exposed individuals.

Yes, I believe it is appropriate to specify a reasonable midpoint for averaging concentrations in fish for a short-term time duration of a 7-year exposure. However, because the Hazard Quotients for PCBs are so large, it is also appropriate to look at subchronic exposures that may affect critical population subgroups. In this case, exposure durations as short as one year should be considered. During a one-year time period, a person could be at much lower body weight than is currently assumed in the risk assessment. The uncertainty in fish concentration at a given

location, as mentioned previously, is not adequately expressed in the present EPA human health risk assessment.

My personal preference would be for the assessment to focus on subreaches of the Upper Hudson River and for the uncertainty in the average fish concentrations to be determined for those subreaches. To average the assessment over the entire 40-mile stretch of the river is inappropriate, and there is the possibility that critical subgroups of the population will be missed because of the very large number of people potentially exposed if no restrictions were placed on utilization of this aquatic resource.

EPA Question 5, The Monte Carlo Analysis: We were asked to discuss whether the Monte Carlo analysis used in the HHRA makes appropriate use of the available data, uses credible assumptions, and adequately addresses variability and uncertainty associated with the fish ingestion pathway; the last item would include defining the angler population, PCB exposure concentrations, ingestion rates, exposure durations, cooking losses, etc.

Having reviewed the Monte Carlo calculations in detail, I find that they capture neither the interindividual variability of potential exposure in risk, nor the uncertainty about a reference average individual or a reference maximally exposed individual.

Monte Carlo calculations serve two distinctly different purposes. The first and perhaps most important purpose is to propagate uncertainty through risk assessment algorithms (when it is difficult or impossible to propagate such uncertainty using algebraic formulae). When Monte Carlo techniques are used to propagate uncertainty, the present state of knowledge is expressed as a subjective probability distribution given all of the evidence available (NCRP, 1996; IAEA, 1989; Cullen and Frey, 1999; National Research Council, 1994). A subjective probability distribution is specified for each variable that can be considered to be a true but unknown quantity.

In the present EPA human health risk assessment, the Monte Carlo calculation is not used to propagate uncertainty, but instead to simulate interindividual variability of exposure. Interindividual variability is assumed to be a stochastic, random process, which, of course, is not the case. There are distinct reasons why some individuals choose to eat more fish than do others.

A more practical approach would be to specify several reference receptors and use Monte Carlo techniques to quantify uncertainty about the health risk to those reference human receptors. At the very least, the Monte Carlo techniques should be used to quantify uncertainty for a reasonable average individual and for a reasonable maximally exposed individual.

In this particular assessment, the Monte Carlo calculation utilizes the average fish concentration of PCBs for a 40-mile reach. This average value is given without uncertainty. The Monte Carlo calculation also assumes that a dietary survey for upstate New York for free-flowing fresh water is directly applicable to the case of the Upper Hudson River. This fish survey is for licensed anglers, of whom 221 responded out of 1000 questionnaires that were distributed.

It is well known that fish surveys that are based on individual recall are biased. The tendency is to overestimate the amount of fish caught and consumed. The extent to which the distribution defined by 221 individuals may be biased has not been assessed. Instead, the 221 values are used verbatim, after unreasonable values at the low and high ends have been censored by the authors of the risk assessment. The censored values include those who reported more than 1,000 fish meals during the year and those who reported no fish meals during the year.

Several items are missing as the result of the empirical use of the 1991 fish survey results: (a) The extent to which the average value for this distribution is biased high, (b) the extent to which the upper end of the distribution is biased high, (c) the extent to which the relative variability reported from the distribution may be biased high, and (d) the extent to which a single-year recall survey is representative of a 7 to 40-year average also needs to be assessed.

The results of the Monte Carlo analysis are uninterpretable. This is due to the failure of the Monte Carlo calculation to consider

- uncertainty in fish concentrations,
- uncertainty in the amount of fish consumed that is actually caught or harvested from the Upper Hudson River, and
- the uncertainty associated with the use of empirical survey data to represent the dietary pattern of the population that might consume fish from the Upper Hudson River over a period of 7 to 40 years.

Simulating individual variability in a large population as a frequency distribution of true values is a daunting task. Relevant data must either be available or the uncertainty associated with partially relevant data must be explicitly considered. My present evaluation is that the high-end exposure is more than likely over-estimated for the true 95th percentile, but the true 99th percentile and above are potentially underestimated because the dietary survey is truncated as a result of only 221 respondents. I believe the Monte Carlo calculation for interindividual variability should include the total number of people potentially exposed so that one can judge how many people could have a risk above a regulatory of concern, and how many people could have risk extending into a region of a likely health threat.

Of course, the present Monte Carlo distribution does not include the variability or uncertainty in cancer slope factors and RfDs. Thus, the risk assessment is interpretable only from the standpoint of regulatory concerns, not from the standpoint of potential health risk.

Because exposure to multiple contaminants in the aquatic medium has not been taken into account, it is virtually impossible at present to make an overall assessment of health risk. All that can be concluded is that if fishing restrictions were removed, it is very likely that many individuals who would consume fish from the Upper Hudson River would be exposed to PCBs at a level that would warrant regulatory concern.

EPA Question 6, The adequacy of EPA's evaluation and use of existing angler surveys in the Monte Carlo analysis of the fish ingestion pathway:

I touched upon much of this in my answer to the above question; however, I think that it is important that the potential sources of bias in any fish angler survey be considered explicitly. If the objective is to try to model the entire population who has consumed fish from the Upper Hudson River, then it is important to evaluate the extent to which angler survey data may be biased and might either under- or overstate the amount of actual fish consumption that occurs.

I believe the upper end of the distribution can be grossly overstated from the true values that would occur over a 10- to 40-year time period. I also believe that the interindividual variability, (or the geometric standard deviation) of the distribution, is overstated because the survey is based on individual recall for a relatively short time period.

For cancer-causing substances, I believe it is more important to look at the uncertainty on the *average* fish consumption than it is to look at the uncertainty on the median. The median in this case will understate the total cancer risk to this population. The total cancer risk (number of cases in the population), is a product of the *arithmetic mean* exposure in the population, the size of the population, and the cancer slope factor.

The sensitivity analysis, which uses (a) a range of variables of fish concentrations in the river, (b) a range of loss fractions due to cooking and preparing fish prior to human consumption, and (c) alternative databases for angler surveys, only partially captures the uncertainty that is present. The cooking loss variable is more appropriately expressed as a source of inter-individual variability of exposure. The uncertainty about the average loss due to cooking over a 10- to 40-year lifetime history of individuals capturing fish from the Upper Hudson River is much less than the range of 0 to 40% assumed in the HHRA.

EPA Question 7, Risk Characterization: Risk characterization in the human health risk assessment summarizes the cancer risk and noncancer hazards to individuals who may be exposed to PCBs in the Upper Hudson River. The reviewers were asked to comment on whether the risk characterization adequately estimates the relative cancer risks and noncancer hazards for each pathway and exposed population. Have major uncertainties been identified and adequately considered, and have the exposure assumptions been described sufficiently?

Again, the assessment adequately discloses that exposure to PCBs is of regulatory concern. However, to evaluate the health risk from exposure to PCBs and other contaminants existing in the Upper Hudson River, far more information is needed than is available at this time. The uncertainty associated with cancer slope factors and RfDs should be taken into account explicitly in order for the uncertainty in the risk estimates to be properly expressed. Without such uncertainty estimates, the risk of making the wrong decision when determining the feasibility of cleanup cannot be evaluated.

My conclusion is that the risk characterization, although adequate to indicate a situation of regulatory concern, is clearly inadequate for expressing the degree of health risk that may be present under baseline conditions. The true health risk may be grossly overstated in some aspects, and in other aspects, it may be understated. The extent to which it is either overstated or understated cannot be evaluated given the information at hand.

EPA GENERAL QUESTIONS

EPA General Question 1, Clarity and Transparency of the HHRA

This HHRA is consistent with other regulatory documents that I have reviewed; however, this and the other documents suffer from being written for an audience of regulators, not for an audience of individuals who might be concerned with potential health hazards associated with consuming PCB-contaminated fish from the Upper Hudson River.

My recommendation is that the overall report be de-jargonized, the use of regulatory acronyms eliminated, and the report re-edited so that an interested individual can readily comprehend the content of this report.

Many of the figures are presented on a linear arithmetic scale. They should instead be plotted in mathematical units on a logarithmic scale, because the intent of these figures is to evaluate *relative differences* as opposed to absolute differences in trends over time. The probability plots that are presented showing differences in fish consumption rates from the various angler surveys are virtually unreadable to all but statisticians. Those plots should be redrawn showing the mathematical units on the y-axis, preferably using a log scale, and showing the relative probability or cumulative probability on the x-axis. Software is readily available that will allow a more transparent presentation of probability plots.

EPA General Question 2, Provide any other comments or concerns about strengths and weaknesses of the HHRA

I would like to understand how exposure contaminants, in combination with the ingestion of PCBs, affect the total cancer risk to individuals consuming fish from the Upper Hudson River, as well as in determining the noncancer health risk. I believe that the uncertainty in the toxicity coefficients, cancer slope factors, and RfDs, should also be taken into account explicitly. I would prefer that the uncertainty analysis focus on reasonably maximally exposed individuals (RME), average individuals, and casual or infrequent consumers of fish from the Upper Hudson River. Monte Carlo calculations can be used to propagate uncertainty for these three separate cases.

I would give a much lower priority to the use of Monte Carlo calculations to simulate interindividual variability, unless it is the objective of the assessment to indicate how many people (the size of the total population of exposed individuals), that would be potentially at risk. If this is the case, then the surveys of licensed anglers are clearly not completely relevant to provide information to meet the objectives of the risk assessment. What is relevant is the survey of all of the individuals who potentially could consume fish from the river, including the families of licensed anglers, unlicensed individuals who utilize the Hudson River as source of food, and those who eat fish from commercial operations. The latter would involve the consumption of fish shipped to restaurants, supermarkets, and so forth.

I believe the strength of the present human health risk assessment is to demonstrate that even under the most optimistic conditions, PCBs in the Upper Hudson River present a regulatory concern. I believe the most pronounced weakness is the failure to disclose uncertainty in the estimate of health risk, including uncertainty in the extent to which PCB concentrations in fish will diminish with time, the uncertainty associated with the cancer and noncancer toxicity coefficients of PCBs, and the cumulative risk from exposure to other carcinogens and non carcinogens present in the environs of the Upper Hudson River.

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Shubat,

**Peer Review of the Human Health Risk Assessment (Volume 2F) of the Hudson River
PCBs Reassessment RI/FS**

Pamela Shubat, Ph.D.

May 5, 2000

Hazard Identification/Dose Response

1. Comment on the reasonableness of the approach for evaluating dose-response, specifically, evaluating new toxicity data (available after the most recent update to the IRIS files) in the context of the uncertainty analysis (HHRA pages 76-77 and Appendix C).

A concern in choosing appropriate reference doses is whether the congener profile of the PCBs found in the fish in the Hudson matches a particular Aroclor congener profile closely enough so that the use of the Aroclor-specific toxicity value is justified. Homologue patterns discussed during Edward Garvey's presentation on March 23, 2000, showed that PCBs in fish matched Aroclor 1248 (slides showed river reaches and specific fish species). The Human Health Risk Assessment, Volume 2F (HHRA) makes it clear that risk assessors could only choose between reference doses (RfDs) available from the U.S. Environmental Protection Agency (EPA) Integrated Risk Information System (IRIS). The two available RfDs were for Aroclor 1016 and 1254. The HHRA states (page 62) that although General Electric primarily used Aroclor 1242 in their operations, the congener profile in fish tissue is more similar to Aroclor 1254 than Aroclor 1016 (in this particular paragraph, the resemblance of the congener profile to Aroclor 1248 was not relevant).

The apparent shift from a release of Aroclor 1242 to a profile in fish tissue resembling Aroclor 1248 is consistent with descriptions of environmental partitioning in which the more heavily chlorinated congeners persist in biological tissues. However, it is not until the reach between river miles 0 and 60 (slides from E. Garvey's presentation) that the homologue pattern appears clearly dominated by the more heavily chlorinated homologues. The change in this partitioning (shift to more chlorinated homologues) is not discussed sufficiently to understand whether spatial (down-stream), temporal, fish species, or age of fish considerations will lead to additional changes in the homologue pattern in fish. If additional changes are anticipated over the time

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frame of the HHRA, there could be discussion in the HHRA about the appropriate RfD to utilize over time or space.

However, since an IRIS RfD for Aroclor 1248 or 1260 does not appear to be available at this time or the near future, the use of this information would not likely lead to a change in the HHRA for non-cancer health effects. However, I did not review the papers by Arnold or Rice, and am not familiar with the non-cancer dose-response reassessment that is underway by the EPA.

Some issues that could be addressed:

- An RfD based on Aroclor 1248 would be desirable. However, it is not clear whether the long term exposure will continue to be a mix of congeners that resemble Aroclor 1248. Over the duration of the risk assessment it is possible that the mix will eventually more closely resemble Aroclor 1254. This strengthens the rationale for using the 1254 RfD.
- It is not clear that an RfD for Aroclor 1248 would be substantially different from the currently available RfD for Aroclor 1254. The HHRA (page 62) and IRIS files (4/12/00 website) suggests that the toxicologic endpoints for Aroclor 1248 are similar to 1254. This also strengthens the rationale for using the 1254 RfD.
- PCBs appear to be recognized as hormonally and immunologically active and have neurobehavioral effects (Brouwer, et. al., 1999). However, no models are available to extrapolate from these data to a dose-response relationship for risk assessment. While the scientific problems of testing for endocrine disruption were discussed briefly in the risk characterization (HHRA page 77) the public health implications were not discussed. Are there reasons to add additional conservatism and uncertainty in a risk assessment when the endocrine system is affected

Exposure Assessment

2. Comment of the use of the 50th and 90th percentile data from the Connelly study for use as the average and RME individuals (point estimate calculations).

The 1991 Connelly survey (Connelly et. al., 1992) reflects a great deal of experience with fish consumption surveys. There are limitations to the study, largely because the primary purpose of the survey was to collect information on fish advisories rather than fish consumption.

Limitations include: (1) anglers were not asked about meal size, (2) anglers were asked to recall fishing activity and fish consumption over an entire year, (3) the mail survey required literacy skills in reading and writing, including entering data into tables, (4) the survey was limited to licensed anglers, and (5) the survey excluded family members (including children) who ate fish caught by an angler.

The strengths of the survey include (1) excellent characterization of non-respondents (unusual to find in fish consumption surveys), (2) a large sample size (1,030 overall, however, for certain purposes it appears that catch data from fewer anglers was used--HHRA Table 3-3), and (3) details on the fishing locale for each fish caught and meal eaten. The HHRA contains an excellent general discussion of the strengths of the mail surveys versus creel surveys, lending support to the use of the Connelly survey.

The following is a detailed discussion of the limitations of the use of the Connelly survey:

(1) Meal Size. The quantity of fish consumed in a single meal is difficult data to collect even with food diaries. A survey participant must receive coaching and use a food scale to accurately report the weight of a serving of fish. Many different approaches have been used to quantify consumption in interviews or mail surveys, with plastic models of serving sizes, scale drawings, or photos of fish arranged on a standard-sized dinner plate being most often used. This survey only collected information on the number of meals consumed per fishing experience. A choice was made by the risk assessors to quantify the meal as 227 g or 0.5 pound based on reports that are unrelated to the Connelly survey.

Other studies support the use of a half-pound fish serving for avid fish eaters. The Chemrisk/Ebert survey of Maine anglers produced an estimated 95th percentile intake for adults

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of 26 g/day for all anglers who ate their catch and 12 g/day for river anglers. These meal sizes were based on an arguably more accurate approach of calculating edible portion from what was known about the size of the fish (self-reported). The EPA Exposure Factors Handbook shows "consuming angler" estimates for the mean intake of 6.4 g/day, the 50th percentile of 2 g/day, and the 95th percentile of 26 g/day.

West surveyed Michigan anglers throughout the opening of a fishing season. Each angler estimated their meal size as larger, smaller, or about the same as 8 ounces. The EPA reanalyzed the West data (Exposure Factors Handbook, 1996) using 5, 8, or 12 ounces estimates of meal size. The study was based on very short-term recall (the past seven days) and the angler referred to a picture depicting the meal in order to judge their own meal size. According to the EPA, the mean intake was 14 grams/day, the 50th percentile was 11 g/day, and the 95th percentile was 39 g/day. The West data are important in providing information about children's intake (0.37 grams fish/kg human body weight/day for children 1-5 years old vs 0.14 grams fish/kg human body weight/day for adults 21-40 years old--however, this precision is not warranted).

In summary, a limitation of the HHRA is that a single meal-size was assumed in both the point estimate and, apparently, the Monte Carlo simulation. Meal frequency varied in the point estimate and meal frequency and body weight varied in the Monte Carlo analysis. This would appear to result in an overestimate of exposure (e.g., 227 g/meal for a 60 kg as well as a 70 kg person) assuming that there will be more simulations using less than 70 kg than simulations using a larger body weight. During the oral presentations there was mention of an assumption that the meal size for a child was 1/3 the meal size assumed for an adult. However, this information wasn't apparent in the HHRA. On page 69 of the HHRA, it says "If it is assumed that a child meal portion is approximately 1/3 of an adult portion....". There was insufficient discussion of the relationship between meal size and human body size to understand what was used in the risk assessment.

Information from the 1989 West study (as reported in the exposure factors handbook) suggests that there are large (almost 3-fold) differences in the intake per body weight for children vs adults. If so, 227 g fish per 23 kg (a six-year-old) could be an appropriate assumption of intake. This should be discussed in the HHRA. A minor irritation is the representation of precision in the HHRA estimates given that the assumed consumption is 0.5 pounds fish per meal.

(2) **Recall Bias.** Recall bias related to the frequency of meals is another extremely difficult issue in fish consumption surveys. The Connelly survey asked the angler to recall a year of fishing including location, catch (numbers of fish per species), and total numbers of meals eaten of each species caught on each body of water. Connelly, in a later recall survey which surveyed anglers who had maintained fishing diaries (Lake Ontario survey), found that anglers who fished frequently were most likely to overestimate the numbers of fish they caught. The implication is that this HHRA will be an overly conservative estimate of intake for the frequent angler. The best fish survey that avoided recall bias was the West study of Michigan anglers, which was based on very short recall (seven days). Multiple “waves” of surveys were sent out to new participants so that the survey covered a large portion of the fishing seasons and, as a result, was administered to a large population.

Encounter surveys (creel surveys) or angler interview surveys are often used for assessing potential exposure, knowledge of advisories, and compliance with regulations and advisories. The more often an angler fishes, the more likely the angler will be included in a creel survey. Therefore, the standard creel survey is most useful for the purpose it was intended—to survey the productivity of the fishery (what is being caught and kept). It was not clear whether creel surveys on the Hudson or its tributaries were available, and any would be difficult to interpret because of the ban on consuming fish. However, the New York State Department of Environmental Conservation River Enforcement Summary of the enforcement on the catch and release fishing program may provide data that could be compared to Connelly survey concerning the type of fish caught and released.

The Connelly survey (Connelly et. al., 1992) did not describe how the species list shown in the survey was drawn up. The list of species and the way they are identified closely resembles the species list in the New York fish consumption advisory for 1991. While this is the appropriate set of data for Connelly to use in a survey of compliance with fish advisories, it is not the set of data to use to determine the species that would be fished if advisories were not in place. It is not clear if the EPA solicited fisheries management data, conservation enforcement data, or recent surveys on fishing preferences.

Shubat,

In summary, it appears that by using the Connelly data for the frequency of fish meals, recall bias could result in a conservative assumption of meal frequency for the frequent angler. It appears that the Connelly data do not provide the best data on the fish that Hudson River anglers would be catching if advisories were not in place. More recent surveys of angler preferences for fish, creel surveys on the lower Hudson, or enforcement data should be used to provide data on the species of fish likely to be sought by anglers.

(3) **Literacy.** The characterization of the non-respondents should have assisted in determining whether literacy concerns limited the response rate. This was not discussed in the Connelly survey or the HHRA. A simple discussion of the literacy rate in the surrounding counties during the years of the Connelly study, perhaps through census data on language spoken at home, enrollment in literacy programs, or immigration data from the state demographer, would provide assistance in understanding whether literacy was potential concern in 1991 and might have biased the survey.

This leads to the need for an overall discussion of whether the demographics of the population surveyed by Connelly still reflect the population considered as current potential anglers on the Hudson. Demographic data for 1999 were not presented and should be examined for income, racial/ethnic makeup, literacy, barriers to licensure, or other factors that would potentially influence fishing for sustenance.

(4) **Licensed Anglers.** The discussion of unlicensed anglers is insufficient (HHRA page 45). An understanding of the fishing and fish-eating habits of licensed and unlicensed anglers is important in understanding whether the Connelly survey data (collected through a mail survey to licensed anglers) are appropriate for use in the risk assessment.

Data are not provided on the number of anglers that are thought to be unlicensed or any demographic data for this population. I have presumed that the New York State Department of Environmental Conservation enforcement summary of the catch and release fishing program pertains to the Hudson River (one page summary given to reviewers on March 23, 2000). These data show that 165 of 324 violations were due to "no license." This included warnings, which might have meant that the officer believed the angler was licensed but was not carrying a license.

Shubat,

Of 1437 anglers checked over a three year period, 72 were ticketed for no license. If these were truly unlicensed anglers, the rate is 1 in 20, and if warnings had been given to unlicensed anglers, the figure could be 1 in 10.

Licenses are not currently required on the lower Hudson (Ed Horne, New York Department of Health, personal communication). The HHRA should include information about whether or not Hudson River anglers must be licensed. If anglers were not required to be licensed in the year of the Connelly study, the HHRA should discuss the uncertainty of applying data collected on licensed anglers to the HHRA.

The obvious question is whether these anglers consume more fish than the participants in the Connelly angler survey (see Hudson River Sloop Clearwater survey below). Barriers to purchasing a license could be poverty, poor literacy skills, or resistance to government control, all of which could influence a choice to eat fish from the river despite postings.

(5) Women and Children. A second concern related to surveying only licensed anglers is that the survey will not include children. The Maine survey data by Chemrisk in 1991 included questions about the family and whether the individuals in the family were consuming fish brought home by the angler. A shortcoming of the survey was that participants were not asked about meal size. The Hudson River Angler Study conducted by the Hudson River Sloop Clearwater, Inc. in 1993 included questions about how a catch was shared with others. Surveyors found that 87 percent of anglers who ate their catch shared the fish with others (Exposure Factors Handbook, August 1996). Presumably this included women and children in households.

Other Comments

The Hudson River Angler Study focused on awareness and compliance with fish advisories. Only 336 anglers were surveyed (1991-1992) and all were shore-based anglers. These data have the same limitations as the creel survey in that the more frequently an angler fishes, the more likely the angler is interviewed. The surveyors found that "more low-income than upper income anglers eat their catch" (Exposure Factors Handbook, 1996). They also found approximately ten percent of anglers were fishing for food rather than recreation. Data available in a thesis by

Wendt were cited in the HHRA to show that the arithmetic mean of meal frequency among low-income families was less than that of the Connelly study. The distribution of meal frequency was not discussed.

While anglers do not always eat their catch, it is appropriate to focus this risk assessment on the exposed population. Therefore, eating one's catch at least once a year is an appropriate criterion for inclusion in the risk assessment. This means that the eating habits of those who eat fish less frequently or episodically or not relevant to completing the HHRA.

It is important to ensure that the most exposed populations have been included. It is not clear whether there was an attempt to investigate the population surrounding the river for demographic characteristics that have been associated with high fish consumption. Although children are not likely to eat fish more frequently than the adults in the household who bring home the catch, they may be more exposed due to a larger meal size per body weight. Children's exposures are not adequately addressed in limiting intake to the meal frequency data from the Connelly survey.

Another potential concern mentioned in comments and the HHRA is that the fishing advisories suppress consumption and therefore suppress the potential intake rate. The HHRA states that the effect of general fishing advisories (e.g., 52 meals per year or less) for New York are taken into account because these were in place during the Connelly survey (HHRA, page 46). The effect of repressing consumption would be constant throughout the state and, unless the state is considering removing these general advisories, the suppressive effect would continue into the future.

Reviewers were asked to comment on the use of the 50th and 90th percentiles for fish ingestion used for the central tendency and reasonably maximally exposed (RME) individuals. I have presented all the other questions that may be important to address concerning the fish that would be eaten today and who is eating them. While the Connelly data may not have provided the best data on subpopulations that have been a concern to those creating fish advisories, the survey does to provide the highest quality data on meal frequency to use in the HHRA. The values of 4 and 32 grams per day can be defended for adults.

(3) Comment of the assumption of a 12 and 40 year exposure durations (HHRA pages 23, 49-57) for use, respectively, of average and RME individuals (used in the point estimate for cancer).

The HHRA (Table 2-12) uses a exposure duration of 40 years of exposure to 2.2 mg PCBs/kg fish averaged over a 70 year lifetime to create the RME dose of mg PCB/kg body weight/day for cancer risk estimates. The central tendency estimate uses 12 years of exposure to 4.4 mg PCBs/kg fish averaged over a 70 year lifetime.

These exposure values appear based on reasonable interpretations of fish survey and residence data and reasonable assumptions concerning movement within a relatively small geographic area. Since the risk assessment is concerned with incremental risk from the Hudson River fish and not incremental risk from all sources of PCB-contaminated fish, it appears appropriate to ignore an angler's exposures to PCBs before 1999 or after moving away from the river. These other PCB exposures will hopefully be considered by risk managers interpreting the results in the broader context of PCB exposures from multiple sources. It is clear from the responsiveness summary that some who submitted comments do not trust risk managers to keep in perspective that this is an incremental risk that does not address past exposures to fish contaminated from the same or alternative sources of PCBs.

Cogliano reviewed a study in rats that included less-than-lifetime exposures to Aroclor 1260 (via food) and measured cancer incidence. The results suggested that cancer risk measured at two years could be attributed to the exposures that occurred during the first year of dosing (Cogliano, 1998). Some consideration should be given to whether or not these less-than-lifetime dose response findings are adequately addressed in the HHRA and reflected in the choice of a 70 year averaging time for exposure duration. The other Aroclor mixtures administered in cancer studies did not show this same potency for less than lifetime exposures.

(4) Comment on the average concentration in fish for 7, and 12 years used in the non-cancer point estimates (HHRA pages 67-68).

The HHRA (Table 2-12) uses an exposure duration of 7 years to calculate a high-end fish PCB concentration and averages the exposure over 7 years. Likewise, for the central tendency, the HHRA uses an exposure duration of 12 years and averages the exposure over 12 years.

The span of years for averaging exposures that is described in Risk Assessment Guidelines for Superfund is not specific for the chemical of concern, the toxicologic endpoint of concern, and the environmental conditions. The HHRA thoroughly discusses the decrease in PCB levels in biota over time and the need to represent current levels in the HHRA. The toxicologic endpoints of concern for the RfD used in the point estimate result from exposures during 25 percent of the life span of rhesus monkeys. This is equivalent to a period in a human life between approximately 4 and 23 years of age. Other endpoints of concern (reproductive and fetal development) may result from very short-term exposures.

The length of the exposure duration and averaging time is appropriate for the toxicological endpoints. Each of the selected exposure and averaging times results in an unacceptable hazard quotient. Since there is no attempt to describe health effects associated with a hazard quotient greater than 1, the choice of averaging times in combination with fish tissue levels does not appear to be worth discussing further.

(5) Comment on the Monte Carlo analysis for the fish ingestion pathway (HHRA pages 72-74)

I am inexperienced with Monte Carol analysis but have discussed use of the angler surveys in the analysis (see the next question). There was remarkable effort to work out the out-migration and residence data of anglers. In contrast, it appeared that the fish data were condensed (?) into a few samples (table 5-24?), data were reduced to a few species, and grouped into a few river reaches (Table 3-4). The river must certainly be a more complex system than portrayed in the HHRA.

I cannot tell whether the Monte Carol analysis would have been different or improved by separating meal size and meal frequency parameters rather than using a distribution for ingestion

Shubat,

rate. I would have preferred treating the meal size (0.5 pounds) as a constrained parameter (Equation 3-1) and using the New York Angler Survey to create the variable for frequency of meals. By using meal size as a constrained parameter, it might have been easier to understand the effect of varying meal size based on age and body weight.

The work that went into the Monte Carlo analysis was best described in the risk characterization section on uncertainty, pages 77-80. It was difficult to understand all of the inputs and permutations that were modeled and it is not clear that these were adequately described in the HHRA.

(6) Comment on the use of the angler surveys in the Monte Carlo analysis (HHRA 37-46).

As discussed above, Figure 3-1 of the HHRA shows that body weight and fish ingestion rate are treated as independent distributions. The HHRA (page 46) stated that the same number of meals per year was used for adults as for children, but scaled according to body weight. It is not clear from the text (page 46) what this means and seems contradicted by the description of meal size on page 42. It is not clear how meal size should be scaled to body weight (see description of the West survey data, above, under "meal size").

The Monte Carlo analysis appears to be responsive to a the concern that anglers preferentially fish and eat certain species of fish (Table 3-3 and text page 48). However, the assumed fish species consumed may not apply to the Upper Hudson if angling for food is assumed. While it is not appropriate to use local surveys conducted while local fish advisories are in place in order to determine ingestion rates, it may be appropriate to use these surveys to examine angler preference for fishing certain species. This is because the fishery itself will have a large impact on angler preference for species. The HHRA did not present this type of data or angler perception of the fishery in the Hudson. It is interesting that very little information on fishery and angler management shows up in the HHRA.

Risk Characterization

(7) Comment on whether the risk characterization adequately estimates risks and hazards (chapter 5, pages 67-80).

The risk characterization is a very straight-forward combination of the dose-response and exposure data. It does not over state the non-cancer health effects by attempting to interpret the meaning of hazard quotients greater than 1, 10 or 100.

The risk characterization clearly lays out the concerns that went into choosing exposure durations of 7, 12, and 40 years, and the effect of these different choices.

I believe there could be greater depth and clarity in the explanation of the impact of using central and upper confidence limit PCB cancer potency slopes in the point estimates (HHRA page 64). The relationship between these values and the results of the Monte Carlo analysis is briefly mentioned on page 71, but further interpretation is not offered where I thought readers would look for information (in Section 5.3.3, comparison of point estimate RME and Monte Carlo results). It is not clear whether these choices are explained sufficiently for decision-making by risk managers.

General Questions

(1) Is the HHRA clear, consistent, reasonable, and transparent, as well as adequate (including children). How adequate are the HHRA and Responsiveness Summary when measured against these criteria?

I would have liked to see the relationship between modeled fish tissue concentrations, fish ingestion rates, and hazard quotients/cancer potency slopes also expressed as the length of time before fish would fall into fish advisory categories of 1 meal per month or 1 meal per week. In other words, the fish tissue concentration modeling would have been more meaningful, and consistent with the applied use of risk assessment in fisheries management, if the HHRA had also reported *when* the fish will be "safe" to eat according to current guidelines.

Although I believe the HHRA is adequate, focused in scope, and probably the best possible analysis using Risk Assessment Guidance (RAGs), there was no overall description of what was

important to pursue with detailed analysis and what was not. There were, however, elements of this type of critical thinking in responding to comments. This was a valuable part of the responsiveness survey (although, I'm sure irritating to the authors of the comments). For example, there was discussion that it was not necessary to calculate exposures/risks from some pathways because the contribution to risk was very small. An overall description of what was important to pursue with detailed analysis and what could be dismissed would have been helpful. Overall, I found the responsiveness summary a very important part of the HHRA and it clarified actions taken and assumptions made in preparing the HHRA.

The HHRA does not present an adequate discussion of risks to children, adolescents, or the fetus either in terms of exposure or toxicologic endpoints. There are unique food intake and toxicologic factors for children and adolescents that might have been discussed. No discussion is given to the results for children versus other age groups in the Monte Carlo analysis. While children as consumers of fish are included in the Monte Carlo analysis, *in utero* exposure is not discussed. No discussion is presented in the risk characterization about who should be protected when the toxicologic endpoint for Aroclor 1016 is reduced birth weight resulting from exposure to the dam, or that the RfD for Aroclor 1254 is based on a study of monkeys exposed during the human equivalent of approximately 4 to 23 years of age. Adolescence is not specifically discussed and is a time in development that may be uniquely susceptible to immunotoxicants (Golub, 2000). A body of work is available on the effects of PCBs on thyroid hormone metabolism that may be relevant to this concern (Brouwer et. al., 1999).

(2) Please provide any other comments or concerns, strengths or weaknesses.

This was a well-written document. The language was clear and direct, the jargon was minimized, and the only thing missing for ease of reading by a general audience of scientists was a glossary of terms. The text was laid out in a logical fashion that followed standard risk assessment protocol. The tables and figures would have been improved with additional text in the titles or footnotes so that they might be more easily understood by someone browsing through the section. It was apparent in the answers to questions posed by reviewers that the data analysis was more complex and complete than presented in this document. More references to other documents might have been helpful.

Shubat,

There were constraints on the authors because standard Risk Assessment Guidance (RAG) for superfund sites was used to create the risk assessment. Some of these constraints were discussed, but probably not often or clearly enough. The authors could clearly anticipate where risk assessment practices are changing and in potential conflict with the RAG. A strength of the HHRA is that the authors utilize an appropriate conservative approach to interpreting non-cancer health effects. For example, there is no speculation about health implications of a hazard quotient of less than or more than 1.

I am impressed by the vast amount of effort that has gone into this site characterization over the years. Every question that came to my mind while reading the HHRA was also posed in a comment letter.

Recommendations

My views are likely to change after discussion with the other reviewers as I have little experience with some of the important parts of the HHRA that I assume will be discussed at the meeting.

At this time, I believe the risk assessment is acceptable with minor revisions. Those revisions will not result in changes in the inputs or outputs of the risk assessment, but would show up as a discussion of uncertainties.

1) Discussion of how the risk assessment fails or succeeds in addressing exposure and toxicology factors unique to children, adolescents, and fetuses. This should include a discussion of the intake per body weight of food. It should include a discussion of how the cancer and non-cancer health studies selected by EPA for the IRIS files specifically address children. **The uncertainty to address is whether or not risks to children, adolescents, and fetuses are adequately characterized in the risk assessment.**

2) Discussion of the demographic makeup of the statewide potential angler population in 1991 and the specific characteristics of those surveyed by Connelly. This discussion of what was happening in 1991 should include what anglers were and were not required to purchase licenses and therefore made up the pool of potential survey participants. The discussion should also should include a comparison with the current demographic makeup of anglers described in the

current HHRA. With all the detail used on migration in and out of surrounding counties, these data must be readily available. The data should specifically address income, race, ethnicity, and literacy. **The uncertainty to address is whether the pool of licensed anglers surveyed in 1991 matches the pool of current, potential, anglers in the counties surrounding the river.**

3) General discussion of fisheries management for the river and its tributaries. This would include a discussion of the fish species present in the river and tributaries, commercial fishing, and angler perception of fishing this river system (desirable species, perception of abundance of fish). It should also include a discussion of the findings of enforcement programs on the current catch and release fishery. **The uncertainty to address is whether the fish species and sizes used to create the exposure data matches the fish that are likely to be taken from the river today.**

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Lee Shull

Hudson River PCBs Human Health Risk Assessment Peer Review Panel

Comments by: Lee R. Shull, Ph.D., NewFields, Inc.
May 12, 2000

Responses to: Specific Questions

Hazard Identification/Dose Response

Question 1)

"Consistent with its risk assessment guidance (USEPA, 1993), USEPA considered scientific literature on PCB toxicity, both as to cancer and non-cancer health effects, published since the 1993 and 1994 development of the non-cancer reference doses (RfDs) for Aroclor 1016 and Aroclor 1254, respectively, and since the 1996 reassessment of the cancer slope factors (CSFs). Based on the weight of evidence of PCB toxicity and due to the Agency's ongoing reassessment of the RfDs, USEPA used the most current RfDs and CSFs provided in the Integrated Risk Information System (IRIS), which is the Agency's database of consensus toxicity values. The new toxicity studies published since the development of the RfDs and CSFs in IRIS were addressed in the context of uncertainty associated with the use of the IRIS values (see, HHRA, pp. 76-77 and Appendix C). Please comment on the reasonableness of this approach for the Upper Hudson River."

Response

My response to this question is based on USEPA's definition of 'reasonableness' as defined in the agency's DRAFT Risk Characterization Handbook (1998). Although this document is DRAFT, I believe definition of 'reasonableness' and the five criteria provided by USEPA to evaluate the reasonableness of risk assessments can be applied to this question. It should be noted that USEPA's 1995 Policy for Risk Characterization does not define 'reasonableness' *per se*. The five criteria in the 1998 Handbook that define 'reasonableness' of risk characterizations are:

- i. 'the risk characterization is determined to be sound by the scientific community...because the components of the risk characterization are well integrated into an overall conclusion of risk which is complete, informative, well balanced and useful for decision making,
- ii. the characterization is based on the best available scientific information,
- iii. the policy judgments required to carry out the risk analyses use common sense given in statutory requirements and Agency guidelines,
- iv. the assessment uses generally accepted scientific knowledge, and
- v. plausible alternative estimates of risk under various candidate risk management alternatives are identified and explained.'

Applying these criteria (only 1-4 apply) to the toxicity criteria used, I conclude that neither the RfDs nor CSFs used in the HHRA are reasonable.

- i. *The toxicity criteria have not been determined to be sound by the scientific community.*
In fact, both criteria have been extensively criticized within the scientific community. Good

summaries describing the major toxicological issues and lack of consensus on scientific interpretation of data regarding both the cancer and non-cancer endpoints for PCBs are given in comments made on the HHRA (Responsiveness Summary, Volume 2F - Human Health Risk Assessment). In particular, comments by Exponent on behalf of Chemical Land Holdings and by General Electric describe many of the disparate views on PCB toxicity, and what are scientifically defensible toxicity criteria for use in human health risk assessment.

- ii. *The toxicity criteria are not based on the best available science.* Again, I do not believe best available science has been employed by USEPA in establishing these criteria. Valuable epidemiological information has been ignored. Too much emphasis is placed on animal toxicity data instead of using human epidemiological data, which is substantial.
- iii. *The toxicity criteria more or less are based on statutory requirements and Agency guidelines.* Although the toxicity criteria used in the HHRA possess serious scientific deficiencies, I believe USEPA has generally followed statutory and Agency guidelines in deriving these criteria (i.e., the process employed is more or less the same as used in deriving criteria for many other chemicals).
- iv. *The toxicity criteria are based only partially on generally accepted scientific knowledge.* As already stated, I believe USEPA has not included valuable toxicological and epidemiological information in deriving both the cancer and non-cancer criteria. Reasons are numerous and have been summarized well in the comments of others on the HHRA.

USEPA counters criticism of using obsolete toxicity criteria by addressing the impact of this deficiency as part of the uncertainty assessment. The inclusion of the toxicity criteria in to the uncertainty assessment is important and is essential to risk managers making decisions about the Upper Hudson River. However, I believe the discussion of uncertainty regarding the toxicity criteria is poorly organized and fails to communicate essential information in a concise way to decision makers.

In addition, I believe the toxicity assessment section (4.0) and Appendix C (PCB Toxicological Profile) could be greatly improved and should be updated. I agree with the comments of several commenters in the 'Responsiveness Summary for Volume 2F - Human Health Risk Assessment', that the discussion of Toxicology and Epidemiology information is out of date and incomplete.

In summary, I do not believe it is reasonable for USEPA's to address the new toxicity studies published since the development of the RfDs and CSFs in IRIS in the context of uncertainty associated with the use of the IRIS values. A critical question is whether it is reasonable for USEPA to update cancer and non-cancer toxicity criteria incorporating new information before finalizing this HHRA. On a scientific basis, the answer is YES. On a policy basis, the answer may not be yes. Clearly, confidence in decision making based on the HHRA (and the toxicity criteria used) can be greatly improved by (1) updating Section 4 and Appendix C, and (2) providing a more concise and deliberate presentation of uncertainty regarding the criteria in Section 5.3, notably 5.3.2.

Exposure Assessment

Question 2)

"Since 1976, the New York State Department of Health has issued fish consumption advisories that recommend not eating fish caught in the Upper Hudson River. To generate a fish ingestion rate for anglers consuming fish from the Upper Hudson River under baseline conditions (i.e., in the absence of the fish consumption advisories), USEPA used data on flowing water bodies in New York State (1991 New York Angler survey, Connelly et al., 1992) to derive a fish ingestion rate distribution. The 50th and 90th percentiles were used for the fish ingestion rates for the central tendency (average) and reasonably maximally exposed (RME) individuals (i.e., 4.0 and 31.9 grams per day, equivalent to approximately 6 and 51 half-pound meals per year, respectively) (see, HHRA, pp. 24 and 37). Please comment on whether this approach provides reasonable estimates of fish consumption for the central tendency and RME individuals for use in the point estimate calculations."

Response

Again, my response to this question is based on USEPA's definition of 'reasonableness' as defined in the agency's DRAFT Risk Characterization Handbook (1998) and discussed in the response to Question 1.

Applying these criteria, I conclude that the fish ingestion rates used in the HHRA are not reasonable.

- i. *The fish ingestion rates have not been determined to be sound by the scientific community.* EPA does base the fish ingestion rates on a peer reviewed scientific study (Connelly et al. 1992). However, summaries describing the additional interpretation of data regarding fish ingestion are given in comments made on the HHRA (Responsiveness Summary, Volume 2F - Human Health Risk Assessment). Comments by Exponent on behalf of Chemical Land Holdings and by General Electric in particular present evidence for using lower fish ingestion rates.
- ii. *The fish ingestion rates are not based on the best available science.* I do not believe EPA used the best available science for establishing fish ingestion rates. EPA discounted important fish ingestion rate information. EPA relied too heavily on the Connelly et al. (1992) data to derive the fish ingestion without utilizing relevant information regarding more applicable fish ingestion rates.
- iii. *The fish ingestion rates more or less are based on statutory requirements and Agency guidelines.* Although the fish ingestion rates over-estimate the exposure to PCBs by fish ingestion, I believe USEPA has generally followed statutory and Agency guidelines in deriving these exposure rates (i.e., the process employed is more or less the same as used in deriving exposure rates for many other chemicals).
- iv. *The fish ingestion rates are based only partially on generally accepted scientific knowledge.* Again, I believe the USEPA has not included valuable information regarding the development of a reasonable fish ingestion rate.

The use of the Connelly et al. (1992) data does not take into account fish ingestion rates which may change from year to year and the individual who may not consume fish every year. In

addition, the fraction from source (Hudson River) of fish ingestion was assumed to be 100%. Although one might expect that anglers may prefer this area if there were no advisories or restrictions, I disagree with the use of 100%. They state in several of the studies used to support exposure duration (3.2.4) that anglers reported that they traveled an average of 34 miles to fish. There are many waterways, both flowing and standing (lakes) within 34 miles of the Hudson that likely support fishing. The assumption of 100% of all fish taken are from the study area seems high. Although USEPA presents evidence from surveys that illustrate the tendency of a large percentage of individuals to fish the same water body, it is unclear whether this assumption would hold true throughout the exposure duration of an angler.

The use of the Connelly et al. (1992) data results in conservative and unrealistic estimates of fish ingestion. The following items provide an overview of problems with the use of the Connelly et al. (1992) data.

- The entire distribution of the Connelly et al. (1992) data is not used. Only non-zero data were used to generate the fish ingestion rate distributions.
- Although the intent is to assess fish ingestion assuming no "health based" restrictions on catch, assuming that all anglers eat all their catch seems overly conservative as well.
- It doesn't account for anglers who are strictly catch and release.
- The ingestion data (Connelly et al., 1992) is based on a State-wide survey, not just Upper Hudson data, so the data is not strictly biased towards those who fish the Hudson but also fish other fisheries, and some of these anglers clearly don't eat their catch. Throwing out zero values seems to assume that the consumption data is strictly related to the Hudson area and it clearly is not.
- There are State wide conservation-based fishing limitations/advisories that have nothing to do with the health advisories for the upper Hudson fishery that encourage people to catch and release without consuming their catch.
- In light of the use of the fraction from source of fish value of 100%, it's especially conservative.
- The Connelly data is based on mail recall survey, which the authors admit may be high due to recall bias (see section 3.2.1.4).
- Use of non-zero values assumes that the recommendations for conservation based limitations are never effective, which is conservative...there are many hunters and anglers who are conservationists as well.
- Further supported by the fact that in the 1996 and 1991-1992 Hudson River Surveys only 2/3 of the people were aware of the health advisories for fish on the Hudson, yet 92% reported never eating their catch, indicating that a significant number of those not eating their catch were not aware of the health advisories and lack of consumption is not health advisory related.

Based on the conservation related fishing advisories, I suggest (as did GE) that the distribution should be truncated at 32 g/day, or at least adjusted to account for catch and release or consumption of fish from other waterways.

In Section 2.3.1, page 14, "PCB concentration weighted by species", it is made clear that several species identified in the Connelly study used to estimate intake rates are not commonly present or caught in the Upper Hudson study area. These species (trout, salmon, bullhead, and "other") were removed from the analysis to estimate the average exposure concentration of

PCBs in fish tissue. However, it is unclear in this section or in Section 3 pertaining to the fish ingestion rates whether any attempt was made to remove the influences of these same species on the ingestion rate, as they apparently contributed upwards of 62% of the species reported in the Connelly et al. (1992) study as being consumed. If not, then the estimate of fish ingestion rates of these Hudson river-specific species may have been overestimated. Please provide additional clarification on this issue.

In summary, USEPA's approach does not provide reasonable estimates of fish consumption for the central tendency and RME individuals for use in point estimate calculations.

Question 3)

"Superfund risk assessments often assume a 30-year exposure duration, based on national data for residence duration. However, because an angler could move from one residence to another and still continue to fish the 40 mile-long Upper Hudson River, USEPA developed a site-specific exposure duration distribution based on the minimum of residence duration and fishing duration. The residence duration was based on population mobility data from the U.S. Bureau of Census (1990) for the five counties that border the Upper Hudson. The fishing duration was developed from the 1991 New York Angler survey (Connelly et al., 1992). The 50th and 95th percentiles of the distribution were used for the central tendency (average) and RME exposure durations (i.e., 12 and 40 years, respectively). Please comment on the adequacy of this approach in deriving site-specific exposure durations for the fish ingestion pathway (see, HHRA, pp. 23 and 49-57)."

Response

My response is again based on USEPA's definition of 'reasonableness' as defined in the agency's DRAFT Risk Characterization Handbook (1998).

Applying these criteria, I conclude that the fish ingestion exposure durations used in the HHRA are adequate.

- i. *The exposure durations have been determined to be sound by the scientific community.* Several comments on the HHRA (Responsiveness Summary, Volume 2F - Human Health Risk Assessment) addressed concerns over the developed exposure durations. However, given the data constraints in constructing the exposure durations and the methodology applied, overall more reasonable approaches were not available for USEPA.
- ii. *The exposure durations are based on the best available science.* USEPA used the best available information in developing the exposure durations. The derivation of site-specific exposure durations seems reasonable given the data constraints.
- iii. *The exposure durations more or less are based on statutory requirements and Agency guidelines.* I believe USEPA has generally followed statutory and Agency guidelines in deriving these exposure durations (i.e., the process employed is more or less the same as used in deriving exposure rates for many other chemicals).
- iv. *The exposure durations are based on generally accepted scientific knowledge.* Again, I believe the USEPA has included valuable information regarding the development of reasonable exposure durations. I agree with the use of exposure durations different than residential exposure durations.

My response is also based on USEPA's definition of 'transparency' as defined in the agency's DRAFT Risk Characterization Handbook (1998). This refers to the "transparency in the risk assessment process. Making the process open and frank helps make the default policy known and helps achieve full disclosure." The transparency relates to many parts of the assessment including assumptions, extrapolations, models, and choices made during the risk assessment process and the impacts they have on the assessment.

The transparency of the approach to derive exposure durations needs to be improved in the document. The approach used appears reasonable given the data constraints. However, the approach is involved and the results cannot be reproduced with the data presented in the tables (although it may not be possible to give the census data). In addition, it is not clear in the document the impact of the many variables used on the resulting exposure durations. For example, is it likely the age at which a person started fishing may over or underestimate risk? This assumption does not account for when people started fishing in this particular fishery.

Overall, I feel the USEPA derivation of site-specific exposure durations for the fish ingestion pathway is adequate.

Question 4)

"PCB concentrations in Upper Hudson River fish generally have declined in past decades and the decline is expected to continue into the future. Therefore, to evaluate non-cancer effects for the RME individual, USEPA used exposure point concentration in each medium (water, sediment, and fish) based on the average of the concentrations forecast over the next 7 years (1999 to 2006), which gives the highest chronic dose considered in the HHRA. For the central tendency exposure point concentrations, USEPA used the average of the concentrations forecast over 12 years (1999 to 2011), which is the 50th percentile of the residence duration developed from the population mobility data (U.S. Bureau of Census, 1990). In addition, for completeness, USEPA averaged the exposure concentration over 40 years (1999 to 2039) to evaluate non-cancer hazards for the same time period over which cancer risk was calculated. Please comment on whether this approach adequately addresses non-cancer health hazards to the central tendency and RME individuals (see, HHRA, pp. 67- 68)."

Response

My response is again based on USEPA's definition of 'reasonableness' as defined in the agency's DRAFT Risk Characterization Handbook (1998).

Applying these criteria, I conclude that USEPA's approach to estimate fish exposure point concentrations used in the HHRA is reasonable.

- i. *The exposure point concentrations have been determined to be sound by the scientific community.* Comments on the modeling used to estimate fish concentrations do support the conceptual basis of the models used.
- ii. *The exposure point concentrations are based on the best available science.* Although full peer review of the modeling may not be complete, this reviewer cannot find fault with science used in developing the models used for media concentrations into the future.

Although the models are not appropriately validated for the use in estimating concentrations for long durations into the future, USEPA did use the best data available to achieve some level of validation, which was based on a data set from 1 year of monitoring.

- iii. *The exposure point concentrations more or less are based on statutory requirements and Agency guidelines.* I believe USEPA has generally followed statutory and Agency guidelines in deriving exposure point concentrations (*i.e.*, the process employed is more or less the same as used in deriving exposure rates for many other chemicals). The selection of the seven year average as the RME chronic exposure concentration is the most conservative approach, as seven years is at the low end of what would be considered a chronic exposure as opposed to sub-chronic exposure.
- iv. *The exposure point concentrations are based on generally accepted scientific knowledge.* Again, I believe the USEPA has included valuable information regarding the development of reasonable exposure point concentrations. I commend the USEPA for the attempt to account for declining PCB concentrations into future.

The transparency and clarity of the approach presented in the HHRA need to be improved. The seven-year average is the maximum concentration that could be used with chronic toxicity information. The conservativeness of this selection is not presented. In addition, the uncertainties from the modeling results are not clearly presented. The attempts to validate the model were limited to a one-year data set. While the USEPA used the best available data set for the validation, it is still not truly appropriate to validate a long-term model using such short-term conditions. In addition, the uncertainties from model compounding are not adequately addressed. The overall prediction of PCB concentrations in fish is based on the use of several models in series. These combined uncertainties are not adequately addressed. In addition, validation attempts seem to only have been completed for individual models and not on the use of the series of models that is ultimately used to develop fish exposure point concentrations. These sources of uncertainty need to be transparent and discussed clearly in the document.

Overall, I feel the USEPA's approach adequately addresses non-cancer health hazards to the central tendency and RME individuals. However, the HHRA should be improved to address the issues of transparency and clarity.

Monte Carlo Analysis/Uncertainty Analysis

Question 5)

"USEPA policy states that probabilistic analysis techniques such as Monte Carlo analysis, given adequate supporting data and credible assumptions, can be viable statistical tools for analyzing variability and uncertainty in risk assessments (USEPA, 1997a). Consistent with this policy, USEPA used a tiered approach to progress from a deterministic (*i.e.*, point estimate) analysis to an enhanced one-dimensional Monte Carlo analysis of the fish ingestion pathway (see, HHRA, Chapter 3, pp. 33-59). Please discuss whether this Monte Carlo analysis makes appropriate use of the available data, uses credible assumptions, and adequately addresses variability and uncertainty associated with the fish ingestion pathway (*e.g.*, defining the angler population, PCB exposure concentrations, ingestion rates, exposure durations, cooking losses) qualitatively or quantitatively, as appropriate, in the analysis (see, HHRA, pp. 72-74)."

Response

My response is based primarily on USEPA's definition of 'reasonableness' as defined in the agency's DRAFT Risk Characterization Handbook (1998). The criteria is used to evaluate the several issues raised in question 5.

Applying these criteria, I conclude that the use of available data for fish ingestion rates and cooking losses are not reasonable.

- i. *The parameters have not been determined to be sound by the scientific community.*
Comments on and review of the use of data for fish ingestion and cooking loss indicate the presence of significant data not used in the HHRA.
- ii. *The parameters are not based on the best available science.* Again, there are significant data available that the USEPA has chosen not to utilize.
- iii. *The parameters more or less are based on statutory requirements and Agency guidelines.* I believe USEPA has generally followed statutory and Agency guidelines in deriving these parameters for probabilistic assessment (*i.e.*, the process employed is more or less the same as used in other HHRA's).
- iv. *The parameters are based only partially on generally accepted scientific knowledge.*
Again, I believe the USEPA has excluded valuable information regarding the development of fish ingestion rates and cooking losses.

The issue of fish ingestion rates was addressed in comments to question number 2. Please refer to those comments for more detail regarding my concerns on the over reliance on fish ingestion data from Connelly et al. (1992).

For RME exposures, a value of 0% cooking loss is assumed. For the central tendency exposures a value of 20% loss is used and for lower end exposures, a value of 40% was used. There is clearly plenty of data on the cooking loss of PCBs, which is presented in the back of the report. This data should be used to develop an RME for cooking loss based on the data. The use of 0% cooking loss is not "RME", but rather worst case. The use of 0% is unreasonable especially in light of the other conservative measures used in the report.

The Monte Carlo analysis did make appropriate use of available and relevant data in development of fish ingestion exposure durations. Please refer to my comments for question number 3 in regards to my conclusion that the exposure durations developed are appropriate.

Overall, the Monte Carlo analysis does not make appropriate use of fish ingestion and cooking loss available data. However, the Monte Carlo analysis for fish ingestion exposure duration did make appropriate use of the available data.

Question 6)

"For the Monte Carlo analysis, USEPA evaluated a number of angler surveys, but excluded local angler surveys, such as the 1996 and 1991-1992 Hudson Angler surveys (NYSDOH, 1999; Barclay, 1993), due to the fish consumption advisories. The 1991 New York Angler survey (Connelly et al., 1992) was used as the base case and other surveys were used to address

sensitivity/uncertainty in fish ingestion rates (see HHRA, pp. 37-46). Please comment on the adequacy of USEPA's evaluation and use of existing angler surveys in the Monte Carlo analysis of the fish ingestion pathway."

Response

My assessment is that the reliance upon the Connelly et al. (1992) survey is unreasonable. Please refer to my comments for questions 2 regarding the assessment and development of fish ingestion rates. The overall assessment that the developed fish ingestion rates are unreasonable is based on the following.

- i. *The fish ingestion rates have not been determined to be sound by the scientific community.* USEPA does base the fish ingestion rates on a peer reviewed scientific study (Connelly et al. 1992). However, summaries describing the additional interpretation of data regarding fish ingestion are given in comments made on the HHRA (Responsiveness Summary, Volume 2F - Human Health Risk Assessment). Comments by Exponent on behalf of Chemical Land Holdings and by General Electric in particular describe evidence for using lower fish ingestion rates.
- ii. *The fish ingestion rates are not based on the best available science.* I do not believe EPA used the best available science for establishing fish ingestion rates. EPA discounted important fish ingestion rate information. EPA relied too heavily on the Connelly et al. (1992) data to derive the fish ingestion rates without utilizing relevant information regarding more reasonable fish ingestion rates.
- iii. *The fish ingestion rates more or less are based on statutory requirements and Agency guidelines.* Although the fish ingestion rates over-estimate the exposure to PCBs by fish ingestion, I believe USEPA has generally followed statutory and Agency guidelines in deriving these exposure rates (*i.e.*, the process employed is more or less the same as used in deriving exposure rates for many other chemicals).
- iv. *The fish ingestion rates are based only partially on generally accepted scientific knowledge.* Again, I believe the USEPA has not included valuable information regarding the development of a reasonable fish ingestion rate.

The use of the Connelly et al. (1992) study is overly conservative. There are several trends in fish consumption that are exhibited in other studies but were disregarded in the development of the fish ingestion rate. The use of Connelly et al. (1992) comes with several assumptions, which include the following:

- An angler's ingestion rate is consistent for the exposure duration period.
- Anglers who may consume fish caught less than once year are to be excluded.
- Evidence that anglers fish the same system during a season can be extrapolated to the exposure duration period.

USEPA does not provide sufficient evidence or rationale for making these assumptions nor are these assumptions clearly stated.

Risk Characterization

Question 7)

“The risk characterization section of the HHRA (Chapter 5, pp. 67-80) summarizes cancer risks and non-cancer hazards to individuals who may be exposed to PCBs in the Upper Hudson River. Please comment on whether the risk characterization adequately estimates the relative cancer risks and non-cancer hazards for each pathway and exposed population. Have major uncertainties been identified and adequately considered? Have the exposure assumptions been described sufficiently?”

Response

Overall the Risk Characterization is lacking. It is missing key components as well as discussions on important sources of uncertainty. The following issues need to be addressed in order for the Risk Characterization to be a sufficient and transparent source of information for decision makers.

- A standard qualitative uncertainty analysis summary table should be included in Section 5.3. An example table is included with my comments (See Table 1). USEPA risk assessors should identify each potential source of uncertainty and subsequently estimate, using professional judgment and knowledge of scientific information, whether the item would result in a under-estimation or over-estimation of risk. In addition, whether the extent of under- or over-estimation would be expected to be low, medium or high should be included in the table. With this information, the decision maker is better able to incorporate uncertainty into decision-making. Table 2 is shown as an example using the sources of uncertainty in selection of toxicity criteria. If the majority of sources of uncertainty regarding toxicity are found to lead to an over-estimation of risk, which is how I believe it would come out, the decision maker has greater confidence in including uncertainty into decisions.

Table 1. Sample Uncertainty Analysis Summary Table

Source of Uncertainty	Results in Under-estimation of Health Risk			Results in Over-estimation of Health Risk		
	Low	Medium	High	Low	Medium	High
Use of animal data					✓	
Exclusion of scientifically valid epidemiological data					✓	
Etc.						

- The risk characterization focuses more on the deterministic rather than probabilistic results. This is counter to the purpose and intent of performing a probabilistic assessment.
- The HHRA does not contain a conceptual site model (CSM). The purpose of developing a CSM is to provide discussion for the selection of receptors as well as receptor pathways. The HHRA does not provide any reasonable explanation for the exclusion of receptors or pathways. For example a breast milk pathway was not evaluated, but from the HHRA it is unclear as to why. Without a properly developed CSM the risk characterization does not provide decision-makers the ability to judge that all relevant and important pathways were evaluated.
- Section 5.1.3. There is no discussion on estimating the intakes of dioxin-like PCBs in the exposure assessment section. This discussion belongs in section 3 as well. It is confusing that the discussion appears for the first time in the risk characterization section.
- Section 5.2, first paragraph. There is a reference to Section 3.5.1. There is no section 3.5.1.
- Section 5.2. There is no discussion of whether the dioxin-like PCB risks were estimated in the Monte Carlo analysis as it was in the point estimate analysis. There should be some discussion of whether this was done, and if not, rationale for not doing this type of analysis in the Monte Carlo analysis as well.
- The fish ingestion rate selected and the assumption that all fish tissue comes from the Upper Hudson is a very conservative assumption. There is no discussion regarding the possibility of or uncertainties associated with people who consume fish from other water bodies, and that all of their fish consumption from sport angling may not come from the Upper Hudson.
- The use of data to develop the fish ingestion exposure duration assumes that the reported age that a person started fishing is the age for starting fishing at the Hudson. There is no discussion related to the uncertainties associated with this assumption. It is possible that the age a person started fishing is not the age they started fishing at the Hudson. It is not clear how this data was used.
- There is no discussion about using a 100% of fish ingestion fraction from the source value and its impact on the uncertainty in the outcome.
- There is little or no discussion about the uncertainties associated with the exposure values held constant in the Monte Carlo analysis (body weight, for example) or used in the point estimate values (the use of certain values and what percentiles they represent, e.g. inhalation rate).
- Fish ingestion rates. "Although the fish ingestion rates reported in the New York Angler survey are presumably influenced by general, non-specific NYSDEC fishing regulations (that would be in effect regardless of PCB contamination levels in the Hudson)..." There is no discussion on what the effect of throwing out the zero values from the original data may have had. It is possible that some of these data points may have represented anglers who consume fewer fish per year as a result of conservation. Some discussion relating to this is warranted, especially in light of the fact that a source fraction value of 100% is used. There is insufficient discussion of the uncertainties associated with the assumption that the angler will consume at least 1 fish meal per year. There are some that may consume fewer, as in 1 every two years.

Response to: General Questions

Question 1)

“A goal for risk assessments is that they be clear, consistent, reasonable and transparent and adequately characterize cancer risks and non-cancer hazards to the exposed population, including children (USEPA, 1995b, 1995d). Based on your review, how adequate are the HHRA and Responsiveness Summary when measured against these criteria?”

Response

In general, I think the HHRA is well done, even though it lacks seriously in some areas as pointed out in previous comments (e.g., the uncertainty section is poorly done, lacks state-of-the-art toxicity criteria, CSM, etc.). Specifically, the transparency and reasonableness of the HHRA are inadequate and need to be improved. My evaluation of whether the HHRA is transparent, clear, consistent, and reasonable is illustrated in Table 2.

Table 2. Transparent, Clear, Consistent, And Reasonable Assessment

Criteria	Assessment	Comment
Transparency	Deficient	<ul style="list-style-type: none">○ Explanations of key assumptions○ Monte Carlo - Identifying sources of all data○ Provide enhanced uncertainty discussion
Clarity	OK	Uncertainty analysis needs improvement
Consistent	Good	Generally consistent with other USEPA HHRAs
Reasonable	Deficient	Reasonable approaches used: <ul style="list-style-type: none">○ Exposure duration○ Fish exposure point concentration Unreasonable approaches used: <ul style="list-style-type: none">○ Toxicity Criteria○ Fish ingestion rates○ Cooking loss

Question 2)

“Please provide any other comments or concerns, both strengths and weaknesses, with the HHRA not covered by the charge questions, above.”

Response

The following are additional comments ordered by section and page of the HHRA

- Executive summary Page 2 paragraph 2. It is not clear whether the drinking water standards used to evaluate the river water are health based. If the standards are not health based, the point estimates for incidental ingestion while swimming should be included.
- Executive summary Page 3 paragraph 1. Please briefly clarify the use of 10 years old as the beginning age for fishing.
- Executive summary Page 3 paragraph 1. Please be more specific on how surveys of fish ingestion rates in states other than New York were included in examining fish ingestion variability.
- Executive summary Page 3 paragraph 3. It should be stated that the basis for the exposure frequency assumptions will be explained later. Also, please clarify why the assumptions had to be made (i.e. no data).
- Executive summary Page 3 paragraph 4. Is it true that children are a sensitive population for PCBs?
- Executive Summary Page 5 Monte Carlo Cancer Risk Summary- Fish Ingestion Table. Please define what low estimate, base case, and high estimate are based on.
- Executive summary Page 6 paragraph 2. The long-term adverse health effects of PCBs in laboratory animals mentioned here are out of place and should be discussed in the toxicity assessment section.
- Executive summary: Please provide information regarding PCB concentrations in fish, such, congeners found, how exposure point concentrations were developed, and how congener data in fish were reconciled with toxicity criteria.
- Executive summary. Please provide information regarding fish species that are included in the evaluation.
- Executive summary. The Monte Carlo tables need further clarification.
- Section 1.4 Page 4. Please define what the Mid and Lower Hudson River areas are.
- Section 2 Page 5. Please reword the last sentence in paragraph 4 regarding PCB intake estimates.
- Section 2.0 Page 5 Paragraph 4. Provide additional clarification in the description of the RME exposures for the point estimate "combining high end values with average values to...come up with a point value estimate of the RME exposure." The statement is confusing in that "high end" exposure factors are not distinguished from "average" factors.
- Section 2.0 Page 5 Paragraph 4. A question the less-educated reader might have is why use both Monte Carlo methods and point estimate methods to estimate risks. This type of discussion would be helpful in this section. Use of a Monte Carlo method is not simply for the estimation of the RME exposure estimate. The author may not intend this interpretation but the phrasing of the exposure assessment leads one to this interpretation. A discussion of the uncertainty using the point estimate scenario, the clarification achieved in using the Monte Carlo simulation, and then comparison of these results to point estimate for risk management may be helpful to a less technical reader.
- Section 2.1 Page 6. Table 2-1 does not state which pathways are complete.
- Section 2.1. Please include more extensive discussion with references for not including certain pathways. Table 2-1 is too brief.
- Section 2.0 Page 6 Paragraph 2. Consider adding the word "potential" or "hypothetical" in front of words like exposure and risks.

- Section 2.1.1 Page 7 Paragraph 1. Recommend against using the term “site” to characterize the “study area”
- Section 2.1.2 Page 7. Recommend not classifying anglers exclusively as those who eat at least 1 self-caught fish meal per year. There are anglers who simply catch and release regardless of health-based fishing advisories.
- Section 2.1.1 Page 7. Is there literature support for the air pathway being significant?
- Section 2.1.2 Page 7. What is meant by “angling effort?”
- Section 2.1.3 Substitute the word pathway for route. The exposure route denotes either ingestion, inhalation, or dermal. The exposure pathway reflects the means or mechanism of contact.
- Section 2.1.3 Page 8. Please clarify if the MCL used to screen PCB health risks from an exposure pathway is health based.
- Section 2.1.3 Page 8. Please clarify if the detection limit used to detect PCBs is sufficient to detect levels that could cause potential health risks. A screening calculation should be done on the PCB milk detection limit. Cows drink a lot of water. This is not considered in evaluating potential sources of PCBs to a cow’s diet.
- Section 2.3. The exposure point concentration development is part of quantification of exposure (Section 2.2). This section should be changed to 2.2.1 and all subsequent sections should be changed accordingly.
- Section 2.3.1 Page 11. Presumably the age of the fish also will have an effect on the PCB concentration.
- Section 2.3.1 Page 11. Please provide a table with fish species used and the rationale for their representativeness.
- Section 2.3.1 Page 12. Concentrations in small fish species are provided for whole body only. This may overestimate the intake of PCBs for these fish, as higher lipid tissues will contain greater PCB concentrations than the fillets.
- Section 2.3.1 Page 12 Paragraph 5. Please clarify which modeled fish species were included in the HHRA.
- Section 2.3.1 Page 13. Please what the results of the location-to-location sensitivity analysis would provide or be used.
- Section 2.3.1 Page 13. Paragraph 4. Change “calculate the concentration of PCBs ingested in fish” to “calculate the dose of PCBs ingested from fish.”
- Section 2.3.1 Page 14. Paragraph 2. Add the last sentence of the paragraph, “The six species from the ...”, as a footnote to Table 3-4.
- Section 2.3.1 Page 14. Paragraph 3. Please clarify “frequency percentage.”
- Section 2.3.1 page 14. “PCB concentration weighted by species”. It is made clear in this section that several species identified in the Connelly study used to estimate intake rates are not commonly present or caught in the Upper Hudson study area. These species (trout, salmon, bullhead, and “other”) were removed from the analysis inasmuch as they contribute to the estimate of average exposure concentration of PCBs in fish tissue. However, it is unclear in this section or in Section 3, pertaining to the fish ingestion rates, whether any attempt was made to remove the influences of these same species on the ingestion rate, since they apparently contributed upwards of 62% of the species reported in the Connelly study as being consumed. If not, then the estimate of fish ingestion rates of these Hudson

River specific species may have been greatly overestimated. Please provide additional clarification on this issue.

- Section 2.3.2 Refer to figure for location reference.
- Section 2.3.2 Page 15. The text here states that two scenarios were assessed in estimating sediment concentrations: 1) assuming a continuing upstream source, and 2) assuming no continuing upstream source. The text then states that the concentrations corresponding to the continuing upstream source were used to calculate exposure point concentrations. No justification for this approach over the other is provided.
- Section 2.3.2 Page 15. Please define cohesive and non-cohesive sediment classes.
- Section 2.3.2 Page 15. It is unclear how segment sediment concentrations relate to fish concentrations.
- Section 2.3.2 Page 16. Modeled Sediment Concentration. There is no discussion why the sediment concentrations were modeled to 20 years in the future rather than 40 years as was done for fish.
- Section 2.3.3 Page 16. Modeled river water concentrations. There is no discussion why the river water concentrations were modeled to 20 years in the future rather than 40 years as was done for fish.
- Section 2.3.3. Are the PCB water concentrations predicted from the PCB sediment concentrations?
- Section 2.3.3. How do the river water segments relate to the river sediment segments?
- Section 2.3.4 Page 17. PCB air concentrations were only detected in May, June, and September during the 1991 sampling efforts. These detections and their associated water samples were used to estimate a water to air transfer coefficient. It is unclear whether there is any known explanation for these detections during only one particular time/season and not during any other times of the year. Is it possibly due to the location of the samples, perhaps water flow rates affect PCB concentrations in the water column? Are there conditions that were present during these times and not present at the others? If these are seasonal, should the modeled concentrations be presented and exposure assessment conducted only during these periods of time as well? Some discussion of these items would be helpful in interpreting the relevance of the coefficient development and the subsequent modeling effort.
- Section 2.3.4 Page 18. Do the empirical air water transfer coefficients relate to the air directly above the water or at air monitoring stations?
- Section 2.3.4 Page 20. Please explain why the Thompson Pool location was selected.
- Section 2.3.4 Page 21 Paragraph 3. The high-end empirical transfer coefficient is given as a concentration. Please add clarification.
- Section 2.4.2 Page 26. Sediment ingestion exposure duration. The sediment ingestion exposure durations and fish ingestion exposure durations are different at both the 50th and 90th percentiles. Although the difference between the values for the same percentiles is only one year and likely has little effect on the overall risk numbers, for consistency, the exposure durations for recreational exposures to sediments and exposure to fish should be the same.
- Section 2.4.2. PCB bioavailability should be addressed for sediment ingestion.
- Section 2.4.3 Page 28. Sediment adherence factor. Given that children will be playing in water and some wash-off will occur, use of the wet soil adherence factor seems as though it will overestimate the adherence of sediment to the skin.

- Section 2.4.3 Page 28. Skin surface area. The skin surface area was calculated using the data for specific age categories (child, age 6-7 data; adolescent, age 12 years data; and the mean for adults). The exposure duration for each age group was 3 years for children, 3 years for adolescents, 23 years for adults. Using whatever definition the assessors have for each age group then the average skin surface area for each age group should represent the average of actual age-specific data within that group and be representative of the entire exposure duration for the receptor group. For example, if children were defined as ages 0 to 7 years old with an exposure duration of 3 years, then the surface area could be calculated as the average of the 3 ages (4-5, 5-6, and 6-7). This approach seems more appropriate than selecting a higher end surface area and applying it for multiple years of exposure.
- Section 2.4.4 Page 29. Swimming time. Additional data in the Exposure Factors Handbook on swimming time is available. A 90th percentile value of 1.9 hours/day may be more appropriate.
- Section 2.4.4 Page 30. Skin exposure to river water. The same comments for exposure duration of sediment ingestion apply to dermal water contact and other recreational events.
- Section 2.4.4. Skin Surface Area Exposed. Please clarify basis for using 100% of full body surface area. Is the assumed exposure activity swimming?
- Section 2.0. A good summary table of chemical data would be useful.
- Section 3.0 Page 33. Paragraph 2. Exchange the word "impossible" for "difficult" in the last sentence.
- Section 3.2. Why couldn't a PDF be developed for exposure frequency?
- Section 3.2.1. Is it proper to group infrequent and frequent anglers?
- Section 3.2.2 Page 48 Paragraph 4. Where are the distributions for the fish consumptions?
- Section 3.2.2 Page 48 Paragraph 5. The reference to Table 3-4 should be changed to Table 3-5.
- Section 3.2.3 Page 49. I do not agree with the assertion that it is not possible to develop probability distributions representing the variability among consumers and cooking methods.
- Section 3.2.4 Page 50. Provide a PDF for exposure duration.
- Section 3.2.4.1 Page 53. Where is the data for the "all angler category?"
- Section 4.1 Page 63. Please provide summary of PCB homologue data in the report.
- Section 5.1.1 Page 68 Paragraph 1. Use "RME" in stead of "high-end" for consistency.
- Section 5.1.1 Page 68 Paragraph 2. Please clarify what is meant by "uniform exposure throughout the Upper Hudson River."
- Section 5.1.1. Stress that the HI's are theoretical.
- Section 5.1.2. Stress that the cancer risks are theoretical and upper bound.
- Section 5.1.2 Page 68 Paragraph 5. Strike "refers to plausible upper bound risks."
- Section 5.1.2 Page 68 Paragraph 6. Clarify the use of "applicable."
- Section 5.1.2 Page 69 Paragraph 6. The discussion of fish ingestion results is confusing?

Recommendations

Based on my review of the information provided, my overall recommendation is:

Acceptable with minor revision (as indicated)

The HHRA needs to be revised by enhancing the Risk Characterization section as well as incorporating available information. The Risk Characterization section is the most inadequate section of the HHRA. This section should be modified to include a comprehensive analysis of uncertainties that arise from the assumptions and procedures implemented in the HHRA. The section should also include an uncertainty analysis summary table to better assist decision makers.

In summary, several assessment parameters were developed without adequately using available scientific data. These issues that need to be re-evaluated are listed below.

- The Toxicity Assessment needs to provide a clear presentation of the new toxicity studies published since the development of the RfDs and CSFs in IRIS in the context of uncertainty associated with the use of the IRIS values.
- The fish ingestion rate should be modified to incorporate additional evidence on fish ingestion behavior. The factors should account for a fraction from source of fish other than 100%, the large number of anglers who do not consume fish due conservation policies, the potential for angler fish consumption to be dynamic from year to year, and anglers eating less than one fish meal per year.
- The RME cooking loss should be based on the available data instead of the worst-case use of 0%.

Harlee Strauss

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Harlee S. Strauss, Ph.D. has a Ph.D. in molecular biology from the University of Wisconsin - Madison and an A.B. in chemistry from Smith College. She was a postdoctoral fellow in biology at MIT and a Congressional Science Fellow sponsored by the Biophysical Society. Dr. Strauss has more than 20 years of experience in the areas of risk assessment and toxicology.

Dr. Strauss is currently the President of H. Strauss Associates, Inc., (HSAI) a consulting firm she founded in 1988. She works on a broad range of projects, from site specific risk assessments, to in-depth evaluations of the toxicity of individual chemicals, to the development of frameworks for risk assessment (e.g., for microorganisms). HSAI clients include private and public sector organizations and citizens groups. Dr. Strauss has served as a member of the U.S. Army Science Board since 1994, and has participated in studies regarding lead-based paint, groundwater and soil remediation at Army facilities, Chem/Bio Weapons Defense, and the Range Rule (pertaining to unexploded ordinance). Dr. Strauss served on the advisory committee for the Society for Risk Analysis Workshop "Key Issues in Carcinogen Risk Assessment Guidelines" and on various peer review committees such as for the EPA Exposure Factors Handbook and the Drake Incinerator risk assessment. She is a community member of the Restoration Advisory Board of the U.S. Army's Soldiers Systems Center (Natick Labs) and an elected Town Meeting member in Natick Massachusetts.

Pre-Meeting Comments
Upper Hudson River Human Health Risk Assessment Peer Review
Harlee Strauss, Ph.D.
May 5, 2000

Introduction

The charge to reviewers asks for a response to several specific questions. Unfortunately, none of these questions are directed to my two major concerns about the risk assessment, which I would like to state up front:

- The lack of consideration of pregnant/lactating women (alternately fetuses, breast feeding infants and young children) as explicit receptors. The omission is particularly problematic in view of: 1) the scientific literature that points to infants/young children as sensitive receptors, 2) the scientific literature that demonstrates that PCBs cross the placenta and that milk is a major route of excretion for PCBs from women's bodies, 3) the scientific data showing that a large fraction of a lifetime PCB dose is obtained in early years, and 4) EPA's initiatives in the area of protecting children.
- Whether the modeled concentrations of PCBs in fish reflect the fattier parts of the fish that may be consumed by some people. For example, it remains unclear to me whether the fillet concentrations that are the output of the FISHRAND model are skin on or skin-less (and whether the model validation took this difference into account). This should be specifically discussed in the HHRA. In addition, there is no accounting for the potential underestimation of exposure for people who may consume the entire fish, either whole or in soups or pastes, even in the uncertainty discussion. Only fillet data are discussed and provided for the three fish species included in the risk assessment.

These concerns could make order of magnitude differences in the risk characterization in the direction of higher risk. Most of the specific points we are being asked to address (outside the dose response question) would make far smaller differences in the risk calculations.

Responses to Specific Questions

Hazard Identification/Dose Response

Consistent with its risk assessment guidance (USEPA, 1993), USEPA considered scientific literature on PCB toxicity, both as to cancer and non-cancer health effects, published since the 1993 and 1994 development of the non-cancer reference doses (RfDs) for Aroclor 1016 and Aroclor 1254, respectively, and since the 1996 reassessment of the cancer slope factors (CSFs). Based on the weight of evidence of

PCB toxicity and due to the Agency's ongoing reassessment of the RfDs, USEPA used the most current RfDs and CSFs provided in the Integrated Risk Information System (IRIS), which is the Agency's database of consensus toxicity values. The new toxicity studies published since the development of the RfDs and CSFs in IRIS were addressed in the context of uncertainty associated with the use of the IRIS values (see, HHRA, pp. 76-77 and Appendix C). Please comment on the reasonableness of this approach for the Upper Hudson River.

I think it is appropriate to use the dose response values published in IRIS as the main basis for the toxicity assessment when relevant values are available. For the cancer risk assessment, EPA appropriately used the results from various Aroclors as a substitute for the mixtures actually encountered in various environmental media. While no one could believe this is a perfect substitute for data on the environmental mixture of concern, it is one that has been thought about in the context of multiple situations, and benefits from consistency of approach in decision-making.

The non-cancer dose-response factors are more troubling. Again, the use of RfDs published in IRIS is appropriate for adults. However, the incorporation of the recent data on neurodevelopmental and immunological effects on children is inadequate. The one short paragraph in the toxicity profile in Appendix C does not give the reader the sense of the extent of the database (three cohort studies, not one, with consistent results) and emphasize that the results are in human children exposed to environmental concentrations of PCBs. Furthermore, the uncertainty, and in this case the potential underestimation of the toxicity, should be considered more fully in the main body of the risk assessment.

To evaluate the effect of PCBs on young children in a more quantitative manner, a margin of exposure approach (rather than a toxicity factor) could be used. In this approach, the doses to which the children in the Upper Hudson River would be exposed could be compared with the exposures received by affected children in the various cohort studies. This dose would have to include those received prenatally (i.e., via transplacental exposure) and via breast milk in addition to direct consumption. The dose should be calculated by averaging over a short exposure duration (days to weeks), as the dose during a critical development window, not a long term average, is relevant. Moreover, a high end concentration of PCB in fish should be used in the calculation, not the means that were incorporated into the point estimate calculations.

Part of the problem with the inadequacy of the dose-response assessment with respect to children is the omission of pregnant and lactating women as receptors in the risk assessment. Pregnant and lactating women may be fish consuming anglers, if the receptor population is required to be thought of in those terms. They may also be the recipients of "gift fish", and the exception to the general case where the exposure assessment for the higher consuming angler is protective of the lower consuming family members.

Exposure Assessment

- 1) *Since 1976, the New York State Department of Health has issued fish consumption advisories that recommend [eat none] for fish caught in the Upper Hudson River. To generate a fish ingestion rate for anglers consuming fish from the Upper Hudson River under baseline conditions (i.e., in the absence of the fish consumption advisories), USEPA used data on flowing water bodies in New York State (1991 New York Angler survey, Connelly et al., 1992) to derive a fish ingestion rate distribution. The 50th and 90th percentiles were used for the fish ingestion rates for the central tendency (average) and reasonably maximally exposed (RME) individuals (i.e., 4.0 and 31.9 grams per day, equivalent to approximately 6 and 51 half-pound meals per year, respectively) (see, HHRA, pp. 24 and 37). Please comment on whether this approach provides reasonable estimates of fish consumption for the central tendency and RME individuals for use in the point estimate calculations.*

I have a two concerns with the EPA's selection of a 90th rather than a higher percentile for the RME calculation: 1) high consuming populations are not broken out separately, so they should be very carefully considered in the ingestion distribution, and 2) the distribution is based on a survey of licensed anglers, who may or may not have the same consumption distribution as unlicensed anglers. However, the impact of the selection of 90th or 95th percentile on the calculated risk is less than two fold, and I don't view this as a large problem.

- 2) *Superfund risk assessments often assume a 30-year exposure duration, based on national data for residence duration. However, because an angler could move from one residence to another and still continue to fish the 40 mile-long Upper Hudson River, USEPA developed a site-specific exposure duration distribution based on the minimum of residence duration and fishing duration. The residence duration was based on population mobility data from the U.S. Bureau of Census (1990) for the five counties that border the Upper Hudson. The fishing duration was developed from the 1991 New York Angler survey (Connelly et al., 1992). The 50th and 95th percentiles of the distribution were used for the central tendency (average) and RME exposure durations (i.e., 12 and 40 years, respectively). Please comment on the adequacy of this approach in deriving site-specific exposure durations for the fish ingestion pathway (see, HHRA, pp. 23 and 49-57).*

This is a reasonable approach and the discussion in the report surrounding it pointed out some of the untested assumptions (e.g., whether or not the mobility of the angler and non-angler population was the same). The EPA approach does not account for people RETURNING to the Hudson River counties (as a residence) and resuming fishing activities, or visiting family during a vacation and going fishing with family or old friends. My personal bias would have been to use a longer fishing duration for the RME to account for some of these uncertainties. However, the incorporation of even a 60 year fishing duration would make little difference to the calculated cancer risk and no

difference to the noncancer risk.

- 3) *PCB concentrations in Upper Hudson River fish generally have declined in past decades and the decline is expected to continue into the future. Therefore, to evaluate non-cancer effects for the RME individual, USEPA used exposure point concentration in each medium (water, sediment, and fish) based on the average of the concentrations forecast over the next 7 years (1999 to 2006), which gives the highest chronic dose considered in the HHRA. For the central tendency exposure point concentrations, USEPA used the average of the concentrations forecast over 12 years (1999 to 2011), which is the 50th percentile of the residence duration developed from the population mobility data (U.S. Bureau of Census, 1990). In addition, for completeness, USEPA averaged the exposure concentration over 40 years (1999 to 2039) to evaluate non-cancer hazards for the same time period over which cancer risk was calculated. Please comment on whether this approach adequately addresses non-cancer health hazards to the central tendency and RME individuals (see, HHRA, pp. 67-68).*

I think that the approach used for the RME is appropriate and should also be used for the CTE. In the model used here, the dose estimate decreases with increasing averaging time. The CTE, like the RME, will be exposed to the fish for 7 years, and then additional years after that. But if a chronic exposure is defined as an exposure for 7 or more years, then both the CTE and RME will undergo chronic exposure to the concentration averaged over 7 years, and should be evaluated as such. In this exposure scenario, factors other than averaging time (exposure duration) will distinguish the RME and CTE.

Monte Carlo Analysis/Uncertainty Analysis

- 4) *USEPA policy states that probabilistic analysis techniques such as Monte Carlo analysis, given adequate supporting data and credible assumptions, can be viable statistical tools for analyzing variability and uncertainty in risk assessments (USEPA, 1997a). Consistent with this policy, USEPA used a tiered approach to progress from a deterministic (i.e., point estimate) analysis to an enhanced one-dimensional Monte Carlo analysis of the fish ingestion pathway (see, HHRA, Chapter 3, pp. 33-59). Please discuss whether this Monte Carlo analysis makes appropriate use of the available data, uses credible assumptions, and adequately addresses variability and uncertainty associated with the fish ingestion pathway (e.g., defining the angler population, PCB exposure concentrations, ingestion rates, exposure durations, cooking losses) qualitatively or quantitatively, as appropriate, in the analysis (see, HHRA, pp. 72-74).*

In general, I found the Monte Carlo analysis acceptable. I thought the comparison of the point estimate and Monte Carlo percentile distributions was illuminating and enhanced the credibility of both analyses. I do have a few comments and concerns, however.

Defining the angler population.

The Monte Carlo analysis, like the rest of the HHRA, defines the at risk population as anglers who consume at least one (self-caught) fish meal per year. Sensitive subpopulations are not considered separately. The HHRA, when justifying this approach, appears to consider only high consumers (including those who use fish as a significant food source) as a subpopulation of concern. This is not the case. As pointed out previously, I consider those who are exposed to PCBs *in utero* and via mother's milk as a population who must be considered separately in terms of both exposure and toxicity.

Another subpopulation of concern is the consumers of single species (especially bottom feeders) who use the entire fish. This subpopulation must be characterized by more than its consumption rate. While the high consuming/single species case is somewhat dealt with as part of the 72 sensitivity analyses (although the use of portions of the fish other than fillets is not considered at all in the HHRA), it is not discussed adequately in terms of how it represents a potential, highly exposed population.

The breadth of the distributions and the sensitivity analysis

For any given percentile, it appears that there is at most a 30 fold difference in cancer risk or hazard index among the various parameters examined in the sensitivity analysis. I am surprised by the narrowness of this range.

Fraction of PCBs lost during cooking, species preferences

I thought the fraction of PCBs lost during cooking of fillets was handled in a reasonable way in the point estimate calculations. In the Monte Carlo analysis, there was no year to year correlation with cooking method, PCB cooking loss, or species preferences. I think that these factors are likely to be correlated from year to year. I am concerned that the lack of correlation will average out risky preparation methods, just as no correlation from year to year in fish ingestion rates would have averaged out continually high consumers. My concern about PCB concentrations in cooked fish if fattier parts of the fish are not removed during cooking was not addressed at all.

- 5) *For the Monte Carlo analysis, USEPA evaluated a number of angler surveys, but excluded local angler surveys, such as the 1996 and 1991-1992 Hudson Angler surveys (NYSDOH, 1999; Barclay, 1993), due to the fish consumption advisories. The 1991 New York Angler survey (Connelly et al., 1992) was used as the base case and other surveys were used to address sensitivity/uncertainty in fish ingestion rates (see, HHRA, pp. 37-46). Please comment on the adequacy of USEPA's evaluation and use of existing angler surveys in the Monte Carlo analysis of the fish ingestion pathway.*

I think the HHRA provided clear justification for its selection of data to include in the analysis. However, the use of the 1991 NY Angler survey does have clear limitations. In particular, this survey of licensed anglers would have underestimated young anglers, who

do not need to have licenses. Yet young children are the most susceptible population to some of the noncancer adverse effects of PCBs.

Another problem with the analysis of the survey data is whether the type of fish consumed is adequately modeled based on the data provided in Connelly et al. 1992 and other surveys. For example, the data summarized in the HHRA indicate that one or a few people ingest large amounts of eel. Are there eel in the Hudson? If so, is the PCB concentration in the fillet of the brown bullhead an appropriate surrogate for the PCB concentrations in the eel?

Comments in the Responsiveness Summary (p.21) suggest that eel (and carp) are caught in the Upper Hudson, but go on to suggest that because the fraction of people who ingest eel and carp is low, so that the risks are averaged out. However, this is not necessarily the case, as people frequently have species preferences (such as the person who eats eels, as reported in Connelly et al 1992). The variability and uncertainty with respect to some species preferences is stated to be captured in the sensitivity analysis for the Monte Carlo runs, but it is not clear to me if this is so. The report also states that the fraction of each species ingested is drawn from a distribution developed from the Connelly et al, 1992 data and are not correlated from year to year, which would average out any species preference. Beyond this, the question of whether a brown bullhead fillet is an appropriate surrogate for eel (and carp) is not addressed in any quantitative way.

Risk Characterization

- 6) *The risk characterization section of the HHRA (Chapter 5, pp. 67-80) summarizes cancer risks and non-cancer hazards to individuals who may be exposed to PCBs in the Upper Hudson River. Please comment on whether the risk characterization adequately estimates the relative cancer risks and non-cancer hazards for each pathway and exposed population. Have major uncertainties been identified and adequately considered? Have the exposure assumptions been described sufficiently?*

In our site visit of the Upper Hudson River, we visited an island area (I think in Ft. Edward) with picnic tables, boat launching facilities, and a beach where children could be swimming all summer. It is clear there is lots of boating on the Upper Hudson, and that people may swim off the boats on hot days. There are also houses along the River which are likely venues for water based recreation including swimming and wading. With these observations in mind, it seems that the assumptions for frequency and duration of swimming (once a week for the RME) and wading activities are too low. That said, however, the exposure scenarios for recreational use of the Upper Hudson are comprehensive, and the correction of the swimming exposure frequency is not likely to significantly change the overall relative risk of the various exposure pathways. The discussion of the uncertainties and exposure assumptions is clear and sufficient.

General Questions

A goal for risk assessments is that they be clear, consistent, reasonable and transparent and adequately characterize cancer risks and non-cancer hazards to the exposed population, including children (USEPA, 1995b, 1995d). Based on your review, how adequate are the HHRA and Responsiveness Summary when measured against these criteria?

I found the risk assessment to be exceptionally well written. It clearly and concisely described the overall methodology and assumptions, with a few exceptions which are noted in these comments. While I sometimes found the tradeoff of conciseness with detail in the report body to lean too much toward conciseness, the appendices did include much of what was missing. However, the tables in the appendix, especially the Monte Carlo sensitivity analyses, should have been fully described so the reader did not have to guess at the report's shorthand schemes.

The HHRA did not address children, either as infants or as young (under 10) consumers. This omission is especially critical for PCBs, as environmental PCB mixtures have demonstrated effects on the immune and nervous system during development. If there is a fatal flaw in the risk assessment, this is it.

Please provide any other comments or concerns, both strengths and weaknesses, with the HHRA not covered by the charge questions, above.

The risk assessment should provide more extensive commentary on the strength of the assumption that the boundary conditions in the baseline (HUDTOX) model, i.e., the concentration of PCBs entering the River (modeled from 0 to 30 ng/L with 10 ng/L results used in the HHRA calculations), is correct and fully reflects the range of possible future conditions. While I recognize that EPA does not consider this in the scope of the HHRA, it may be the single most important assumption of the whole risk assessment, as it forms the basis of the predictions of the fish (and sediment) concentrations far into the future.

Recommendations

Based on your review of the information provided, please select your overall recommendation for the HHRA and explain why.

1. *Acceptable as is*
2. *Acceptable with minor revision (as indicated)*
3. *Acceptable with major revision (as outlined)*
4. *Not acceptable (under any circumstance).*

My recommendation is to accept the report with revisions (I'm not sure if they are minor or major). The report is acceptable as it now stands with respect to the majority of the population of adult anglers and other adult recreators. It is deficient, and likely

underestimates the risk for children especially those exposed *in utero* and via ingestion of mother's milk. Exposures and hence risk to children swimming and wading in the Upper Hudson River are also underestimated. The uncertainties regarding anglers who consume portions of the fish other than fillets needs to be investigated. However, even if the children were included and showed a high risk, and the eel eaters are at higher risk than shown in the assessment, the conclusion from the risk analysis would be the same as presented in the HHRA and revised HHRA in the Responsiveness Summary: the risks are higher than the benchmark range of acceptable risks.

Robert Willes

REVIEW COMMENTS ON USEPA BASELINE HHRA ON PCBs IN THE UPPER HUDSON RIVER

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May 5, 2000

1. INTRODUCTION TO COMMENTS:

The following text summarizes the review comments of Robert Willes on the USEPA's Baseline HHRA of PCBs in the Upper Hudson River. The issues and points raised in this review are briefly presented, and more detailed discussions, with appropriate reference materials, will be provided at the up-coming meeting of the review team on May 30/31.

This review has identified and focusses on two separate issues that will be considered when addressing the "Charge" of the HHRA reviewers:

- ii) The suitability/accuracy of the HHRA in the estimation of potential health risks/impacts from PCBs found in the Upper Hudson River; and
- iii) The usefulness of the HHRA in assisting the USEPA (and potentially other parties) in the selection and application of remedial strategies for PCBs in the Upper Hudson River.

In my view, these are separate questions. For example, in my opinion, the HHRA has serious limitations with respect to accurately delineating the potential health risks/impacts to humans (and other receptors for that matter). However, the HHRA may provide reasonable evidence, when combined with other information (e.g., potential escalation of impacts due to catastrophic-event scouring of sediments, political will, regulatory policies, inferences on potential global impacts) that the current situation related to PCBs found in the Upper Hudson River is not acceptable. Such a conclusion could lead to the decision to proceed with some type of remediation of PCBs in the Upper Hudson River.

Once the decision is made that some type of remediation is required, it is critical that methods are available to evaluate the impact reduction of various remediation options, plus, more importantly, to enable the evaluation of the potential impacts of the remediation options *per se*. For example, it is possible that the end result of a given remediation technique may satisfactorily reduce impacts compared to the "do nothing" option; however, impacts may be unacceptably increased during the remediation process. The HHRA methodology and approach must be suitable to assist in providing information to assist remediation managers in the required decision-making process.

Different attributes and issues need to be considered when judging these two quite different uses

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of the HHRA. The first case requires accuracy in the HHRA predictions of human health risks/impacts. The second case requires that the HHRA is sensitive to factors related to the remedial options under consideration that change environmental concentrations and consequent exposures of people and other receptors to PCBs. In addition to assisting in the evaluation and final selection of remedial options for the Upper Hudson River, the HHRA would be useful in identifying which parameters and locations require monitoring (and indications of the frequency of monitoring) to ensure that the selected remediation options do not result in unacceptable impacts/risks to the river system and the various receptors (human and otherwise) of concern.

2. Responses to “Charge” Questions

2.1 Hazard Identification/Dose Response

The consideration by the HHRA methodology of hazard identification and dose response issues related to PCBs is not considered adequate. The approach followed in the HHRA does not present a balanced evaluation of the available scientific information on PCBs and related compounds such as polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF). Conventional USEPA methodology is followed, with a discussion of one aspect of the uncertainties in such assessments. The discussion of the conservatism in the methodology followed should consider, at least briefly, the various issues discussed in the USEPA (1996) update in guidance of methods and the scientific basis for alternate approaches for estimating potential health consequences from exposure to carcinogens.

Specific points related to the toxicity assessment are given below:

- Pg 28 – Issuance of “tickets” for violation of fishing restrictions has nothing to do with HHRA, the characteristics of the dose/response relationships, or the validity of the risk estimates. For example, law enforcement agencies use a threshold approach for “ticketing” automobile drivers for excess alcohol consumption, whereas the health evaluation information demonstrates that this threshold does not apply to fetal alcohol syndrome (occurs at much lower doses). Using logic on pg 28, the fact that a “ticketing” threshold exists would indicate that there is no concern about fetal alcohol syndrome.
- PgES-4 – Critique of Kimbrough, 1999 that 75% of workers were not exposed — same problem with other studies, but the remaining 25% of Kimbrough cohort that were exposed represents a large number of subjects.
- Pg C3 – There is a lack of detail on discussions of epidemiological studies, except for Kimbrough, 1999 – lacks balance. There are many discussions of these studies by recognized scientific experts in the published literature.
- PgC2 & 3 – The conclusions of HHRA discussions of the conclusions of the epidemiological studies disagrees with the TERA assessment.
- PgC-3 – Overview of the carcinogenic potency in animals – does not mention the Brunner et al (1996) study used with the Norback and Weltman (1985) study to establish the CSFs for PCBs.
- Pg C-2 & 3 – no discussion of genotoxic potential, the uncertainty, lack of consideration of metabolism/repair systems, etc. and the relationship of these issues to the conservatism in the CSF estimates for PCBs. Should at least discuss these issues

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qualitatively, and qualitatively consider their impact on the risk characterization. This was done with potential endocrine issues with PCBs, why not when looking at the conservatism in the CSF estimates?

- Pg C-4 – no discussion of whether or not there is evidence that the “low risk and persistence” tier are tumor promoters, genotoxic – similar problem to above.
- PgC-4 – mild liver damage with high doses – doesn't agree with pathology descriptions of the studies. The studies demonstrate severe liver damage, that is believed to result in extensive hyperproliferative responses on liver parenchyma. These hyperproliferative responses are known to result in the expression of genetic lesions related to ageing in rodents, particularly in Sprague Dawley rats. There are a number of peer reviewed publications in the scientific literature by recognized experts in rodent pathology that outline this issue, and the difficulties it causes in the validity of the extrapolation of such effects to humans exposed to doses well below those associated with liver toxicity.
- PgC-4 – large paragraph on work by Patandin (1999) and Lant'ing (1999) – these are theses, and have not been published in peer reviewed, scientific journals. This is a dangerous practice. Non-peer reviewed data should not be considered other than as supporting information, and does not deserve the apparent weight given here. There is a large body of work that has been published on the issue of the effects of PCBs and Dioxins/Furans on the development of children (e.g., Jackson's group) – this is not quoted or discussed in the PCB toxicity profile, but one reference is given in the reference list. The critiques I am aware of regarding these studies (see Kimbrough and others) seriously discount the causal linkage of the effects observed to PCBs or Dioxins/Furans. This can be discussed in more detail during the May 30/31 reviewer's meetings.
- PgC-4 – endocrine disruption handled qualitatively, why not use this approach for evaluating the degree of conservatism inherent in the toxicity limits for PCBs??
- PgC-5 – no discussion of the problems in interpreting the Barsotti and Allen work – just to mention a few of these problems: lack of measurement of PCB concentrations in diets, coincident studies in the same animal facility on chlorinated dioxins/furans, inadequate documentation of good laboratory practices (GLP) for the studies. This can be discussed in more detail during the May 30/31 reviewer's meetings.
- Pg C-6 – no discussion of relative evidence of relative sensitivity of monkeys versus humans regarding eye, nail and skin lesions – should at least discuss these issues qualitatively. There is abundant evidence that these effects are not observed in workers exposed to very large quantities of the same mixtures of PCBs as used in the monkey studies.
- Pg65 – PCB congeners are “believed to be” responsible for only part of the carcinogenicity of a Total PCB mixture. This is an overstatement of the known science,

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and provides a level of confidence to the assessment that is not warranted based on the data available.

- Pg 65 – need a discussion of how international jurisdictions (e.g., WHO, Europe, Canada, Australia, Japan) evaluate PCBs and PCDD/PCDF – not considered genotoxic carcinogens, and use a non-linear dose-response evaluation similar to that outlined in the EPA (1996) Carcinogen Assessment Guidance document. There is an abundance of scientific evidence supporting these contentions (references will be provided at the May 30/31 meeting).

In addition, the hazard evaluation sections should address recent development in the application of the EPA (1996) Carcinogen Risk Assessment Guidance document. In particular, the application of the guidance to formaldehyde, and the extrapolation of the conclusions of the formaldehyde re-evaluation on PCBs and PCDD/PCDF. In addition, the recent court actions on the assessment of chloroform. Both the formaldehyde and chloroform issues demonstrate the applications of non-linear dose-response methods for the evaluation of non-genotoxic carcinogenic substances, and result in decreases in the estimates of carcinogenic potency (increases in the CSFs) by 10- to 100-fold. These issues need to be discussed in the Risk Characterization section to provide a perspective on the degree of conservatism in the HHRA.

- Pg 65 – Need to discuss the coincidence between the PCDD/PCDF CSF used by the USEPA ($150,000 \text{ mg/kd.day}^{-1}$), the observed background exposures of PCDD/PCDF in people in the US (between 2 and 3 pg/kg BW/day) and elsewhere, so that the use of CSFs for PCBs, Dx/Fr can be put into perspective. For example, using the above CSF and the background exposure estimates for Dx/Fr in the U.S., the liver cancer incidence (basis for the Dx/Fr CSF) would be between 0.3 (30%) and 0.45 (45%):

CFS for Dx/Fr = $0.15 \text{ pg/kg.day}^{-1}$

Background Dx/Fr exposure ≈ 2 to 3 pg/kg/day

$0.15 \times 2 = 0.3$; $0.15 \times 3 = 0.45$

These results mean that current exposures to PCDD/PCDF would be responsible for 100% of the lifetime risk of death of cancers from all causes; and the U.N. estimates that only about 10% of cancers are from environmental sources of chemicals, the remainder are from other causes such as genetics, diet, tobacco smoke, etc.

Further, the above 30% to 45% incidence rate is the prediction for liver cancer. In the U.S. the total incidence of liver cancer from all causes is between 1 and 5 per 10,000; and the major risk attribution for liver cancer is alcohol consumption and other liver diseases.

These issues need to be discussed in the risk characterization section and seriously impinge on the accuracy of the HHRA methodology in providing realistic estimates of health impacts to people living in the environs of the Hudson River.

- Pg 65 – wording indicating “sparse data currently available” re: breast cancer and PCBs is inappropriate. State the amount of data that is available (I will provide these References). More appropriate wording, and still very conservative, is that the “available evidence for a causal association is weak to non-existent”. This is equivalent to the conclusions of the HHRA report for endometriosis.
- Pg 65 – humans have mechanisms to maintain hormone homeostasis – so do other mammals and animals generally – would be a much stronger statement if included all animal species. In addition, the same homeostatic mechanisms exist for responding to potential carcinogenic insults, for example, those that all people experience from cosmic radiation.
- Pg 65 – the HHRA identifies endocrine disruption agents in foods as a rationale for not being concerned about other endogenous endocrine disruptors – This same argument applies to background PCB and PCDD/PCDF exposures.
- The implications of the above issues on the accuracy of the HHRA in predicting health impacts/risk to people living in the environs of the Upper Hudson River will be discussed later.

2.3 Exposure Assessment

The consideration by the HHRA methodology of exposure issues related to PCBs is considered adequate. Specific issues are noted below related to exposure duration and implications on specific types of receptors. The use of Monte Carlo simulations could have been expanded to assist in the identification of critical factors affecting impacts; however, these issues are more important in the use of the HHRA in assessing the feasibility of various remedial options and their application (discussed later).

- The approach used in the HHRA for estimating fish consumption appears reasonable. An additional recommended approach would be to calculate (using the HHRA model) the fish consumption that would result in acceptable impacts/risks, then evaluate the reasonableness of this hypothetical value. The two approaches should be complementary.
- The approach used to develop the 12 (central tendency) and 40 (RME) year values for exposure duration are reasonable.
- The procedures used for estimating the central tendency and RME average tendencies of 7 years for estimating fish concentrations is reasonable for the general population; however, it is not suitable for concerns about exposures of pregnant women, or women nursing infants. The exposure durations of concern for such receptors are much shorter (e.g., a few months related to the gestation and nursing time periods). A suggestion for the assessment of potential impacts to such people would be to conduct a “rolling average” exposure assessment using 3 to 6 month averaging intervals beginning at 1999

and proceeding forward, perhaps to a maximum period of 40 years. This will enable an evaluation of potential impacts to pregnant and lactating women in the Upper Hudson area.

- 90th percentile issue – see pg 29 of responsiveness summary – typographical error on pg 15-16 of HHRASOW.
- Pg5 – components of analysis included variability of concentrations, likelihood of exposure via various pathways, frequency and duration of exposure.
- Pg7 – assume anglers consume fish from Hudson, even though there are fishing bans and Hudson-specific health advisories. This point needs to be stressed in the Risk Characterization section to clearly state that the HHRA considers hypothetical situations that would exist if fishing bans were not in place.
- Pg-8 – refer to New York State data, and Dr. Buckley's data on beef, dairy, and crops, but do not provide the reference.
- Pg12 – were spottail shiner, pumpkinseed and white perch included in the FISHRAND model??
- Pg 13 – Cannot quantify fishing preferences or frequency at specific locations on the Hudson – sensitivity analysis in Chapter 5 to evaluate this issue.
- Pg 13 – Brown Bullhead and White Perch had the highest concentrations, spottail shiner and pumpkinseed were lowest.
- Pg33 – Monte Carlo – was sensitivity analysis conducted on distributions – i.e., what was the impact of assuming different distributions for parameters on the final impact estimates?
- Pg 34 – largest advantage of Monte Carlo simulations are that they avoid the problems of unknowingly combining worst-case or upper bound parameter values, and consequently obviating the understanding of the probability associated with the final impact estimate. This point needs to be emphasized in the risk characterization section.
- Pg 37 – duration of average – may not be appropriate to arbitrarily use 365 days – need to address how the duration is related to clearance $t_{1/2}$ for the substance. For example, duration of exposure should be approximately 5 times $t_{1/2}$ to achieve equilibrium (maybe want to go 7 times $t_{1/2}$ to improve certainty). If $t_{1/2}$ for some PCB congeners is as much as 9 years (some data to support this); then 5 times the $t_{1/2}$ means 45 years of exposure would be required to reach equilibrium state. This is very important because of differences in $t_{1/2}$ between test animals and humans. If it is assumed that the response of all mammalian systems is the same for a given tissue concentration of PCBs (this may or

may not be true), and the primary factors responsible for differences in response are related to the $t_{1/2}$ (metabolism, excretion, storage, etc.); then assuring that equilibrium body burdens are attained would be critical to the impact assessment. Therefore, assuming 365 day exposures for non-cancer end points may not be adequate to achieve equilibrium. For other congeners that have a shorter $t_{1/2}$ (e.g., 1 year), 5 years exposure durations would be sufficient to reach equilibrium body burden concentrations. It is unclear how this issue was considered in the assessment?

- Pg 49 – low toxicity of PCBs by inhalation versus fish consumption – disagree with this statement – likely that differences in apparent toxicity are totally related to exposure rates – in fact, exposure by inhalation, if the doses were great enough, would likely be more hazardous because of lack of “first bypass” through the liver. These statements need to be modified.
- Pg 58 – discuss sensitivity/uncertainty analysis as an alternate means of addressing 2-D issues to assess precision of the analysis – involved repeating the Monte Carlo simulation for separate input distributions for 72 combinations of Fish Ingestion, Exposure duration, Fishing Location and Cooking Loss to assess PCB intake. Performed 10,000 iterations for each of the 72 scenarios evaluated.

2.4 Risk Characterization

The Risk Characterization section does not discuss the issue of the degree of conservatism in the assessment; rather it focuses solely on uncertainties, and leaves the reader with the impression that the conclusions of the assessment have a high degree of uncertainty. A clear discussion needs to be included on how uncertainties are addressed in the USEPA RfD and CSF values, and how these procedures result in a high degree of conservatism in the overall results of the assessment. The inclusion of the discussions outlined in review section 2.2 will provide the balance needed for the reader to draw conclusions about the accuracy of the HHRA in predicting health impacts/risks to people in the environs of the Upper Hudson River.

The exposure assumptions are adequately discussed. The issue of exposure duration for pregnant women and nursing mothers needs to be discussed as appropriate depending what the addition of this analysis shows.

- Pg 69 – CT Cancer risk $\approx 3.2 \times 10^{-5}$; RME $\approx 1.1 \times 10^{-3}$ – these are outside the 10^{-4} to 10^{-6} range considered acceptable. When use PCDD/PCDF TEQ approach, get about the same value. Need a discussion regarding background exposures to PCBs and PCDD/PCDFs in other regions of the U.S. in order to interpret these risk estimates.
- Pg 76 – Need to discuss concept of uncertainty and conservatism. CSFs represent upper bound risks – unlikely that risks would be under estimated. Risks could range from zero

to the upper bound value. CSFs do not consider non-genotoxic nature of PCBs, or Dx/F. Need to discuss what risks mean with respect to background cancer incidence; e.g., using Dx/Fr cancer slope factor, and PCB cancer slope factor, compared to background PCB and Dx/Fr exposures and projected cancer risks.

- Pg 76 – TEFs – order of magnitude estimates – correct statement, but need to discuss conservatism together with uncertainty – antagonisms between congeners & ability more potent congeners to stimulate metabolisms of other congeners may result in overestimations of toxicity. Problem with non-dioxin-like congeners; not included in TEFs, but may alter metabolisms, etc.
- Pg76 – Endocrine disruption – the end results of possible endocrine disruption effects are considered in lifetime, multi-generation exposure studies – these studies consider the integrated effects on reproduction and development through two or more generations. If truly adverse endocrine effects occur, their impacts would be observed through studies of intact animal systems.
- Pg 77 – Monte Carlo analysis captures much of the uncertainty, and serves to demonstrate that the CT and RME point estimates are reasonable values. However, the Monte Carlo analysis does not consider the degree of conservatism inherent in the toxicity components of the assessment. If these are combined with the exposure uncertainty, there will be a greater tendency to “shift” the cancer risks and HIs to lower values than to higher values.
- Pg 78 – Fishing location issue – approach reasonable, but likewise need interpretive statements relative to the toxicity uncertainties.
- Pg79 – Characterization needs to discuss exposure duration & $t_{1/2}$ issue in addition to residency time issues – if $t_{1/2}$ is long (e.g., 7 to 9 years), need 35 to 45 years of exposure to achieve equilibrium. Changing concentrations of PCBs in fish over time, means that equilibrium will never be reached. Particularly important with the more persistent PCB congeners with their longer $t_{1/2}$'s. A sensitivity analysis should be conducted estimating body burdens of “anglers” for groups of congeners with different $t_{1/2}$ values to determine the most critical exposure duration relative to body burdens.

3. Comments on the Suitability and Accuracy of the HHRA in the Estimation of Potential Health Risks/Impacts from PCBs in the Upper Hudson River

Based on the issues outlined in Review Sections 2.2 and 2.4, it is my opinion that the results of the HHRA have a low degree of accuracy in predicting the absolute level of potential health risks/impacts from exposure to PCBs in the environs of the Upper Hudson River. This opinion has nothing to do with the fact that fishing advisories undoubtedly mean that actual risks to the community from PCBs in the Upper Hudson River are negligible. It is also my opinion that the

inability of the HHRA to provide realistic predictions of possible health risks/impacts should be clearly and unequivocally communicated to the reader of the report by appropriate inclusion in the Risk Characterization section, and as part of the conclusions to the HHRA.

The above opinion is not specific to the Upper Hudson HHRA, but is an outcome from the HHRA methods recommended in USEPA guidance documents. This is not the fault of the guidance documents, rather it is a reflection of the use intended for the HHRA.

Clearly, the HHRA guidance procedures followed would provide a high degree of confidence in rates of exposure to PCBs that would not result in measurable adverse impacts/risks to people in the environs of the Upper Hudson River. The conservatism inherent in the USEPA HHRA methodology readily supports the conclusion that, providing the estimated exposures do not exceed the guidance hazard parameters recommended, no unacceptable or measurable adverse health impacts/risk would occur. However, predicting exposures that would not result in unacceptable impacts/risks this is a very different task than predicting the levels of impact/risk that could actually occur from exposures to PCBs that actually exist in the Upper Hudson River. It is here that the HHRA methodology falls short.

In order to assess the accuracy of the HHRA methods, either much greater accuracy is required in the hazard assessment and exposure assessment paradigms, or reference comparisons are necessary that involve comparisons of predicted risks/impacts with real data on human disease, or lack thereof, observed following real-world exposures. This information is available, but the comparisons have not been conducted in the HHRA of the Upper Hudson River. For example, comparisons of impacts/risks that the HHRA would predict based on equivalent exposures should be made with those exposures actually measured or estimated in the epidemiological studies. In addition, comparisons of risks from background exposures, combined with the causal factors causing the diseases predicted assist in interpretation of the impacts/risks predicted by the HHRA methods. Section 2.2 provides such a comparison of the risks predicted using the hazard assessment information for PCDD/PCDF against those actually observed in a population.

Clearly, it can be concluded with a great degree of confidence that the impacts/risks to people in the environs of the Upper Hudson River would not be greater than those predicted by the HHRA. However, the actual impacts/risks may actually be as little as zero, particularly cancer risks.

It is my opinion that the most realistic conclusion from the HHRA conducted on PCBs from the Upper Hudson River is that the situation is not acceptable relative to the environmental occurrence of PCBs in other regions of the U.S. This conclusion, plus other information on estimates of ecological impacts/risks and policy issues, can be used in the decision-making process of the Remediation Investigation/Feasibility Study.

4. Applications the HHRA in the Selection and Application of Remedial Strategies for PCBs in the Upper Hudson River

The HHRA methodology can provide a powerful tool in the decision-making processes involved

in the remedial investigation/feasibility study for the Upper Hudson River. The conservatism and uncertainty associated with predictions of actual levels of impact/risk are not as important in such uses because the HHRA methods are used in a comparative manner (e.g., one remedial option would be compared to another option, including the "do nothing" option). This means that the various sources of conservatism/uncertainty cancel when using the HHRA as a comparative tool.

In order to ensure that the HHRA, ERA and environmental fate methods are optimal for the risk/impact comparisons of various remedial options, it is necessary to ensure that the methods are appropriately sensitive to the specific and unique features of the various remedial options under consideration. Sensitivity analysis techniques, similar to those already conducted as part of the baseline HHRA, are powerful tools in assessing whether or not the various HHRA components are sufficiently sensitive to assess remedial options. It is considered beyond the scope of this review to go into the details required to enable an evaluation of the suitability of the existing HHRA as a tool in comparing remedial options. However, based on my review, the current HHRA approach, especially with the Monte Carlo simulation approaches used, is largely ready for such comparative uses. Care should be taken, however, to ensure that the characteristics of specific remedial options are adequately captured by the HHRA in its current form.

5. Recommendations

Overall, I recommend that the HHRA is acceptable with major revision.

These revisions should primarily involve the use of various comparisons, as discussed in my specific review comments, to provide an evaluation of the overall accuracy of the HHRA. The discussion of these comparisons in the risk characterization section would provide the reader with a more realistic impression of the degree of conservatism inherent in the HHRA methodology, and the usefulness of the HHRA in evaluating the acceptability, or lack of acceptability of the current situation on the river.

Additional discussions, and possibly sensitivity analyses, should be presented on the use of the HHRA as a comparative tool in the remediation option/feasibility study. For example, sensitivity analyses, using Monte Carlo simulation methods, should be conducted to determine the appropriateness of the HHRA methods in assessing sediment mobilization that could result from specific remediation options. Such sensitivity analyses must also involve the ERA and environmental fate modelling used on the project.

Ecological Risk Assessment Review

Ecological Risk Assessment Charge

The goal of the Ecological Risk Assessment is to evaluate the risks to ecological receptors associated with exposure to PCBs in the Hudson River in the absence of remedial action of the PCB-contaminated sediments (i.e., under baseline conditions). The following documents will be provided to the peer reviewers:

Primary

- Baseline Ecological Risk Assessment, August 1999
- Responsiveness Summary for the Baseline Ecological Risk Assessment, March 2000

References

- Ecological Risk Assessment Scope of Work, September 1998
- Responsiveness Summary for Ecological Risk Assessment Scope of Work, April 1999
- Executive Summary for the Baseline Ecological Risk Assessment for Future Risks in the Lower Hudson River, December 1999
- Executive Summary for the Human Health Risk Assessment, Upper Hudson River, August 1999
- Executive Summary for the Human Health Risk Assessment, Mid-Hudson River, December 1999
- Executive Summary for the Revised Baseline Modeling Report, January 2000
- Suggested charge questions from the public for the ERA, February 2000

The reference documents listed above are being provided to the reviewers as background information, and may be read at the discretion of the reviewers as time allows. The reviewers are not being asked to conduct a review of any of the background information.

Additional Reassessment RI/FS documents are available on USEPA's website (www.epa.gov/hudson) and/or by request. Additional documents include the following:

- Hudson River Reassessment RI/FS Database, August 1998
- Executive Summaries for other USEPA Reassessment RI/FS Reports
- Peer Review Reports from first two peer reviews
- Responsiveness Summary for first peer review

Specific Questions

Problem Formulation/Conceptual Model

1. Consistent with USEPA guidance on conducting ecological risk assessments (USEPA, 1997), the problem formulation step establishes the goals, breadth, and focus of the assessment. As part of the problem formulation step in the ERA, a site conceptual model was developed (Chapter 2.3, pp. 11-19). Please comment on whether the conceptual model adequately describes the different exposure pathways by which ecological receptors could be exposed to PCBs in the Hudson River. Was sufficient information provided on the Hudson River ecosystems so that appropriate receptor species could be selected for exposure modeling?

Assessment and Measurement Endpoints

2. Assessment endpoints specify the valued ecological resources to be protected, such as local fish populations. They focus the risk assessment on particular components of the ecosystem that could be adversely affected by contaminants from the site. Please comment on whether the assessment endpoints selected (pp. 19-20) adequately protect the important ecological resources of the Hudson River. Are major feeding groups and sensitive species sufficiently covered by the selected assessment endpoints?
3. Measurement endpoints were used to provide the actual measurements used to estimate risk. Please comment on whether the combination of measured, modeled, guideline, and observational measurement endpoints used in the ERA (pp. 20-29) supports the weight of evidence approach used in the ERA.

Exposure Assessment

4. USEPA used several exposure models to evaluate the potential risks due to PCBs (see, ERA, pp. 37-71). Sampling data from USEPA, NOAA, NYSDEC, and USFWS collected from 1992-1996 were used to estimate current fish body burdens and dietary doses to avian and mammalian receptors. Future concentrations of PCBs were derived from USEPA's fate, transport, and bioaccumulation models, which are the subject of a separate peer review. Concentrations of PCBs in bird eggs were estimated by applying a biomagnification factor from the literature. Please comment on the appropriateness and sufficiency of this approach to estimate ecological exposure to PCBs.
5. Have the exposure assumptions (ERA, pp. 46-66 and Appendices D, E, and F) for each fish and wildlife receptor been adequately described and appropriately selected? Please discuss in detail.

Effects Assessment

6. For field-based toxicity studies, only a NOAEL toxicity reference value (TRV) was developed because other contaminants or stressors may be contributing to observed effects. Please comment on the validity of this approach. Also, please comment on whether the general approach of using uncertainty factors (interspecies, LOAEL-to-NOAEL, and subchronic-to-chronic) is appropriate in developing TRVs that are protective of Hudson River receptor species.

Risk Characterization/Uncertainty Analysis

7. USEPA calculated toxicity quotients (TQs) for all receptors of concern on both a total PCB and dioxin-like PCB (TEQ) basis. Please comment on whether the methodologies used in calculating these TQs are adequately protective of these receptors.
8. The risk characterization section of the ERA (Chapter 5, pp. 117-151) summarizes current and future risks to fish and wildlife that may be exposed to PCBs in the Upper Hudson River and current risks to fish and wildlife in the Lower Hudson River. Please comment on whether the risk characterization adequately characterizes the relative risks to ecological receptors (e.g., piscivores, insectivores) posed by PCBs in the Hudson River.

9. The uncertainty analysis is presented in Chapter 6 of the ERA (pp. 153-165). Have the major uncertainties in the ERA been identified? Please comment on whether the uncertainties (and their effects on conclusions) in the exposure and effects characterization are adequately described.

General Questions

1. A goal for Superfund risk assessments is that they be clear, consistent, reasonable and transparent and adequately characterize risks to sensitive populations (e.g., threatened and endangered species). Based on your review, how adequate are the ERA and the Responsiveness Summary when measured against these criteria?
2. Please provide any other comments or concerns, both strengths and weaknesses, with the ERA not covered by the charge questions, above.

Recommendations

Based on your review of the information provided, please select your overall recommendation for the ERA and explain why.

1. Acceptable as is
2. Acceptable with minor revision (as indicated)
3. Acceptable with major revision (as outlined)
4. Not acceptable (under any circumstance).

Peter deFur

May 7, 2000

EPA posed three principle study questions in this phase of the RI/FS:

1. When will PCB levels in fish meet human health and ecological risk criteria under continued No Action?
2. Can remedies other than No Action significantly shorten the time required to achieve acceptable risk levels?
3. Could a flood scour sediment, exposing and redistributing buried contamination?

General comments:

The Ecological Risk Assessment (EcoRA) was then designed and performed to provide information relevant to these three study questions. Most of the EcoRA addresses questions 1 and 2 in that the EcoRA predicts harm from present and future PCB exposures, including predictions of PCB levels now and into the future.

Generally, the EcoRA is designed and conducted in accordance with accepted practice. The assessment does seek to draw on several types and sources of data such as direct measurements, modeled PCB levels and comparisons with data from other investigations. The assessment also uses field observations in this analysis, not relying solely on lab or computer estimates. In this regard, the EcoRA is commendable.

The EcoRA could be improved by determining the presence (and abundance) or absence of large macroinvertebrates in the Upper Hudson River and tidal freshwater Lower Hudson River. This point is made in detail below and is not elaborated here. If present, the EcoRA has omitted an important component of the system. If absent, then the EcoRA should address why certain species or groups expected to be present, or historically present, are no longer found in their anticipated habitat.

Problem Formulation/Conceptual Model

Consistent with USEPA guidance on conducting ecological risk assessments (USEPA, 1997), the problem formulation step establishes the goals, breadth,

and focus of the assessment. As part of the problem formulation step in the ERA, a site conceptual model was developed (Chapter 2.3, pp. 11-19).

4. Please comment on whether the conceptual model adequately describes the different exposure pathways by which ecological receptors could be exposed to PCBs in the Hudson River.

The conceptual model does rely on measured and modeled values here and in other systems with PCB contamination to characterize the exposure pathways for ecological components. The Ecological Risk Assessment (EcoRA) indicates multiple exposure pathways (not sources), as food, water and direct (incidental) consumption of contaminated sediments. These three pathways are the known and measured pathways for PCB's from contaminated sediments into living ecosystem components based on other field, lab and computer modeling work. In this regard, the EcoRA is quite complete.

5. Was sufficient information provided on the Hudson River ecosystems so that appropriate receptor species could be selected for exposure modeling?

The EcoRA provided a great deal of information on which to base the selection of receptor species. But the approach used, as recommended by EPA at the regional and national level, is not complete in how this question is approached. The present EcoRA identified the sources of PCB's from local sources, sediments, etc., and quite effectively examined species that are or are likely impacted by the toxic effects of PCB exposures. The EcoRA, however, did not, however, begin with a complete (or nearly so) characterization of the ecosystem(s). The difference is whether the risk assessment effort is started with an assessment of ecosystem status, or with a source characterization. Because the sources have been known for decades, the EcoRA began with the present PCB contamination, and followed the PCB's through the known ecosystem components. Any elements of the ecosystem not known and already under consideration would be omitted, and I fear were not considered.

If major or important species or groups are present and not included in the EcoRA, then the ecosystems are not sufficiently well characterized to be sure that the receptor selection is appropriate.

Assessment and Measurement Endpoints

Assessment endpoints specify the valued ecological resources to be protected, such as local fish populations. They focus the risk assessment on particular components of the ecosystem that could be adversely affected by contaminants from the site.

6. Please comment on whether the assessment endpoints selected (pp. 19-20) adequately protect the important ecological resources of the Hudson River.

Ecosystem components that have changed since the earlier assessments may well have been overlooked. Even major ecosystem elements that were not already known or anticipated could well have been overlooked. Several groups or species fall into this category. The following would be expected in the Hudson River system, yet were given little or no treatment in the Eco RA:

- Crayfish in the upper reaches of the study area
- Zebra mussels in the entire study system
- Freshwater mussels in the upper reaches of the study area
- Blue crabs in the lower portions of the system, but especially in the tidal reaches of the freshwater Hudson River

The two decapod crustaceans are mobile, large, predatory and move substantial distances. Preliminary investigations on the part of this reviewer (Pers. Comm. with Bob Daniels of NY State Museum and Dave Strayer of Inst. For Ecosystem Studies), revealed the likely or known presence of all the above species or groups of species.

7. Are major feeding groups and sensitive species sufficiently covered by the selected assessment endpoints?

As raised in the answer to charge question number 6, if the large bivalve or decapods crustaceans are present in the Hudson River ecosystems, then the endpoint selection may not be adequate, depending entirely on the abundance and distribution of such species.

Of the above mentioned groups, both crayfish and blue crabs are omnivorous/carnivorous, highly mobile (blue crabs are migratory) and among the largest members of the benthic/epibenthic invertebrate community. Both crayfish and blue crabs disturb the sediments and feed on infaunal invertebrates and/or dead animals. These two features offer enhanced pathways for movement of sediment borne contaminants to move into the water column or the food web.

The endpoint of "habitats" was selected, and the lower river includes the tidal freshwater portion of the river. According to this reviewer's initial research, this portion of the river is used by blue crabs, especially small male crabs that will molt in these habitats, as in other tidal freshwater rivers of the east coast (see research by deFur in 1990, by A.S. Hines and by T. Wolcott and colleagues). As such, the crabs utilizing this habitat are more sensitive than usual to the effects of chemicals that alter hormone-driven systems, as molting is controlled by a steroid hormone (ecdysone).

Measurement endpoints were used to provide the actual measurements used to estimate risk.

8. Please comment on whether the combination of measured, modeled, guideline, and observational measurement endpoints used in the ERA (pp. 20-29) supports the weight of evidence approach used in the ERA.

The combination of several types of measurement endpoints is a strength of the EcoRA. The use of these types of endpoints that use information from quite different sources means that the weight of evidence can include consistency of data in the assessment.

Exposure Assessment

USEPA used several exposure models to evaluate the potential risks due to PCBs (see, ERA, pp. 37-71). Sampling data from USEPA, NOAA, NYSDEC, and USFWS collected from 1992-1996 were used to estimate current fish body burdens and dietary doses to avian and mammalian receptors. Future concentrations of PCBs were derived from US EPA's fate, transport, and bioaccumulation models, which are the subject of a separate peer review. Concentrations of PCBs in bird eggs were estimated by applying a biomagnification factor from the literature.

9. Please comment on the appropriateness and sufficiency of this approach to estimate ecological exposure to PCBs.

This approach of using multiple exposure "models" in the exposure assessment is appropriate and provides a richer result than if all the exposures were assessed from one type of information. While direct measurements from actual field data are often harder to explain, owing to the larger number of variables and the inability to identify and control variables in field work, their use makes the outcome more reliable and credible. Using modeled, measured and estimated exposures provides the opportunity to examine consistency and to make a more complete and accurate assessment. Without using multiple exposure "models", the exposure assessment would be limited. The values obtained from the literature have been peer-reviewed and evaluated in several different contexts, lending strength to their use here. These values are scientifically defensible for use on the same or similar species here in the Hudson River system.

10. Have the exposure assumptions (ERA, pp. 46-66 and Appendices D, E, and F) for each fish and wildlife receptor been adequately described and appropriately selected? Please discuss in detail.

3.4.1 Benthic invertebrates. This exposure pathway does not include carnivorous invertebrates such as crabs and crayfish. In the lower river,

estuarine snails and polychaetes (e.g. *Nereis*) may also be carnivorous and thus will also be exposed via diet from consuming contaminated invertebrates. At present, the model treats all benthic invertebrates as the same trophic level, when, in fact, they are first level carnivores if the decapods are present. Blue crabs are reported in the lower Hudson, and may be present in large numbers in the tidal freshwater reaches.

3.4.2 Fish. It is not clear from this explanation if the exposure analysis includes direct exposure to the eggs and fingerling fish; presumably it does, based on EPA's experience with fish egg susceptibility to PCB's and dioxins in the great Lakes system (EPA 1993. Interim Report on Data and Methods for Assessment of ,3,7,8 Tetrachlorodibenzo-p- dioxin Risks to Aquatic Life and Associated Wildlife", EPA /600/R-93/055. US EPA ORD Washington DC 20460), and the abundant literature on the topic (see Rolland, Gilbertson and Peterson, 1997 for review). The exposure of the egg to PCB's through the yolk also has to be addressed. Again, it is not clear if this exposure is adequately considered in the present model effort.

The comment that direct uptake of PCB's by invertebrates could not be assessed due to data and model incompleteness does not seem adequate to this reviewer.

3.4.3 The Avian exposure pathways seem complete, given that the uptake and distribution also addresses the deposition into the yolk and subsequent exposure of the developing embryo. Presumably, the reproductive and developmental endpoints rely on this exposure pathway. Section 3.4.3.3 refers to invertebrates as a single dietary source – this is correct so long as all occupy the same trophic level. If, however, significant dietary consumption of carnivorous or omnivorous or scavenger invertebrates occurs (crabs, crayfish), then this assumption is not valid and a second category of diet items must be added.

3.4.4 Mammalian exposure pathways and factors are standard as used in other assessments for similar situations. The use of data for mink make the data and results less uncertain. As with the avian exposures, this mammalian exposure through food will have to be adjusted if it turns out that the wildlife

species are consuming crabs, crayfish, zebra mussels or freshwater mussels, as would be expected if these groups and species are present.

The statement of the first paragraph page 63 regarding the sources of information on diets for the mammals is not fully satisfactory; compiling the information into a table would be a great help for ease. Are there any species or major food groups that occur (or not) in the areas studied in the literature cited, and not directly applicable to the Hudson River system?

Effects Assessment

For field-based toxicity studies, only a NOAEL toxicity reference value (TRV) was developed because other contaminants or stressors may be contributing to observed effects.

11. Please comment on the validity of this approach.

The use of only NOAEL toxicity values would seem to be an appropriately protective method for using data from field-derived data. That is, when data on toxicity were obtained from actual field experiments, only NOAELs were used. This approach is valid if toxicity to the endpoint in question is principally determined by PCB's, AND if the interaction between PCB's and any other stressors is neither synergistic nor resulting in novel outcomes. Another way to consider this point, is if there is reason to believe that removing the stressor of the PCBs will likely diminish the harmful effect to the ecosystem endpoint.

On the other hand, if other chemicals have highly synergistic interactions with PCB's, then the use of NOAELs will not provide sufficient protection. In the present case, data from Cook (see chapter in Rolland, R., M. Gilbert and R. Peterson, eds. 1997. Chemically Induced Alterations in Functional Development & Reproduction of Fishes. 220 pp. SETAC Press, Pensacola, FL) and from Bemis and Seegal (Bemis, J.C. and Seegal, R.F. 1999. Polychlorinated biphenyls and methylmercury act synergistically to reduce rat brain dopamine content *in vitro*. Environ. Health Perspect. 107: 879-885), indicate that PCB's can act synergistically with other contaminants that are common in many areas, including the Hudson River. These contaminants include dioxin and methyl

mercury, both of which are found throughout waters of the US. If these compounds act synergistically in the Hudson River system, then the actual effects could be many times greater than anticipated by the EcoRA.

12. Also, please comment on whether the general approach of using uncertainty factors (interspecies, LOAEL-to-NOAEL, and subchronic-to-chronic) is appropriate in developing TRVs that are protective of Hudson River receptor species.

The general approach of using uncertainty factors has proven to be protective, notwithstanding criticisms in the literature. Uncertainty factors are not appropriate if there is reason to believe that the factor of safety is either much greater or less than the actual difference between real and expected values. EPA's data used in other applications (such as the Great Lakes and national guidance on water quality criteria and standards) suggests that ten fold safety factors are appropriate for interspecies, NOAEL to LOAEL and subchronic - chronic extrapolations. Considering that the present applications use only a single safety factor, and thus never extrapolate more than an order of magnitude, there is less chance that the results dramatically over estimate the risks to aquatic life and wildlife.

The greater concern is whether there are enough data and experience with PCB's and related compounds for the receptors in this case to be confident that the results are not dramatically under estimating the risks.

Risk Characterization/Uncertainty Analysis

USEPA calculated toxicity quotients (TQs) for all receptors of concern on both a total PCB and dioxin-like PCB (TEQ) basis.

13. Please comment on whether the methodologies used in calculating these TQs are adequately protective of these receptors.

Based on the concept of using TEQ based evaluations that are accepted internationally, this EcoRA is wise to use both forms of toxicity quotient

analysis. The strength of this approach is that it has been worked out for problem

The risk characterization section of the ERA (Chapter 5, pp. 117-151) summarizes current and future risks to fish and wildlife that may be exposed to PCBs in the Upper Hudson River and current risks to fish and wildlife in the Lower Hudson River.

14. Please comment on whether the risk characterization adequately characterizes the relative risks to ecological receptors (e.g., piscivores, insectivores) posed by PCBs in the Hudson River.

The risk characterization does a good job of characterizing the risks as described in the body of the EcoRA. If the EcoRA has failed to identify a significant food item or trophic component (e.g. crayfish, crabs), then the risks may be much greater than characterized here. The greatest source of error is likely to be the presence of crayfish in the upper Hudson in sufficient numbers that they are a major food source for such animals as mink, raccoon, some birds, etc. If crayfish make up a significant part of the diet, and the crayfish are not contaminated, then the actual dietary uptake of PCB's is less than predicted in the EcoRA. If crayfish are in the diet and contaminated, then the actual PCB uptake will be greater than predicted at present.

A related issue is the role of zebra mussels in the trophic system of the Hudson River. The EcoRA gives some consideration to zebra mussels, but does not adequately evaluate the consequences to the trophic system and transfer of PCB's through the food web. Such a large biomass and of filter feeders is known to alter the trophic system of a system. Two recent evaluations have demonstrated this point – the loss of oysters from the Chesapeake Bay, and the population explosion of zebra mussels in certain Great Lakes systems. This point needs for analysis in the present EcoRA.

The uncertainty analysis is presented in Chapter 6 of the ERA (pp. 153-165).

15. Have the major uncertainties in the ERA been identified?

Yes, with the exception of the elements of the ecosystem – does the system

contain the species or groups identified in the earlier section? The uncertainty analysis is almost exclusively qualitative. Not being a quantitative uncertainty analysis expert, it is not clear that more quantitative analysis could be or should be conducted. But I look forward to reading the comments of the other reviewers, some of whom have expertise in quantitative uncertainty analysis.

16. Please comment on whether the uncertainties (and their effects on conclusions) in the exposure and effects characterization are adequately described.

The results are adequate, but could be presented and likely conducted more quantitatively. It is not clear how the results are influenced by the use of tri+ PCB's in the model estimates as used here. Did EPA attempt any alternative approaches and obtain results that could be compared and presented? Such comparisons would be more than helpful in satisfying concerns that the tri+ PCB assessment introduces an error that could be corrected AND that alters the outcome of the assessment.

General Questions

A goal for Superfund risk assessments is that they be clear, consistent, reasonable and transparent and adequately characterize risks to sensitive populations (e.g., threatened and endangered species).

17. Based on your review, how adequate are the ERA and the Responsiveness Summary when measured against these criteria?

The EcoRA is more than adequate in conforming to the EPA criteria.

Improvements could be made in avoiding jargon and in stating conclusions in a more direct and obvious fashion. When several lines of evidence converge in a clear and obvious pattern, some with an obvious outcome (e.g. the consistent and large TQ's), the EcoRA does make a clear conclusion. Other areas are not so clear and the conclusions or outcome statements in most of these cases are less definitive.

I do not recommend writing an additional characterization, or dramatically altering the present one. The present EcoRA can and should be improved as indicated in the peer review.

One of the areas not discussed was the return of species that are now excluded from the area because of the PCB contamination. Some consideration is given to this issue with regard to individual species – bald eagles. But the EcoRA should address whether other species may increase dramatically or return if the PCB levels fall below some point, or by 90%.

10. Please provide any other comments or concerns, both strengths and weaknesses, with the ERA not covered by the charge questions, above. The major comment is included in general comments above – an assessment of the status of the ecosystem should have revealed the expected presence of zebra mussels, freshwater mussels, crayfish, and blue crabs in the tidal freshwater Hudson River.

One strength of the EcoRA is the structure and consistency from section to section. Some of the repetition of structure and following the form of the EcoRA as set out results in a larger document that repeats material. That result is an unavoidable consequence of needing to follow a strict form.

The EcoRA needs to conduct more analysis of the presence or growth of the populations of zebra mussels, especially in the upper reaches of the river. The brief discussion does indicate that this species may represent a massive flux of PCB's out of sediments (or the water column) and into the food web. If this is the case, as may be happening in the Great Lakes, then the entire model for PCB changes in the future may be in error, although the magnitude of this error is not clear. The most likely outcome is an extension of the time for PCB levels to fall, given the extensive and massive loading of PCB's in this river system.

Recommendations

Based on your review of the information provided, please select your overall recommendation for the ERA and explain why.

1. Acceptable as is
 2. Acceptable with minor revision (as indicated)
 3. Acceptable with major revision (as outlined). The recommendation is for a major element to be added to the EcoRA. This element is determining the distribution and abundance of crayfish, blue crabs, zebra mussels and freshwater clams in the study area. This work may be simple and straightforward and not require extensive modification, but these are important elements of the system that may have been omitted. This reviewer's initial research indicates that blue crabs are abundant in the tidal freshwater portions of the river, that crayfish do occur in the upper portion of the river and that zebra mussels and freshwater mussels have historically occurred in the upper regions.
 4. Not acceptable (under any circumstance).
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Lawrence Kapustka

Ecological Risk Assessment

The goal of the Ecological Risk Assessment is to evaluate the risks to ecological receptors associated with exposure to PCBs in the Hudson River in the absence of remedial action of the PCB-contaminated sediments (i.e., under baseline conditions). The following documents will be provided to the peer reviewers:

Primary

- *Baseline Ecological Risk Assessment, August 1999*
- *Responsiveness Summary for the Baseline Ecological Risk Assessment, March 2000*

References

- *Ecological Risk Assessment Scope of Work, September 1998*
- *Responsiveness Summary for Ecological Risk Assessment Scope of Work, April 1999*
- *Executive Summary for the Baseline Ecological Risk Assessment for Future Risks in the Lower Hudson River, December 1999*
- *Executive Summary for the Human Health Risk Assessment, Upper Hudson River, August 1999*
- *Executive Summary for the Human Health Risk Assessment, Mid-Hudson River, December 1999*
- *Executive Summary for the Revised Baseline Modeling Report, January 2000*
- *Suggested charge questions from the public for the ERA, February 2000*

The reference documents listed above are being provided to the reviewers as background information, and may be read at the discretion of the reviewers as time allows. The reviewers are not being asked to conduct a review of any of the background information.

Additional Reassessment RI/FS documents are available on USEPA's website (www.epa.gov/hudson) and/or by request. Additional documents include the following:

- *Hudson River Reassessment RI/FS Database, August 1998*
- *Executive Summaries for other USEPA Reassessment RI/FS Reports*
- *Peer Review Reports from first two peer reviews*
- *Responsiveness Summary for first peer review*

Specific Questions

Problem Formulation/Conceptual Model

1. *Consistent with USEPA guidance on conducting ecological risk assessments (USEPA, 1997), the problem formulation step establishes the goals, breadth, and focus of the assessment. As part of the problem formulation step in the ERA, a site conceptual model was developed (Chapter 2.3, pp. 11-19). Please comment on whether the conceptual model adequately describes the different exposure pathways by which ecological receptors could be exposed to PCBs in the Hudson River. Was sufficient information provided on the Hudson River ecosystems so that appropriate receptor species could be selected for exposure modeling?*

The Conceptual Model developed for the Hudson River Ecological Risk Assessment (EcoRA) was adequate for a preliminary examination of broad categories of potential exposures to ecological resources in the Main Channel of the Hudson River. Construction of a Conceptual Model requires multiple iterations among stakeholders, risk managers, and risk assessors. To facilitate this process, the USEPA instituted Biological Technical Advisory Groups (BTAGs)¹ in the early 1990s. BTAGs were intended to provide a forum to engage critical discussions on major issues related to any particular site. It is remarkable, that with the opportunity to air views on a major resource such as the Hudson River, that this reassessment effort was constrained by such an elementary-level Conceptual Model.

¹ In some Regions called Ecological Technical Advisory Groups (ETAGs).

One would hope that the administrative record, which should have included minutes of BTAG meetings, would have documented decisions made with respect to finalizing the Conceptual Model for the EcoRA. No such materials were provided for this review. Moreover, in response to a direct question I posed during the briefing meetings in March 2000, we were informed that no published materials characterizing the biological communities were available from this project. Ultimately, the Conceptual Model should be simplified to focus discussions on selection of assessment endpoints, guide the selection of surrogate species used as assessment species, and to evaluate potential measurements endpoints that would address the assessment endpoints. If such dialogue occurred, it was not captured in any of the documents available for review. The detail provided regarding the Conceptual Model fails to meet minimum standards of completeness, openness, and clarity of the process.

At a minimum, the Conceptual Model for an EcoRA of the scale of this project required a succinct description of the major ecological resources of the system. To do this, one requires a description of the major physical/biological units that ecologists would routinely use to describe the resources. For different recognized resources, this means descriptions of habitat. From an ecological view, this requires consideration of connections among critical habitats for the dominant species and for those of greatest interest to the public. To be of value for ecological analyses, this requires more than a generic list of the species that inhabit the river. Clearly, much more is known about the Hudson River system. Only after this ecological system overlay is added to a conceptual model of contaminant fate and transport (potential exposure) can meaningful discussion of assessment endpoints be occur. Most of the populations of species of interest identified in the EcoRA (particularly the fish, birds, and mammals) are not confined to the channel of the River. The influence of tributaries, wetlands, and other features of the flood plain on these populations is not considered in this EcoRA. By these omissions, one has little context to understand mitigating factors that relate to exposure or population-level effects. The superficial nature of the Conceptual Model foreshadow many of the subsequent deficiencies that define the character of this EcoRA.

Assessment and Measurement Endpoints

2. *Assessment endpoints specify the valued ecological resources to be protected, such as local fish populations. They focus the risk assessment on particular components of the ecosystem that could be adversely affected by contaminants from the site. Please comment on whether the assessment endpoints selected (pp. 19-20) adequately protect the important ecological resources of the Hudson River. Are major feeding groups and sensitive species sufficiently covered by the selected assessment endpoints?*

Articulating Assessment Endpoints is both the most difficult and the most important feature of an EcoRA. Considerable dialogue is needed among stakeholders and risk assessors to ensure (1) the ecological resources of interest to stakeholders are identified; and (2) that the expressions of these values are articulated in terms that can be assessed through scientific processes. If the values to be protected (assessment endpoints) are not assessable through hypotheses testing or weight-of-evidence approaches, then they cannot be addressed properly in the EcoRA. Poorly stated assessment endpoints

are ambiguous and do not suggest reasonable measurement endpoints that allow meaningful determination of risk.

The Assessment Endpoints stated for this EcoRA were defined poorly. Of the five bulleted "assessment endpoints" (middle of page 20, August 1999 Vol. 1 of 3), the lead action is "Protection." Protection is a regulatory or management activity, not an ecological condition.

The first assessment endpoint (first bullet) could have been improved if it were phrased in terms of viable populations of fish and wildlife. Subsequent, component (or subsidiary assessment endpoints) could then have specified which fish populations and which wildlife species were selected as surrogates for guilds, trophic groups, or other groupings. Typically, the maintenance of viable benthic communities would be defined as a subordinate assessment endpoint to fish populations instead of being granted equal standing with the fish or wildlife populations. In other words, this first assessment endpoint should have been subsumed into portions of the second and third bulleted items.

Apart from the problem with "protection" being included in bullets two and three, these statements of assessment endpoints were reasonable starting points. Unfortunately, the path forward from these broad statements was not described sufficiently, nor was it apparent that much thought went into placing these broad statements into project specific context. It was at this point in the process, that the BTAG should have engaged in an iterative process to refine the Conceptual Model and to refine the Assessment Endpoints. Explicit descriptions of the interface of critical ecological relationships among key valued fish and wildlife species and potential PCB exposure routes should have occurred. If such discussions occurred, they were not captured in the reports and background information provided for this review.

The third and fourth bullets (Protection of Wildlife and Protection of Significant habitats fail the formal tests of assessment endpoint². These may well have been expressions of valued resources forwarded by various stakeholders. However, the obligation of the risk assessment team was to have become sufficiently engaged in the dialogue so that these expressions could be translated into endpoints that could be assessed. Instead the assessment states vaguely that there were "discussions with agency representatives." There was an obligation to articulate the critical factors for the eight designated areas in terms that could be assessed formally.

No description or explanation was provided regarding the selection of species in the macroinvertebrate, fish, avian, or mammalian that are to be assessed or that served as surrogates for species to be assessed. This section begs for a coherent description of the biological communities (composition and

² Assessment Endpoint-Formal expression of the actual environmental value to be protected; Measurement Endpoint-The physical, chemical, biological, or ecological condition that is quantified; ideally, this yields information on the effect of a hazard; to be useful in site assessment, the measurement endpoint must correspond to or be predictive of an assessment endpoint.

abundance of species) which include the assessment species. This section should also have included explicit criteria which were used to select the species of interest, so that a reader might know which other species were considered, which species represent collections of other species, reasons why other species were not selected, etc. Instead, there was a rather authoritative presentation without any supporting documentation. What was the rationale for choosing largemouth bass over small mouth bass? Were crayfish considered? What assumptions were imposed on the selection process?

The criticisms presented are not raised from mere academic perspective, but rather as cornerstones for conducting quality EcoRAs. Each assessment species has different requirements or habitat preferences. Each also has different behavioral features which influence habitat use (i.e., where they feed, when they feed, where they loaf, where they breed, and others). Without such information, it is impossible to determine whether the procedures used to estimate exposures were reasonable or whether they were wildly biased in one direction or another.

The report submits that the assessment endpoints were phrased as assessment questions and paired with measurement endpoints. That was not done effectively. Most of the "measurement endpoints" simply restate the "assessment question" without providing meaningful information. An example of the construction that would have been appropriate is:

Assessment Endpoint:sustainable populations of largemouth bass

Assessment Endpoint Question: ...Are PCB concentrations in the Hudson River sufficiently high to adversely affect reproduction of largemouth bass?

Measurement Endpoint₁:concentration of PCBs in largemouth bass tissues (whole body, eggs) to be compared against toxicity response relationships.

Measurement Endpoint₂:size (age)-class distribution of largemouth bass at (appropriate number) sites in the Hudson River system.

Methodology for ME₁obtain fresh tissue samples from selected locations; process and analyze the tissues [state analytical chemistry procedure and detection limits]

Methodology for ME₂[choose among several seining, electroshocking, trapping methods] to enumerate populations of

different size/age classes at representative locations in the River; [state DQOs].

The first measurement endpoint would permit direct measurement and modeling efforts to extend or interpolate sampling data to other portions of the river. It would permit comparisons to threshold values and re-calculation of data into TEQ values. But it would also strive to use more than a mere threshold concentration (and thus be limited to a quotient); it would provide a basis for expressing a probability of a 10%, 20%, or 50% impairment in reproduction.

The second measurement endpoint would ask the central question of whether the exposures are translated into ecological effects. If the size (age)-class distribution shows an abnormal profile, then one has evidence to corroborate predicted effects. Alternatively if the data indicate a normal profile, then it suggests that either recruitment from other areas is occurring or the predicted effects are being mitigated by factors that lower exposure levels or other important biological processes.

As they were stated in the report, the assessment questions and measurement endpoints restrict opportunities for developing a robust EcoRA. They forecast that the EcoRA would be an exercise in Quotients and that ecological data would have very little importance. For example, for fish, the first four measurement endpoints were structured to look solely at measured or modeled PCB concentrations in relationship to point estimates (TRV, AWQC, or sediment benchmark). The fifth endpoint ("available field observations on presence or relative abundance...") provided little basis for setting meaningful data quality objectives to be used to make this assessment.

The specific directive and question we were presented for this review [*Please comment on whether the assessment endpoints selected (pp. 19-20) adequately protect the important ecological resources of the Hudson River.*], and [*Are major feeding groups and sensitive species sufficiently covered by the selected assessment endpoints?*] can be answered yes. But a more important question, "Were the assessment endpoints articulated properly?" the answer is clearly no.

3. *Measurement endpoints were used to provide the actual measurements used to estimate risk. Please comment on whether the combination of measured, modeled, guideline, and observational measurement endpoints used in the ERA (pp. 20-29) supports the weight of evidence approach used in the ERA.*

As described under Charge 2 above, the assessment endpoints were not properly articulated and the measurement endpoints were inappropriately restrictive. In effect, the focus of the EcoRA was on PCB concentrations in various media (water, sediment, and selected tissues). These data were analyzed in different ways to generate total PCBs and TEQ values, which were then compared to TRVs. Not discounting the tremendous effort this involves to qualify all the analytical data, in the end this distills down to different ways to calculate ratios. In order to have a solid weight-of-evidence approach, much

greater credence to ecological data was required. EPA made no effort to characterize any of the populations of fish, birds, or mammals of interest in the project area. Their field work was limited to a small benthic community survey, and in the end the results of the survey was largely discounted.

If one applies Hill's (1965)³ logic tests to the suite of measurement endpoints, it becomes obvious that field data are needed to establish a weight of evidence argument (partial list of Hill's criteria):

- *strength* – [Is the magnitude of effect associated with exposure to the stressor high?],
- *gradient* – [Does a positive correlation between stressor and effect exist, (i.e., is there a "dose"-response relationship?)],
- *experimental evidence* – [Did the data analysis confirm or reject the null hypotheses?], and
- *coherency* – [Are the hypotheses tested relative to the stressor effects consistent with ecological and toxicological knowledge?].

Absent collection of ecological data specifically for the project, it was possible to rely on data collected for other purposes. Most of the ecological data from other sources that were cited in the EcoRA are counter to the predicted adverse effects generated by the modeling approach. If one wishes to claim reliance on a weight-of-evidence approach, then such data cannot be dismissed. So to respond to the charge of the reviewers, the simple answer is yes a weight-of-evidence approach could be developed from "the combination of measured, modeled, guideline, and observational measurement endpoints used in the ERA," but in the end, that was not done.

Exposure Assessment

4. *USEPA used several exposure models to evaluate the potential risks due to PCBs (see, ERA, pp. 37-71). Sampling data from USEPA, NOAA, NYSDEC, and USFWS collected from 1992-1996 were used to estimate current fish body burdens and dietary doses to avian and mammalian receptors. Future concentrations of PCBs were derived from USEPA's fate, transport, and bioaccumulation models, which are the subject of a separate peer review. Concentrations of PCBs in bird eggs were estimated by applying a biomagnification factor from the literature. Please comment on the appropriateness and sufficiency of this approach to estimate ecological exposure to PCBs.*

Characterization of PCB concentrations in selected sampling stations in the Upper and Lower Hudson River in water and sediments were quite extensive. There were also a number of measures of PCB congener concentrations in benthic invertebrates and in fish tissues. These measured values were used to describe changes in congener pattern downstream and were compared among media for co-located samples. An analysis of congener data was undertaken to bridge different analytical techniques reported from various studies. This analysis was quite elegant and provided reasonable descriptions of downstream and temporal changes in patterns among congeners. For the most part, the "fate and transport"

³ Hill, A. B. 1965. The environment and disease: Association or causation? Proc. Royal Soc. Med. 58: 295-300.

components of exposure assessment within the physical compartments was done well. The two most problematic features of this fate and transport effort were the change in sediment sampling cores and the failure to consider that bio-perturbation could account for seasonal or episodic bursts of suspended materials in the water column. Spawning activities, particularly by common carp; burrowing by various benthic organisms; as well as wake and prop motion from boats, disturb sediments. Although the cause for the episodic events was not attributed correctly, the description of patterns of suspension was probably still reasonable.

The modeling effort to project the various concentrations into biological tissues cannot be dissected adequately here, because the documentation of exposure models was not provided and review comments from a separate panel are not yet available. There was some indication that the predicted values were tested against measured tissue concentrations from fish sampled between 1992 and 1996. Typically, there are many assumptions in exposure models. It is not clear which were calibrated to fit the measured data, (i.e., which parameters were adjusted in the benthic or fish bioaccumulation models to bring the predicted values in line with the measured values). The accuracy of the long-term predictions depends on what was done in these calibration steps. Presumably, these features will be addressed thoroughly by the other peer review panel.

The use of measured concentrations in food items to estimate "current" dietary exposure was appropriate. However, there are many other critical assumptions in exposure models. One needs to revert to the Conceptual Model to address whether the underlying assumptions were reasonable or not. There were several starting assumptions that should have been evaluated more thoroughly in this section (albeit that would have required a more sophisticated conceptual model than the one reported). The assumption that piscivorous birds received 100% of their diet from main channel fish was appropriate for a first-cut screening level risk assessment. However, for a project at the stage of this re-assessment, much more was warranted.

Bald eagles for example are quite opportunistic in their feeding preferences. Individuals (more accurately nesting pairs of) bald eagle diets in other portions of the country range from <10% to nearly 100% fish. Foraging, though it may focus on the main channel, would also extend a few kilometers overland and into other water bodies. Eagles will also take ducks, rabbits, and other similar sized animals when an opportunity presents itself. Tests of different scenarios are important to explore the likelihood of different levels of exposure. Similarly, the exposure assumptions for raccoon, otter, and mink should have considered different scenarios that could be refined with site specific information. Too little effort went into these critical steps.

The importance of getting exposure right, is made more important by the overall approach used by EPA in this EcoRA. In particular, the sole reliance on Hazard Quotients, and underscoring the different magnitudes of exceedence, elevates the importance of exposure assumptions. The practice of selecting

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the lowest threshold values (No Effect Levels discussed in the effects charges) and often dividing these concentrations by 10, provides a very small TRV. When one has a very small TRV, a very small change in exposure produces a very large quotient. If indeed the dietary exposure is overestimated, as it might be, these assumptions alone can result in 50-fold changes in the hazard quotients. Clearly, more importance and more effort should have been placed of refining the exposure assumptions for current conditions. If this were done, then the forecasts for exposures over the long-term would be more accurate.

5. *Have the exposure assumptions (ERA, pp. 46-66 and Appendices D, E, and F) for each fish and wildlife receptor been adequately described and appropriately selected? Please discuss in detail.*

One of the most critical assumptions in the exposure estimates for fish and wildlife was setting the "Area Use Factor" or "Forage Effort" equal to one. Though the total area covered by the channel of the Hudson is large, it is relatively narrow with respect to landscape use patterns of wildlife. Even for fish, consideration of connections to tributaries, or connections between deep-water areas and shallows have great influence on exposure.

In general, the equations used to estimate exposure project a sense of detailed knowledge that far exceeds reality. Each of the input parameters to the equations in itself is an estimate with many underlying assumptions. When used as algebraic expressions, one simply calculates a value. The choice of input parameters appears to have been skewed to provide "protective" levels. The problem this introduces is that each "protective" value gets applied on top of other "protective" values. After two such protective values are pieced together, the result is that predictions cannot be verified because the calculated value is outside the range of experience (measured values). Two steps that may have been performed, but are not prominent in the report, that could illuminate problems with assumptions, are sensitivity analysis and probabilistic risk assessment.

A detailed sensitivity analysis should have been performed to identify the level of precision required for different input parameters (and assumptions). Such an effort would rather quickly focus on a select few parameters that could then have been given special consideration. Critical parameters such as K_{ow} have different reported values. How sensitive are the exposure models to variations in the K_{ow} ? How critical is the lipid fraction of the receptor? ... metabolic rate? assimilation efficiency? rate of depuration? and many others.

A probabilistic approach would have permitted additional sensitivity analysis as well as place the estimates in closer agreement with field data. For example, the actual concentrations of PCBs in benthic invertebrates could have been "sampled" through thousands of runs to produce percentiles of different exposure concentrations. Each of the major assumptions could have been described as a function about the mean to eliminate the compounding of error that occurred from using multiple "protective" values.

It is important to note, that the basic structure of the exposure estimating procedures, outlined in this section, follows normal practice for EcoRAs. However, the level of detail achieved in this EcoRA was appropriate for a preliminary study, or a screening-level EcoRA. The use of *protective* assumptions is fully warranted for screening level efforts – if despite the assumptions, there is no indication of a problem, then the work is done; however, if there is an indication of a problem, it is a signal that more effort is needed. In this project, it was quite surprising to find that the follow-up definitive work was not done. The effort describe here was fine for studies at the start of the reassessment. It was quite inadequate for the current stage of the project.

Effects Assessment

6. *For field-based toxicity studies, only a NOAEL toxicity reference value (TRV) was developed because other contaminants or stressors may be contributing to observed effects. Please comment on the validity of this approach. Also, please comment on whether the general approach of using uncertainty factors (interspecies, LOAEL-to-NOAEL, and subchronic-to-chronic) is appropriate in developing TRVs that are protective of Hudson River receptor species.*

For most of the last decade EPA and others have known of the significant technical limitations pertaining to the use of NOAELs and LOAELs. The arguments were presented by Chapman *et al.* (1996)⁴ and are the basis of a growing consensus that the ANOVA designs used to estimate threshold values are inappropriate for ecotoxicology or for risk assessment. Briefly, they have shown that the concentration interval, the number of replicates, and variance, (both in responses and in measurement of concentrations), have bearing on the value obtained than the *true* toxic response. Moreover, the point estimates do not provide any information related to the shape of the concentration-response relationship. There is no distinction between steep-sloped responses or shallow-sloped responses. A much more useful construct is one that uses a regression model to describe and effect-level (e.g., EC₂₀). The regression approach provides confidence intervals as well as a ready means of translating the information into a risk characterization. Also, all the data from a regression model study are used to arrive at the point estimate, providing a more robust analysis of the data (i.e., less subject to nuances of study design. In using NOAEL and LOAEL values from individual studies to calculate TRVs, there is no opportunity to know how much experimental error is imbedded in the number. It would be better (if one felt compelled to use NOAEL-LOAEL data) to use data from more than one study. The NOAELs and LOAELs of different studies could be arrayed ala Long and Morgan (1991)⁵. Alternatively, the MATCs (Maximum Acceptable Toxicant Concentration determine as the median or the geometric mean of NOAEL and LOAEL), of individual studies could be calculated and a grand mean of all studies used as the TRV.

⁴ Chapman, P. M., R. S. Caldwell, and P. F. Chapman. 1996. A warning: NOECs are inappropriate for regulatory use. *Environ. Toxicol. Chem.* 15: 77-79.

⁵ Long, E. R. and L. G. Morgan. 1991. The potential for biological effects of sediment-sorbed contaminants tested in the National Status and Trends Program. NOAA Technical Memorandum NOS OMA 52, National Oceanic and Atmospheric Administration. 175 pp.

There is no technical foundation for a decade safety factor being applied for any extrapolation (i.e., either interspecies or sub-chronic to chronic). The arguments against using assessment factors were presented by Chapman *et al.*, (1998)⁶. The use of an assessment or safety factor is entirely a policy decision, notwithstanding that some scientists might wish to hedge their answers and favor assessment factors.

Even more troubling than the use of NOAELs and LOAELs, is the extensive reliance on the TRV construct. The purpose for developing Threshold Toxicity Response Values is to provide a rapid means of screening chemicals into or out of a more detailed risk analysis. The comparison of the TRV and the Environmental Concentration provides the simplest means for identifying situations of "little or no concern" versus situations with "possible concern." Because the Hazard Quotient that emerges from this comparison is a unitless value, and because there is no scalar to equate the severity of an increasing quotient to a toxicological response, the approach has no further utility than to classify situations into the two categories. A quotient of 100 should not be characterized as being 10-fold worse than a quotient of 10. A quotient of 100, based on a *protective* TRV and a high-end concentration range (e.g., 95% UCL) may still be below the toxicity threshold response level. Due to the several policy decisions that force the risk assessor to pick the lowest threshold levels and the highest possible environmental concentrations, many (maybe even most) exceedences are in the toxicity *di minimus* range.

The use of Hazard Quotients has great value in streamlining EcoRAs. The role for the Quotients, as stated above, is in the screening phase of a risk assessment, to focus on key receptor groups, on selected portions of the site, and to suggest topics for more detailed investigation. When used properly, one can justify the high bias toward protectiveness. The consequence of not being screened out is that the costs of investigation increase. As one proceeds through the more detailed EcoRA, the *protective* default assumptions are replaced by empirical site data. Moreover, as one reaches the later stages of the EcoRA, the shift of emphasis should proceed from "what is possible toxicologically" to "what is probable ecologically." In other words, one begins to place the toxicological data in context. Whereas the toxicological data (especially laboratory studies) were developed for individual level effects, the ecological data incorporates population- or community-level dynamics. This is not how EPA conducted this EcoRA. Rather, EPA used a screening-level tool for what should have been a definitive-level EcoRA. Consequently, EPA has greatly overstated the level of risk to receptors.

Risk Characterization/Uncertainty Analysis

7. USEPA calculated toxicity quotients (TQs) for all receptors of concern on both a total PCB and dioxin-like PCB (TEQ) basis. Please comment on whether the methodologies used in calculating these TQs are adequately protective of these receptors.

⁶ Chapman, P. M., A. Fairbrother, and D. Brown. 1998. A critical evaluation of safety (uncertainty) factors for ecological risk assessment. *Environ. Toxicol. Chem.* 17:99-108.

As with the TRV approach, the TEQ is a simplified tool to handle a lot of complicated information quickly and easily to arrive at a screening-level determination of risk. There has been much written to justify the TEQ, mostly for human health applications. But one should not lose sight of the large number of assumptions imbedded in the summaries. A frank analysis of the process highlights that the underlying data set used to establish relative risk among compounds is far from robust. There are multiple assumptions, all biased to be protective, that contribute to the relative values. The physical chemical properties of dioxins, makes them extremely difficult to work with. Large measurement errors are the norm. Add to this the reality that quantifying concentrations of PBB congeners can be as much art as science, one has a large uncertainty. Again, the nature of the process is to err on the side of protection.

Whereas this may be appropriate for most human health concerns, and it may be fine as a forecasting effort for siting a new facility, there is little reason to rely so extensively on the TEQ approach for a definitive EcoRA. EcoRAs of existing sites have the luxury of relying on analyzing populations and communities of receptors directly. A TEQ approach might have merit in assigning causality to a documented adverse population or community condition. However, as a stand-alone forecaster, the TEQ approach is designed to be biased and as such will predict harm when none may exist.

8. *The risk characterization section of the ERA (Chapter 5, pp. 117-151) summarizes current and future risks to fish and wildlife that may be exposed to PCBs in the Upper Hudson River and current risks to fish and wildlife in the Lower Hudson River. Please comment on whether the risk characterization adequately characterizes the relative risks to ecological receptors (e.g., piscivores and insectivores) posed by PCBs in the Hudson River.*

Virtually all of the Effects Characterization (Chapter 4) dealt with evaluation of exposure concentrations and toxicity tests reported in the literature. No effort was made to relate the exposure levels to effects – and certainly there was no effort to relate toxicity measurements to population-level effects. Experience in ecotoxicology is that concentrations shown to have effects on individuals, typically requires similar or higher concentrations to be manifest in the field. Here, however, toxicity data were routinely divided by ten as an *uncertainty* factor. This policy issue belongs with risk managers and should not be imbedded in the technical portion of the EcoRA. The "Effects" chapter set a target concentration well below all known no-effect levels. Subsequently, exceedence of these target concentrations, biased toward protection, were used as confirmatory *evidence* to claim adverse effects were occurring, that unacceptable risks were prevalent, and for the future unacceptable risks were projected.

The risk characterization chapter also relied on national water quality or sediment quality criteria that were established to regulate discharges. Even for discharges, site specific characteristics are used to adjust the values. It is inappropriate to merely compare concentrations to these values without more in-depth analyses. As with the HQ, exceedence does not mean harm will occur. It is merely indicating that under some circumstances harm may occur. The approach used here would have been appropriate for a screening-level EcoRA, but is inadequate for an 11-year reanalysis of a site.

Exceedence of a toxicity NOAEL adjusted by a 10-fold safety factor should not be construed as an indicator of adverse population effects. Even exceedence of a toxicity-based LOAEL requires evaluation of all circumstances affecting bioavailability and compensatory mechanisms that govern populations before a conclusion of unacceptable risk is warranted.

Fish Populations (p. 128-)

Point 1: ... *bass, bullhead, spottail shiner, yellow perch, pumpkinseed* ... The information of survey data was described as qualitative. Because some would interpret this to mean *presence* versus *absence* comparison, instead of quantifiable population data, this characterization is misleading. For the EcoRA, had legitimate assessment endpoints been articulated in terms of sustainable populations, the clear conclusion would have been that PCB concentrations were not adversely affecting the populations. As is, the EcoRA rejected critical information that would have dismissed PCBs as harming fish populations. This apparently was done in favor of elevating an untested speculation that problems are occurring, even though population data indicates no such harm.

Point 2: This characterization even more boldly rejected relevant data so as to accept untested speculation in a most unscientific manner. It was quite disingenuous to dismiss sustained monitoring information over the entire period of peak contamination (a couple of decades) in order to hold out that with a little more time, with substantially lower levels of contamination, effects will manifest into dire consequences.

Point 3: In the face of a longer period of monitoring showing that an endangered species has continued to increase in population, in spite of the major insult from PCB contamination, the EcoRA again reached for extraneous dismissals such as "decades are too short to evaluate populations of sturgeon" (that require seven to ten years to mature).

Point 4: This further exposes an apparent pre-disposition to find that PCBs were harming populations. Coupled with the first three points, there is evidence that *r*-selection and *K*-selection species have increased their populations during the period of highest PCB exposures. Those data refute any speculative assignment of adverse effects of PCBs to these receptors. The data demonstrate the extensively protective nature of the TRV-HQ process, appropriate for screening-level work, where exceedingly little opportunity exists for gathering field data, but inappropriate for the level of EcoRA needed for this project.

Bird Populations (p. 129-)

As above, exceedences of safety-factor adjusted NOAELs should not as proof of adverse effects.

Overall, the focus on the Hazard Quotient approach based on NOAECs or LOAECs fails to consider the slope of the response curves for PCBs. Experimental designs for PCB studies should be more robust than they have been. However, even with the limitations of the predominantly ANOVA based studies, the NOAEC-LOAEC ranges are an order of magnitude. So a quotient of ≈ 10 for a NOAEC may still be below a LAEC. In that assessment factors of 10 were applied routinely, a quotient of ≈ 100 is still likely to be below the LOAEC. And, if a true LOAEC were determined, this still would not translate automatically to a population-level effect.

Through p.137, modeled values from HUDTOX were used to predict exposure levels. No documentation of HUDTOX was available for this review. However, if it also incorporates protective assumptions, then the quotient would be biased further so that exceedences of the quotient of $\approx 1,000$ still might have no population-level effects. Indeed, this is precisely what the various population monitoring data indicate.

EPA chose to downplay the value of field observations. The survey (pp. 137-138 and 146), which was conducted, was described as "not formally structured." The social sciences have well-established procedures to structure formal surveys. Why was the opportunity lost? The rationale offered, that the "diversity of experiences of interviewees" diminishes the value of the information should not be accepted as a legitimate excuse for dismissing critical information. Indeed, conflicting views among interviewees on some topics were selectively presented when they favored EPA's conclusions. It was not clear what was intended (page 139) by writing paragraphs attributing observations to certain individuals. Is something implied because it was Mike Brown, or Jim Brushek? What is the relevance of a professional tracker to observations, or more correctly non-observations, of birds?

What is even more interesting was the presentation on page 148. This paragraph began by praising the knowledge of professional fishers in terms of their observational skills (having previously remarked that file observations had limited value). The report then named one fisherman, provided no context for the statement made, but used the statement to refute a statement of Mr. Brushek regarding mink. So, EPA managed to use Mr. Brushek (a tracker, trapper) to refute bird experts on issues of osprey and a fisherman to refute Mr. Brushek on matters central to his expertise, mink.

Throughout this section, there were unexplained conflicting statements. For example (p.138 second full paragraph) the statement "... however studies in this area are limited" was followed (next paragraph) by "Avian wildlife are well studied along the Upper Hudson River." The statement, "The king rail was reported to be nesting, but nests haven't been confirmed" required better description of the nature of the initial report and the criteria needed for confirmation. In a subsequent paragraph, EPA used an authoritarian argument to cover apparent absence of definitive information. If additional insights were needed to understand the claims made, then those insights should have been described in sufficient detail.

The observations, reported in the first paragraph on page 139, should have been presented prominently with detailed tabulation of observations in earlier chapters. Putting these data at this point in the document as an aside, signaled that the data were not particularly important – when in fact they were the most germane data for the EcoRA. Throughout the section, it was clear that EPA took a stance that modeled data would take precedent over real observations.

On page 142, the discussion of mink tissue concentrations from the mid-1980s was legitimate. What was needed for this reassessment was a re-survey of mink now. EPA chose to report observations from a Mike Brown (page 147). If his observations are reasonable, the information in that one paragraph should carry more weight than all of the modeled exposure estimates, TRV derivation, and Hazard Quotient work undertaken in this project. Those observations would establish the important population consequences that had proper assessment endpoints been articulated would have concluded:

- a) past conditions adversely lowered populations;
- b) populations have improved with source control; and
- c) in the future, there will likely be continued improvement of populations.

On page 148, the discussion regarding modeling largemouth bass to represent sturgeon was superfluous. Solid data showing an improving sturgeon population already addressed this issue.

Collectively, this section was unnecessarily constrained to reliance on screening-level tools, which by design were biased significantly toward protection. This EcoRA deserved a much more credible treatment. The methods for sound characterization of effects at population and community levels exist. They are not prohibitively expensive when applied correctly. Nor are they fraught with large uncertainty, certainly not nearly as ambiguous as all of the uncertainties imbedded in the modeled exposures, TRVs, and Hazard Quotients. The clear answer the formal review question is that absolutely the conclusions reached here are protective of all ecological receptors. A more appropriate review question would address whether the conclusions are reasonable and useful. In light of monitoring data presented or alluded to in the report, the clear answer is that the conclusions grossly overstate the severity of the contamination. The conclusions were reached only by dismissing credible data in a most unscientific manner. And finally, if one hopes to inform decision-makers, there is nothing particularly useful to evaluate any management options. One could only define attainment criteria in terms of water or sediment chemistry, and this is without regard to any ecological considerations. Ultimately, someone will have to evaluate certain remediation options in terms of benefits (reduction of risk) realized against cost. In that no adverse population risks were demonstrated, any active removal of sediments would almost certainly have greater consequences than the contaminants. Sadly, a well-focused EcoRA would have provided the basis for such evaluations. This one falls far short.

9. *The uncertainty analysis is presented in Chapter 6 of the ERA (pp. 153-165). Have the major uncertainties in the ERA been identified? Please comment on whether the uncertainties (and their effects on conclusions) in the exposure and effects characterization are adequately described.*

Beginning with page 156 (Conceptual Model), the discussion was unnecessarily vague. A conceptual model should represent what occurs at the site. To say it is generalized and "not intended to mimic actual individuals or species" may be fine for a classroom activity pursuing a theoretical case, but it shows deficiency in the application of the process intended to address a significant site. It lacks the necessary rigor and renders the results merely hypothetical instead of contributing directly to management decisions.

A statement on page 158 "Typically no more than 10" was used as a safety factor – were larger factors applied? I interpreted the materials to be: no safety factor greater than 10 was used.

The uncertainty chapter was cast in very generalized terms. Nothing in the chapter leads to the several statements that claim uncertainty in this analysis is low. What the EPA appears to have meant is that it is highly unlikely that any problems greater than projected could occur. To this extent, the statement is correct. However, the uncertainty as to whether any of the projected problems would develop is very large – indeed refuted by the various monitoring reports. In that regard, the uncertainty of the process employed for this EcoRA is very large; it just happens to be biased in a many consistent with screening-level efforts.

The uncertainty analysis provided little documentation to support its statements. No effort was made to quantify uncertainty in the individual components of the assessment. There were no analyses of the uncertainty in the individual toxicity studies relied upon to set TRVs. There were no sensitivity analyses reported to show how the Hazard Quotients would respond to the choice of the TRV or the modeled exposure concentrations. There were no efforts to display probabilistic data to show how the assumptions in selecting a 95% UCL, would change predicted quotients. As many of the assumptions used were screening-level assumptions intended to be overly protective, there was inadequate description of how those policy-driven decisions impacted the results. But more importantly, EPA's dismissal of field data as being too erratic to rely on, presented a false description of the science of ecology. The uncertainties in monitoring data can be described fairly. Ironically, many seem to have lost sight of the connection that it was field data on the condition of ecological resources that led us to understand the effects of toxic substances. It is disingenuous to discard current ecological information that can provide demonstrative evidence of improving conditions. In this regard, one must conclude that EPA underplayed the protectiveness bias inherent in the HQ approach and overplayed the uncertainty in monitoring data and other ecological observations.

General Questions

- 1) *A goal for Superfund risk assessments is that they be clear, consistent, reasonable and transparent and adequately characterize risks to sensitive populations (e.g., threatened and endangered species). Based on your review, how adequate are the ERA and the Responsiveness Summary when measured against these criteria?*

If the goal was to be clear, then the decision to issue a response summary to a draft, but not re-write the risk documents was incongruous. As it is, one must migrate back and forth between a review draft document and a "responsiveness" report to piece together the final position EPA is making. This is inexcusable for such a high-profile project. The cost of reprinting a complete document is trivial compared to the costs already incurred in assembling the report.

The traits of clarity and consistency are challenged in the Executive Summary. I began my review by reading the executive summary to understand where the body of information in the various reports was headed. This was one of the most confusing executive summaries I recall reading. For each receptor group, there was a leading statement that suggested that overall there were no adverse effects from PCBs for that receptor group. The subsequent sentences contradicted that umbrella position by claiming that the receptor group in each of the sections for the Upper Hudson exceeded the TRV; and for the Lower Hudson the effects may be less. These statements are internally inconsistent and irreconcilable. Ultimately, what became clear is that the first statement was supported by the data, but that the modeled screening-level analysis suggested there should have been grave problems.

In response to EG8 (page 24 Responsiveness document on the SOW), EPA exposed a significant inconsistency in its policies while commenting on the rebound of fish populations following fish advisories.. The argument forwarded by EPA that fish advisories may have had a greater overall effect on abundance of various species is accurate. However, the gist of the comment ignores the most important issues posed. The comment underscores that purported effects of the contaminants are overestimated and exaggerated. The fact that fish advisories led to a rebound in populations, even with the contaminants, provides sound technical evidence that the contaminants have a minor impact on the fish population-level endpoints. The oft-stated policy of EPA is that it focuses on populations. The decisions made in this EcoRA were clearly inconsistent with EPA policy.

In terms of transparency, I would think this EcoRA would be extremely difficult to follow for most stakeholders. The data presented in companion volumes is not particularly illuminating. In a number of situations, I went to the supplemental volumes to find data referred to in the main volume, only to find that no substantive information was there. A very large quantity of trivial information was packaged into the supplemental volumes. For example, instead of a description of the communities of interest within the site, one finds a table of species that might inhabit the site. All of the information on modeling was the

purview of a different review team. Because the models were so prominent in terms of the conclusions, it would seem that much more detail was appropriate to meet a standard of transparency.

The conclusion section is not balanced. For example, the presentation of benthic community data showed differences in five sites, but the TOC normalization erased the differences. It is now interesting that in the conclusion, this very tenuous connection with PCBs was attributed as a solid line of evidence. The pattern of selectivity exercised by EPA was evident with each conclusion. Fundamentally, the analyses presented in the body of the EcoRA do not support the categorical conclusions stated in this chapter.

Failure to assess populations makes it impossible to characterize risk at the site with any sense of realism. Moreover, there is no foundation to judge any aspects of risk reduction, to select among remediation options, or to convey sound information to the public. This was described as a way to do the risk assessment without unacceptable delays and to control costs. In reality, each of the major groups of interest could have been evaluated directly in much less time and at no greater cost than this lesser effort apparently required. This is especially true, as those studies will be required if one is to use scientifically valid information to evaluate remediation options. Indeed, the total cost to correct the problems introduced by this limited effort will be much greater than had they been incorporated into the original scope of work.

In its assessment (p29) the statement was made, "The major strength of observational studies is that the receptor is examined directly and the results have a 'real world' feel. People often have greater confidence ..." It is not just 'people' in general; it is the core of science. If it is not observable (testable), it fails. Despite this statement, the assessment was designed to avert direct observations as a matter of policy. In the next paragraph, there were inaccuracies that compromised the assessment by taking it out of the realm of science. It was wrong if not disingenuous to avert field observations as having lesser importance than other approaches due to variability in natural systems. Virtually all we know about ecology is grounded in field observations. The assertion that modeled estimates have higher precision is not and cannot be supported. The concern that a receptor may be harmed by other factors is a one-sided concern and this is one that the regulator would not have to contend with if the population is doing fine. If the population of interest is doing poorly, then it is true that more proof is required. But in the case at hand, if the population parameters were nominal, then one should conclude that the claims of adverse effects predicted by models would be refuted.

The Addendum (Dec. 1999) was less of an addendum than a selective repeat of major segments of the August 1999 report. The key differences are that the modeling data was incorporated and ecological descriptions or observations were not reported. The effect was to move further from reality and made it more difficult to challenge conclusions. The extremely tenuous conclusions made in the earlier report at least had to sidestep or otherwise ignore contrary data. Here, these conclusions were posited with much

greater authority and as being factual. The fundamental reality remains – each of the target populations are reportedly doing well since source controls were implemented. The conclusions of risk can be achieved only through discounting several independent lines of data from trustees. This required discounting all direct observations that were contrary to the hypothetical adverse conditions. Thus the refinement of these risk estimates appear to be attempts to demonstrate adverse conditions into the future (when exposure levels should be declining) despite several independent observations that demonstrate that extant conditions are already better than the risk assessment predicts should be happening now.

2) *Please provide any other comments or concerns, both strengths and weaknesses, with the ERA not covered by the charge questions, above.*

There are several troubling aspects of this situation that beg for candid disclosure. Having been at the forefront of development of procedures used in EcoRA for more than a dozen years, and having performed large-scale EcoRAs, I cannot reconcile what happened here. The Hudson River PCB problem has been one of the most prominent high-profile sites in the Nation. EPA guidance going back to 1989⁷ promoted use of ecological data to characterize conditions at sites. EPA's *Framework*⁸ documents advanced many of the critical aspects of setting assessment endpoints introduced in the 1989 guide. Judged against those documents, which were highly visible and widely used across the Agency when the Hudson River reassessment began, there is no convincing explanation for the major deficiencies of this EcoRA. The several reports here emphasized compliance with the newest EPA guidance with the eight-step process. This is despite the reality that most of the work had been completed prior to issuance of this 1998 document. Moreover, the 1998 Guidance emphasizes use of field observations. Clearly, the practices followed here never got beyond a preliminary assessment typical of a screening-level effort. In that the site had already been examined in some detail, one could easily have skipped the screening-level exercises and moved toward definitive analyses.

Relying on policy, EPA decided to rely solely on toxicity endpoints and "individual risk" in lieu of population-level metrics. This was consistent with the bottom-up policy. However, the position as described suffers from being technically false. The consequence is that the EcoRA is based on untested concatenated hypothetical situations. This is despite the technical feasibility to test many of the assumptions directly. Failing to test what is testable should not be hidden behind policy. Proposing things that are not testable is counter to the foundations of science. The response is technically weak. Indeed the acknowledged limitations of the TRV process in this response would seem to have laid the foundation for greater reliance on field measurements of populations.

⁷ W. Warren-Hicks, B. Parkhurst, & S. Baker, Jr. (eds.). *Ecological assessment of hazardous waste sites*. EPA/600/3-89/013. U.S. Environmental Protection Agency, Environmental Research Laboratory, Corvallis, OR.

⁸ US EPA. 1992. *Framework for Ecological Risk Assessment*. Risk Assessment Forum. EPA/630/R-92/001.

Perhaps the most troubling of all the concerns about this EcoRA has been the attitude portrayed in the responsiveness documents. Collectively, the responses to legitimate technical issues were dismissed by citing policies. Each of the legitimate questions posed during the comment periods that asked for clarification of assumptions of models or to rectify predictions of risk with contrary observations on the conditions of the resource were rejected. The consequence is the stance taken by EPA was to diminish the quality of the EcoRA. Had the legitimacy of the technical questions been acknowledged, there would have been ample time to address them properly. If this current review process is a serious one, EPA must revisit its position to preempt scientifically sound data by imposing policy.

Detailed Comments:

The following statements contain additional observations and questions raised in reviewing the various reports that were not covered in my responses to the nine specific charge questions and the two general charge questions. These appear in sequence of appearance and are attributed to the specific reports.

Responsiveness to Scope of Work – Sep. 98

p. 13. The response to EP-3. Not making comparisons to other sites may have legal justifications, but it is nonsensical from a scientific or technical perspective.

p. 13. The response to EG-1. The response is policy driven and is unsupported by science. By ignoring field information at the start of the assessment, there is no context for basing the substantial effort of the study. Indeed early field observations provide the most focused work and substantial lower costs. The same goes for the response to EG-5 on page 15. The policy is without foundation if one is conducting a technical assessment. It may well be a choice of managers to then move in these directions, but it is inappropriate to respond to technical questions by invoking policy. The questions remain unanswered.

The third part (bottom of p. 15) averts direct analysis of populations or communities, relying instead on key biological receptors. However, the "key" receptors were not selected through a rigorous process. Justification for the selection is documented poorly.

p. 17. Here and in several places the odd redundant combination "potential risk" is used. Risk implies a potential expressed in qualitative or quantitative terms for some adverse consequence to occur in the future.

p. 18. Response to EG-7. The policy line is repeated.

p.20. Response to EG-12. The declaration of the choice of largemouth bass over smallmouth bass begs for justification of this selection.

p.25. The assertion that assimilation rate, metabolic efficiency, and the structure of PCB congeners ingested "is beyond the scope of the EPA SOW" underscores the reason field data on tissue residues and populations would be more important in process than modeling alone.

p.26. The assumption of using "location" data instead of looking at average areas fails to account for the long time for equilibrium to establish in tissues (body burden) and the magnitude of movement of fish diurnally and seasonally within the River system.

p.31. Response to EN13 cites policy that is ignored in the reply to EG-8 on page 24.

The response to EG-19 again relies on policy to trump the legitimate technical concern. The policy fails to in that it is counter to science. The list of rationalization points provided in response to Eg-2 and EC-3 on p. 32 underscores the weakness of the policy. EPA appears to have consistently hidden behind a policy to cover serious deficiencies of the modeling approaches and assumptions it used. Moreover, it used these policies to side-step direct field data that would answer the question posed.

Phase 2E – Aug. 99

p.17. It is interesting that aquatic plant uptake of PCBs was considered in as much as uptake through roots of terrestrial plants is virtually non-existent and in general relative to terrestrial plants, aquatic plants are less dependent on root uptake.

p.18. It is not clear why terrestrial exposure was discussed at all as it was not part of the SOW,

p. 76. The statement "The TEQ/TF provides a toxicity measurement for all AhR-binders" is not accurate. The method is not a measurement, it is merely an estimator.

p.78. The explanation about differences between terrestrial and aquatic animals pertaining to dose and concentration is not accurate. Fish eating fish eat the entire animal. What is different is that some portion of exposure comes directly from adsorption/absorption across gill tissue.

p. 78-79. To do the TRVs, all of the data (from the initial toxicity study plus the environmental sample analysis must report concentrations for each congener. In that these conditions are seldom met, there is great uncertainty introduced in these derivations. This uncertainty was largely ignored in the EcoRA.

p.80. The first bullet regarding toxicity tests of other species assumes that the target species is more sensitive than the test species. There is no basis presented for this policy decision for an assessment factor. It automatically creates an impression of adverse conditions and is a major reason for the use of field observations to document population-level effects.

Also, there is no basis for relying on the human health default policies regarding RfDs. The processes are fundamentally different in part because HHRA focuses on the health of individuals whereas an EcoRA is purported to focus on sustainable populations.

p.94. It seems strange to adjust an eight-week study by a factor of ten when the endpoints measured were reproductive endpoints.

p.96. McCarty and Secord (1999) reported field data from three locations along the Upper Hudson River during 1994 and 1995 field seasons evaluating various reproductive endpoints. They compared results to reference areas (Ithaca and Lake Champlain) as well as other published results on tree swallow reproduction. Several important reproductive endpoints did not differ significantly among sites or between the reference areas and the assessment sites. The authors suggested that nest abandonment was a strong indicator of an adverse effect from PCBs. Though there was an apparent increase in abandonment and eggs failing to hatch between references and assessment areas, there was an unexplained relationship that the relationship among assessment sites was inverse; that is as PCB concentration rose there was less hatching failure and fewer eggs abandoned. Other critical reproductive endpoints such as growth of nestlings, return of adults in the second year, and such were not significantly different. Variance between years was much greater than the differences attributed to PCB concentration. On the whole, it appears that some interesting observations were made. There is a clear indication that exposure to PCBs is occurring, but there is not strong evidence that populations-level effects are being manifest at the site. EPA repeatedly holds out this study as an indication that population-level effects were demonstrated, but a fair reading of the data indicates that the claims should be tempered considerably.

p. 98. It is wrong to conclude that interperitoneal injections simulate oral exposure because the material is absorbed by the liver. What is missed in this oversimplification is the portion of contaminant taken orally that passes through the feces unabsorbed.

p. 118. It is not clear why there were five sampling stations. Also there were no selection criteria provided. It may have been a compromise imposed by cost, access, safety, or some other factors; but no explanation was provided.

p. 119. In reality, each of the diversity indices is highly correlated with the others. Any will provide some comparative base to look at community composition, although there is limited useful information regarding stressor effects that can be deduced from diversity indices.

p. 121-122. It seems as if the NOAA SEC should have been introduced in Chapter 3 not here.

Hudson River Ecological Risk Assessment
Draft Review Comments – L. A. Kapustka

Responsiveness Report – Mar. 00

p.16. Response to Eg.1.1. The policy on "bottom-up" approaches should not be cited as justification for technical limitations. It should remain as part of the risk management package. By asserting policy, EPA effectively dismisses legitimate challenges in interpretation. It is particularly disturbing, because this is not an either/or situation – both approaches could be handled nicely in the EcoRA (without incurring delays or adding costs in the long run). In this case, field data would likely demonstrate minimal population impacts from the contaminants, would show the large uncertainty (or highly protective assumptions used) in the TRV, HQ approach, and require re-evaluation of conclusions of risk. Though a diminished population would not necessarily equate to causality being assigned to the contaminant, a nominal population would demonstrate that adverse effects were not occurring. Adhering to the policy ignores the very important conclusions that might have been reached.

p. 24-25. EF-1.4 and EP-2.1. The argument that habitat mapping was not feasible given the large size of the site fails a test of reasonableness. A key feature of an EcoRA is Ecology! To organisms, habitat is everything. The largest factor in calculating risk to fish or wildlife is exposure; exposure is determined by habitat first and bioavailability second. Ultimately, the results of the EcoRA are to be considered in light of remediation options. By failing to consider habitat, the EcoRA becomes largely irrelevant.

p.27. Response to EG-1.14. (repeated in response to EL-1.8 and EL-1.10) EPA justified its estimates of TEQ on the basis of not being over-estimated by more than a factor of 2. However, the projected decline of PCB levels by a factor of 2 drops some HQs to 1 or below. The concluding sentence further *justifies* the approach by claiming that "calculated risk levels exceed acceptable levels by orders of magnitude." It is fundamentally incorrect to argue that an HQ of <1 is "the acceptable level of risk at a site." The HQ of a toxicity endpoint says nothing about the population-level effect, especially when the quotient was based on a NOAEC. Moreover, what is "acceptable" is defined by stakeholders in the broad sense and may have little relationship to a toxicity quotient.

p.31. Response to EF-1.17. Again EPA incorrectly equates HQ with risk. This is not valid, the HQ \neq risk; rather it is a signal that effects may be occurring. The procedure requires evaluation of exposure in terms of habitat use, bioavailability, and relationships among toxicity endpoints and ecological effects (at the population-level).

p.35. Response to EL-1.17. EPA's response fails to take into account the numerous tributaries flowing into the Hudson as well as nearby wetlands and lakes that would be used by birds. Though the site is large, bird use does not conform to the site boundaries. Accordingly the estimated risks to birds overestimate the conditions.

p.36 and 37. Same as above.

10 May 2000

p. 62. Response to EP-2.4. The assertion that there was insufficient toxicological data to conduct a probabilistic effects assessment is curious. Ranges of values were discussed in the reviews discussed in the TRV sections. Such data could have been used to describe the response relationships instead of electing to use only NOAEC or LOAEC values. A probabilistic approach also could have incorporated variability in measured exposure parameters. EPA elected not to do a probabilistic study; the decision had little to do with how robust the data set was.

p. 62. Response to EG-1.27 By acknowledging that an order of magnitude error was acceptable, it would have been more truthful to acknowledge in the uncertainty sections that a HQ of 10 or more would suffice as the warning flag for a screening level assessment which would trigger a more detailed risk characterization.

p.75. Response to EL-1.41. The argument that ecological samples were biased toward samples containing invertebrates is not a credible explanation for differences in the modeled output. That explanation would work partially if PCB levels were so high as to kill all invertebrates. As a basic premise of science, measured observations always take precedent over hypothetical expectations. Models cannot be validated; they can be calibrated – but ultimately, they still generate hypothetical expectations. Whenever real data exists, it should displace all modeled projections. The implication of modeled values being superior to measured data further indicates that the predicted exposures in the food chain models magnified these errors. Therefore, real world conditions are less adverse than estimated in this risk characterization.

p.84. EG-1.9. Again EPA posits that important endpoints such as “reduced fecundity, decreased hatching success, and similar kinds of reproductive impairment” are “difficult to observe in the field” is not supported scientifically. These endpoints were identified historically as being important because they explained observed changes in populations in the field. EPA in effect elected to ignore relevant ecological data when the data did not conform to the hypothesized situations. Science practice requires the reverse action – that is, reject the modeled or hypothesized conditions in favor of observations.

p.89. E.G. 1.36. EPA argues that one eagle plasma sample and one eagle fat sample “are high enough for concern” but dismisses bald eagle breeding data from 1992 to 1999 that illustrates a trend toward successful reproduction with fledglings of 1, 4, and 5 from 1997, 1998, and 1999. EPA’s stance is consistent in that ecologically relevant data was again given less credence than direct measures.

p. 96. Response to E.P-2.9. Acknowledging that salmonids are the most sensitive species to dioxin-like compounds, EPA nevertheless uses this value as the TRV to apply to non-salmonid species. If it is appropriate to use an assessment factor to account for unknown interspecies sensitivity, then by the same logic, one should use a fractional assessment factor to adjust for species known to be less

sensitive. The use of the salmonid value virtually assures that the resulting H.Q. would be overly protective of non-salmonids (as reflected by the population data for fish in the system).

p. 101. Response to EL-1.46. This response by EPA asserts its policy over relevant ecological data. This signals that principles of science are not to be considered if they are inconvenient.

p. 101-102. Response to EF-1.64. "Although all the Thompson Island pools had viable benthic macro-invertebrate communities that could support local fish populations, the PCB concentrations ... indicate that some benthic species may be adversely affected." This statement clearly ignores the thrust of EPA guidance that focuses on population level effects to the assessment endpoint species. It signals further a disregard for the process, after repeatedly citing policy, and process to justify other actions.

p.102. Response to EG-1.34. EPA argues that "The gradient of PCB concentration along the 200 mile river ... increases the difficulty of ascribing particular effects to PCBs" is patently contrary to principles of ecology. Indeed, gradients provide the most powerful tool for assigning causality. Gradient analysis is at the heart of ecology. That PCB concentrations do not correlate with population responses; that other factors (e.g., fishing ban or improved water quality) are reflected in the improving conditions, underscores the limited adverse effects of PCBs on the populations.

p. 103. Response to E.G. 1.37. Whether or not duck meat is considered safe for human consumption is not a concern for the EcoRA. That is solely a concern for HHRA.

Section III. Revisions.

p.1. "...Revisions do not change the conclusions of the August 1993 (presumably 1999) EcoRA for any receptors of concern" appears to have been an *a priori* decision rather than a serious consideration of the comments. Proper attention to several of the concerns raised should have resulted in substantial modification of the presentation and the conclusions of risk.

p. 3. "...considered to be a field study ..." If a study is done in the lab (even with field collected samples), then it is a lab study. This explanation is a poor example of communication.

p.4. The discussion of the Hazelton and Prouty (1980) study points to the problems that occur from relying on a single study, which was conducted using a woefully inadequate study design. There is no basis for accepting an unbounded LOAEL or an unbounded NOAEL (except in limit studies). Even more troubling is the application of "extrapolation factors to such toxicity parameters.

p.4. The extensive discussion on bald eagle data seems to be reaching. It is doubtful that truly significant differences in reproductive endpoints existed between mean concentrations of 5.5 and 8.7 mg/kg given all the uncertainties associated with analytical detection. This seems like a lot of effort to

change a value from 3.0 to 5.5, especially knowing that the assumptions imbedded in the exposure estimates overwhelm these minor changes.

p.5. The uncertainties of EPA's approach to setting TRVs is illustrated in the laboratory based NOAEL and LOAEL (0.02 and 0.01; presumably reversed) and the new field NOAEL of 0.214. This indicates that for these studies the lab values substantially overestimated the hazard (by more than an order of magnitude). If one were to apply the 10x assessment factor to the NOAEL to get at the earliest threshold response of ecological relevance, then the lab value would be 200x lower than appropriate.

There is no scientifically accepted practice for using NOAELs or LOAELs. This is solely a policy-driven precedent.

The entire revision undertaken seems to have hinged on adjusting models to make minor changes in predicted exposure concentrations (on the whole, the adjustments provided higher "exposure" estimates) and to tinker with TRVs to produce relatively insignificant changes. The overall effect was to predict slightly longer periods of "unacceptable risks."

Recommendations

Based on your review of the information provided, please select your overall recommendation for the ERA and explain why.

5. *Acceptable as is*
6. *Acceptable with minor revision (as indicated)*
7. *Acceptable with major revision (as outlined)*
8. *Not acceptable (under any circumstance).*

For the purposes of this reassessment effort, I must conclude that this EcoRA is Not Acceptable. The effort was unnecessarily constrained to a screening-level assessment. Elegant chemical analyses were performed to characterize sediments and water (and to a lesser extent biota) along the River. However, the decision to ignore ecological data, to forego opportunities for analyses of populations of interest, and to reject population trend data are fatal errors of omission which require this recommendation.

Sean Kennedy

Problem Formulation/Conceptual Model

- 1) As a general description of exposure pathways, and as a description of which ecological receptors are potentially exposed to PCBs, pages 11-19 appear adequate. However (as indicated with examples in my answers to question 2), this section does not provide sufficient rationale to indicate why certain fish and bird species were *not* selected as receptors for this Risk Assessment.

Assessment and Measurement Endpoints

- 2) This section, along with Table 2-7 indicates the selected assessment endpoints. It was certainly appropriate to select a broad range of taxa and species with different exposure pathways. However, it is not explained why certain species were *not* selected. For example, Tables 2-4 indicates that snapping turtles are "potentially" found along the Hudson River. They would have been a useful additional receptor because the work by Bishop *et al.* in the Great Lakes (1991, 1998) could probably have been used to develop a field-relevant NOAEL. Similarly, Table 2-5 indicates that the Double-crested Cormorant and Osprey are "breeding birds of the Hudson River". If Cormorants and Osprey had been selected as receptors, then data from several studies in the Great Lakes and other locations could have been used to establish field-relevant NOAELs (p. 138 of the Risk Assessment indicates that Everett Nack has observed, "... small numbers of osprey following the herring runs"). Common mergansers are also indicated as "breeding birds of the Hudson River" (Table 2-5), and Mark Brown of the New York State Department of Environmental Conservation has reported seeing this species of diving duck along the Hudson River (p. 139). Why were they not included? I am also curious to know why none of the species of fish that have been studied for their relative sensitivity to PCBs and dioxins (e.g., Elonen *et al.*, 1988) were included. I would not expect a complete listing of why *all* species were not included, but it seems to me that the Risk Assessment should include statements on why certain obvious (at least to me) 'candidate' receptors were not selected.
- 3) The combination of measured, modeled, guideline and observational measurement endpoints is supportive of the "weight of evidence" approach used for US EPA ERAs, but I do not know how the phrase "weight of evidence" is used officially. In my experience at other locations of concern in the United States, I have not seen the phrase particularly well defined. Perhaps there should be discussion of its meaning of at the Peer Review meeting.

Exposure Assessment

- 4) My expertise is not in the area of model development and validation, but in the area of toxicological and biochemical effects of PCBs and dioxins on birds and fish. However, I reviewed chapter 3 carefully, and I have the following comments:
 - The Risk Assessment recognizes that there are changes in PCB patterns as they

move up the food chain. However, it is not clear to me if this fact is taken into account when estimating TEQ concentrations in birds from PCB concentrations in their diets. Would one expect bioconcentration of the dioxin-like PCBs, and if so, what effect would this have on TEQ estimates?

- BZ#126 was below the detection limit in many of the samples, and its detection limit was used for TEQ calculations for these samples. It is stated in the Risk Assessment (p. 40) that, "The exact magnitude of the error introduced by the omission of BZ#81 and setting BZ#126 equal to the detection level is not known, but is likely within an order of magnitude at most". As far as I can see, no justification for this conclusion is made. The Risk Assessment should show the rationale to this conclusion. I view this as an important point because if BZ#126 were indeed 10-fold lower in some samples (e.g., dietary dose for mallards in Thompson Island Pool; fish in several locations), then the TEQ-based hazard quotients would become 10-fold lower, thus affecting final conclusions regarding likely risk. This problem, if taken into consideration with possibly unreasonable over-estimates of NOAELs from laboratory-based bird studies (see below), needs to be considered for establishment of appropriate and un-biased final conclusions. Because BZ#81 has a low TEF in fish, and because it is usually present in the environment at very low concentrations, I would imagine that it would have little influence on the TEQ concentrations in fish.
- It is assumed that the diet of bald eagles is 100% fish from the Hudson River. I would be surprised if the year-round diet is 100% fish. In other locations, bald eagle diet is not 100% fish; small mammals and birds are included.
- Were there no other data on PCB concentrations in avian eggs from the Hudson River (other than tree swallows and one mallard egg) that could have been used for the Risk Assessment? Such data would not, in themselves, allow for definitive conclusions, but they would have strengthened the quality of the assessment. Why were other eagle blood/egg data from eagles that spend only part of each year feeding on fish in the Hudson River not included (p. 33 implies that there are data in a 1999 paper by Nye) ?
- Comments by General Electric suggest that there are considerable residue data in fish in the Hudson River that were not used for the Risk Assessment. Is this true? I have no idea how such data would affect conclusions of the Risk Assessment, but I wonder why they were not included if they indeed exist. Regardless of whether the models for predicting fish concentrations are adequate or not, an explanation for not including all available residue data in the receptors chosen should be made clear in the Risk Assessment.

5) I made a few comments regarding my concerns with how exposure was estimated under question 4, above. As indicated in my opening sentence that question, I am not an expert in model development and application. However, it is certainly obvious that many

assumptions are made, and it might be better to express exposure in terms of likely ranges of exposure rather than absolute amounts. If done in this manner, one would be able to use exposure estimates along with estimated ranges of hazard quotients to help provide a better assessment of impacts of PCBs to biota in and along the Hudson River

Effects Assessment

- 6) The first part of this question asks for comments on the validity of using *only* NOAEL TRVs from field-based toxicity studies. In my opinion, this approach is appropriate because, as the authors correctly state, there is the potential of exposure to contaminants other than PCBs which makes it difficult (and often impossible) to establish reliable LOAEL-based TRVs. However, I do not know if the use of NOAELs and LOAELs is the *only* requirement for assessing risk in EPA risk assessments. Can/should EPA Risk Assessments also include studies to look for site-specific evidence of demonstrable effects in fish and wildlife?

Unfortunately, there are practical limitations associated with using field-based studies for establishing NOAEL TRVs for PCBs and TEQs because there are usually very few, or no, data for the species selected for a particular risk assessment. This is certainly a problem for the Hudson River Risk Assessment. The authors located only one study (Weimeyer *et al.*, 1993) for the bald eagle, and the studies by Secord and McCarty (1997; paper by McCarty and Secord, *in press*) were the only papers used for the tree swallow. There were no field PCB NOAEL TRVs for other species of birds. Laboratory-based PCB NOAEL TRVs were derived for only one-half of the species of fish selected for the risk assessment, and there were no field TEQ NOAELs for fish. Because there are so few data, one must be cautious when interpreting hazard quotients.

It should be noted that at least two field studies on bald eagles were not used for the Risk Assessment. A paper by Donaldson *et al.* (1999) suggests that the PCB NOAEL TRV for bald eagles might be closer to 20-30 mg/kg egg rather than 3 mg/kg egg (revised to 5.5 mg/kg egg in the Responsiveness Summary, March, 2000). A paper by Elliott *et al.* (1996) on bald eagles in British Columbia suggests that a mean TEQ concentration of 0.3 ug/kg egg (using hatching success as the endpoint) was the NOAEL for embryotoxicity of the mixture of PCDDs, PCDFs and PCBs found in these bald eagles. This is 30-fold higher than the NOAEL used in the Hudson River Risk Assessment. I am not familiar enough with the literature on fish field-based studies to know which studies might not have been included.

The second part of this question asks for comments on whether the general approach of using uncertainty factors (interpecies, LOAEL-to-NOAEL, and subchronic-to-chronic) is protective of Hudson River receptor species. The approach is certainly protective in most cases; in fact, there are situations where the TRVs are likely to be unrealistically low. For example, with the exception of the Great Blue Heron, all laboratory-based TRVs for PCB NOAELs and LOAELs were derived from a study with chickens (Scott, 1977). The authors of the Risk Assessment recognize that the chicken is the most sensitive species to

PCBs and dioxin-like PCB congeners, yet they simply use the chicken study to calculate hazard quotients. Although perhaps unconventional, would it not make some sense to **multiply** chicken TRVs by 10 (or more) to get more reasonable TRVs based, in part, on the findings of Brunstrom *et al.*, Sanderson *et al.*, Hoffman *et al.*, Powell *et al.*, Peterson *et al.*, Kennedy *et al.* and other investigators that show that **all** species are less sensitive to PCBs than chickens. This alternative approach could certainly be done with some confidence with the mallard in my opinion, because Brunstrom's egg injection studies showed that the mallard is approximately 10-50 times less sensitive to the lethal effect of PCB 77 than the chicken.

The Risk Assessment indicates that the NOAEL for all species of birds is 0.33 mg/kg egg. This conclusion is based upon the paper by Scott (1977). In my opinion, it is obvious that a PCB concentration of 0.33 mg/kg egg is unlikely to cause problems with growth, development and reproduction in wild birds. Otherwise, all birds in North America would still be at risk from PCBs. This concentration is approximately the background level in many un-contaminated areas of North America (e.g., the Bay of Fundy, which has been used as a reference site for herring gulls for many studies carried out by the Canadian Wildlife Service). Similarly, a TEQ concentration of 0.01 ug/kg egg (derived from the paper by Powell *et al.*, 1996a) is unlikely to be toxic in wild birds. This is approximately the background TEQ concentration in bird eggs in many areas of North America and elsewhere.

In summary, my opinion is that the laboratory-derived PCB- and TEQ-NOAEL TRVs for wild birds (with the exception of the ring-necked pheasant, which was not a receptor for this Risk Assessment) are unrealistically low, thus making the hazard quotients unrealistically low. Similarly if the LOAELs were truly applicable to wild birds, then one would come to the conclusion that **ALL** birds in most areas of North America are being affected. This conclusion is simply not supported by evidence of sustainable (and often growing) populations of many species exposed to PCB concentrations of 2.2 mg/kg egg and/or 0.02 ug/kg egg TEQ. There are similar problems with several of the fish TRVs. Because the authors found no laboratory data on several species, results from lake trout were used. Lake trout are well known to be one of the most sensitive species; thus the TRVs for some of the species of fish selected for the Hudson River might be too low. Alternatively, some of the receptor species of fish might have similar sensitivities, but we simply do not know the answer to this question due to the paucity of laboratory studies and site-specific studies to determine population changes.

- 7) As indicated in my response to question number 6, it is my opinion that the toxicity quotients calculated using both total PCBs and TEQs are likely to be protective of the receptors. However, my concern is that there the toxicity quotients derived from laboratory studies for birds and fish may be unrealistically high because chicken and lake trout data were used in several cases.
- 8) This section appears to adequately characterize the relative risks to the receptors selected, if one accepts the exposure estimates and NOEALs presented earlier in the report.

However, I think that the meaning of some of the wording (e.g., "suggest the potential for population-level adverse reproductive effects") is difficult to interpret, in part due to my concerns outlined above regarding exposure estimates and highly conservative application of chicken and lake trout based NOAELs. What are the criteria for making this statement?

As I write, I do not know if the evidence from bird "Observational Studies" (pp. 137-139) is, indeed, comprehensive. Note that Everett Nack (p. 138) has seen osprey, but Jim Brushek has not (p. 139). Were more systematic studies not conducted by the US Fish and Wildlife Service and/or other agencies?

- 9) The uncertainty analysis is written in terms that are too general, in my opinion. This section would be substantially improved if the uncertainties were clearly identified, and applied to the actual and modeled data to show the range of uncertainty for the hazard quotients.

General Questions

- 1) Overall, it appears to me that the authors of the Risk Assessment have attempted to write a clear, consistent, reasonable and transparent report. However, I think that the conclusions and Executive Summary need to be modified such that the uncertainties inherent to the TRVs and hazard quotients are presented in a manner that is much easier to interpret. Could this be done by showing ranges of hazard quotients for the different receptors (or, perhaps by more sophisticated manners that I presume have been developed and used at other sites)?
- 2) The major weaknesses of the Risk Assessment include (i.) the limited amount of site-specific data on exposure of potentially vulnerable organisms to PCBs, (ii.) limited, to apparently non-existent, documentation of changes to fish and wildlife populations that might have occurred due to PCB exposure, (iii) limited attempts to document pathological and physiological effects of chosen receptors, and (iv.) explanations why certain receptors were not studied at all.

Recommendations

I do not view the Risk Assessment as "unacceptable". It appears to be "acceptable", but I do not know the criteria for deciding between "acceptable with *minor* revisions" and "acceptable with *major* revisions. Certainly, I think changes are required to address the points I address above. I will wait until the Peer Review meeting before I provide my final recommendation.

General Comment: I do not understand why there were so few field studies to assess the effects of PCBs on fish and wildlife during the past nine years. The Hudson River is one of the most PCB-contaminated rivers in North America. Data from field studies would have been extremely useful for the Hudson River, and other risk assessments in PCB-contaminated sites in the United States, and globally.

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Biography - Dwayne Moore

Dr. Dwayne Moore has a B.Sc. in Biology from the University of Western Ontario, and a M.Sc. and Ph.D. in wetland community ecology from the University of Ottawa. After graduating, he worked for six years at Environment Canada, the first two years developing environmental quality guidelines for industrial chemicals, and the last four years conducting ecological risk assessments for priority substances. He has been a Senior Associate with the Cadmus Group for the last four years.

Dr. Moore has considerable expertise in ecological risk assessment, the development of environmental quality guidelines and criteria, community ecology, multivariate statistics, uncertainty analysis, and analysis of toxicity data. Since joining the Cadmus Group in May, 1996, Dr. Moore has managed over 40 projects for Canadian, U.S. and international clients in government and industry and participated in many others. Dr. Moore has led projects to assess the ecological risks of a variety of chemicals including hexachlorobenzene, chloroform, chlorinated wastewater effluents, waste crankcase oils, phenol, mercury, PCBs, and hexachlorobutadiene. Dr. Moore has also been involved in the Environment Canada probabilistic risk assessments of ammonia and chloramines. He led the effort to update and considerably expand Environment Canada's guidelines for the conduct of ecological risk assessments of priority substances under the *Canadian Environmental Protection Act*. Recently, Dr. Moore authored the chapter on probabilistic risk assessment in *Ecological Risk Assessment and Prioritization Process* for the Department of Energy (DOE). The chapter includes state-of-the-art statistical and modeling techniques for use in higher tier assessments including: first and second order Monte Carlo analysis, variance propagation, probability bounds analysis, fuzzy arithmetic, interval analysis and cost-benefits analysis. To illustrate these and other techniques, Dr. Moore prepared a case study that estimated the effects of methylmercury and PCBs to mink and kingfishers at a CERCLA/RCRA site near Oak Ridge, Tennessee and compared these effects to the costs and benefits of several remediation alternatives. Dr. Moore is currently involved in projects to prepare guidance, training, and case studies for probabilistic risk assessments for several agencies including the CMA, CEFIC, and the U.S. EPA Office of Pesticide Products. He also is conducting a detailed evaluation of a large spatially-explicit population model (PATCH) for the U.S. EPA Office of Research and Development, and is leading the development of ambient water quality criteria for mercury for the Water Environment Research Foundation. Dr. Moore recently co-chaired the Society of Environmental Toxicology and Chemistry (SETAC) Pellston conference on the use of uncertainty analysis in ecological risk assessment and co-edited the book that followed from the conference. He is currently serving on the SETAC Pellston steering committee for Probabilistic Risk Assessments of Pesticides, and has served on a past steering committee to develop an ecological risk assessment decision support system. Dr. Moore has participated in several other Pellston workshops (e.g., assessing multiple stressors, re-evaluation of environmental quality criteria), and has participated in numerous EPA Science Advisory Panels and other EPA peer review workshops. He is a charter member of the SETAC Ecological Risk Assessment Advisory Group. Dr. Moore has been a member of the editorial board for *Human and Ecological Risk Assessment* journal since its inception and is a member of the editorial board for *Environmental Toxicology and Chemistry*.

Hudson River PCBs Baseline Ecological Risk Assessment

- Peer Review Comments From Dwayne Moore -

In the charge to peer reviewers, reviewers are asked to determine whether the baseline ecological risk assessment (ERA) "is technically adequate, competently performed, properly documented, satisfies established quality requirements, and yields scientifically valid and credible conclusions." If the Phase 2 Hudson River ERA had been intended as a screening level assessment, I would answer yes to the question, although I have concerns about the lack of documentation on the modeling exercises and the uncertainty analysis briefly referred to in section 6.5 of the baseline ERA. My understanding, however, is that the baseline ERA was intended to be a higher tier ERA that could be used "to back-calculate to appropriate levels of PCBs in fish and to compare various remedial alternatives, including the No Action alternative ... required by federal Superfund law." As a higher tier ERA, the Hudson River PCBs baseline ERA is lacking, primarily because it relied on highly conservative and deterministic quotients as quantitative indicators of risk. In addition, no information was provided on what remedial alternatives were being considered, what risk reductions they would provide, and what countervailing risks they would introduce. As outlined in a recent publication (Moore et al. 1999a), I believe that risk management decisions should not rely on toxicity quotients because:

"(1) quotients do not quantify, or even acknowledge, the uncertainties inherent in ecological risk assessment, (2) the degree of conservatism of quotient-based risk estimates are unknown, (3) the appropriate place for applying conservatism is during the risk management stage (i.e., the stage at which societal interests are normally considered), and (4) quotients do not provide the basis for estimating the likelihood that a desired level of risk reduction will be achieved for any given remedial action alternative ... At best, conservative quotients can be used to screen out negligible risk scenarios, but otherwise provide little useful information to the risk manager or public. At worst, the use of conservative quotients results in situations where low level risks are subjected to costly mitigation measures."

In my opinion, a higher tier ERA should answer the following questions (see Kaplan and Garrick 1981):

- What can happen (i.e., what can go wrong)?
- How likely is it that it will happen?
- If it does happen, what are the consequences?

The toxicity quotients relied on in the Hudson River PCBs assessment were useful in answering the first question, but provided next to no information on the probabilities of effects of differing magnitudes, and the consequences of those effects to Hudson River populations and communities, if they were to occur. The tools exist to conduct probabilistic risk assessments (Landis et al. 1998; Warren-Hicks and Moore 1999), case studies have been published (e.g., Dakins et al. 1994; Moore et al. 1999a,b; Sample and Suter 1999) and guidance is available on how to use these tools in Superfund ERAs and other EPA programs (U.S. EPA 1997, 1999). Similarly, sophisticated ecological modeling tools are available for prospective risk assessments of effects to populations and communities (e.g., Caswell 1989; Bartell et al. 1992; Jorgensen et al. 1996; RAMAS software tools) and have been used in the past to support environmental decision making with, for example, striped bass populations (e.g., Barnhouse et al. 1990). Given the uncertainties and complexity of the Hudson River assessment and the economic costs that could arise as a result of this assessment, I am disappointed that readily available higher tier tools which would substantially improve our understanding of risks were not employed in the baseline Hudson River ERA.

Problem Formulation

- (1) For the most part, the conceptual model adequately describes the different exposure pathways by which ecological receptors could be exposed to PCBs in the Hudson River. I would have used the problem formulation and the results of the Phase I risk assessment to have reduced the scope of the baseline ERA to consider only the most important exposure pathways for the species and communities at highest risk. Because PCBs are persistent and bioaccumulative, it seems likely that piscivorous fish, birds and mammals are receiving the highest exposures, particularly long-lived species with small home ranges that forage exclusively in or near the Hudson River. For these species, food web exposure is really the only important exposure pathway. Thus, I would have used the problem formulation exercise to eliminate the need to consider dermal, air and water pathways of exposure. Had

this been done early on, a greater proportion of the monitoring effort could have been targeted to prey species that are important components of piscivore diets.

Although floodplain soils are not likely a major exposure pathway for piscivores, I do not understand why they are "beyond the scope of the assessment." It would seem that some of the assessment endpoints chosen for the Hudson River PCBs ERA (e.g., raccoons, brown bats, tree swallows) receive a significant proportion of their exposure via terrestrial sources that are in contact with floodplain soils. Perhaps more importantly, floodplain soils may act as a long-term, continuous source of PCBs to the Hudson River through leaching, erosion, resuspension during flooding, etc. If true, remedial decision making ought to account for this source. No information was provided to determine whether floodplain soils are a major source of PCBs to the river or to terrestrial biota.

- (2) I believe the list of assessment endpoints could have been shorter by focussing only on high risk, piscivorous species. This would have eliminated the need to consider benthic invertebrates, forage fish and insectivorous birds and mammals. I also would not have chosen species that spend a significant portion of their time foraging outside the Hudson River area (e.g., bald eagles), because this behaviour will likely reduce their overall exposure. With a reduced set of assessment endpoints, it would have been possible to consider use of population models, uncertainty propagation techniques, etc to better understand risks and consequences of possible remedial actions for the high risk species.

The assessment endpoint entitled "Benthic community structure as a food source for local fish and wildlife" strikes me as a curious choice. The approach for this endpoint was to examine benthic community structure and to compare water and sediment levels to generic water and sediment quality criteria. None of these approaches makes any attempt to assess how risks to benthic species could be transmitted to "local fish and wildlife." Based on the analyses that actually took place, this assessment endpoint should be re-labelled to "protection and maintenance of local benthic invertebrate communities" to reflect the analyses done and to be consistent with other endpoint descriptions (e.g., local fish populations, local insectivorous birds, etc).

Although I understand their importance, a separate assessment endpoint for protection of threatened and endangered species is not required. Previously stated assessment endpoints for maintenance and protection of fish, birds and mammals overlap this endpoint. Further, the approaches taken to assess effects to fish, birds and mammals are aimed at estimating risks to individuals, the level of organization which is usually the focus for threatened and endangered species. Finally, the measures of effects and exposure for threatened and endangered species are not specific to these species (e.g., shortnose sturgeon, bald eagle), and completely overlap approaches for previously selected species. No new studies or analyses were conducted aimed specifically at improving our understanding of risks to threatened and endangered species. Thus, the ERA appears to be doing the "right thing" by focussing on threatened and endangered species, but the reality is that we have gained no further understanding of risks to these species by including this assessment endpoint in the ERA.

The assessment endpoint for protection of significant habitats is meaningless. The concern for PCBs is with maintenance and protection of biota, which was adequately addressed with the other stated assessment endpoints. It is difficult to imagine how PCBs could affect habitat in any other way (e.g., increased habitat fragmentation, alteration of physical characteristics of habitat, etc). My point is reinforced by the fact that the assessors (again) chose some of the same measures of exposure and effect as were used for the assessment endpoints aimed at maintaining and protecting biota. Thus, no new understanding of risks is gained by including this assessment endpoint in the ERA.

- (3) The combination of measured, modelled, guideline and observational measurement endpoints used in the baseline ERA are inadequate and do not support the weight of evidence approach that the authors claim to be using. Nearly all of the "weight" for the assessment amounts to nothing more than comparing conservative and deterministic measures of exposure in tissues or the surrounding media to hyperconservative and deterministic effects thresholds. For most of the assessment endpoints (e.g., piscivorous birds, waterfowl, local wildlife, threatened and endangered species), the "observational studies" were limited to anecdotal evidence or studies designed for purposes other than

assessing the risks of PCBs. Perhaps most surprisingly, no ambient or *in situ* toxicity tests were conducted (e.g., caged fish studies, sediment and water bioassays, fish feeding studies to mink, etc). This is an important line of evidence in a site-specific ERA, and has been a major component of assessments conducted for other contaminated riverine systems (e.g., Clinch River, Clark Fork River, East Fork Poplar Creek)(Kemble et al. 1994; Jones et al. 1999; Halbrook et al. 1999). Such studies are currently being conducted as part of the Housatonic River PCBs ERA (personal communication with Roy Weston staff and subcontractors). The weight-of-evidence approach simply means use of information from all sources, but particularly from three techniques, a "triad" of (a) toxicity tests, (b) chemical measurements, and (c) biological surveys in the field (Environment Canada 1999). For sediment, the approach has been formalized (Chapman 1986, 1990, 1996). The weight of evidence approach should not necessarily attribute equal strength to each line of evidence – Menzie et al. (1996) have proposed a formal and quantitative means to combine lines of evidence when estimating risks. Thus, in addition to the shortcomings in the ERA with respect to not obtaining the data required to build a weight-of-evidence assessment, none of the available methods for formally combining lines of evidence were used in the assessment. At best, the measures of effect and exposure specified in the problem formulation could be used in a screening level assessment. For a Phase II baseline assessment, the measures of effect and exposure and their use in a weight-of-evidence assessment fall far short of what is required. The argument in the responsiveness document that there was insufficient time for additional toxicity tests and field studies is unacceptable given the 10 year timeframe since this assessment began.

Exposure Assessment

- (4) The general approach of using the HUDTOX and FISHRAND models to estimate concentrations of PCBs in water, sediment and fish tissues is a reasonable one and likely the only feasible approach for estimating concentrations well into the future. It also appears that the models have been calibrated to existing data and their performance shown to be generally acceptable. I had two major frustrations in evaluating the appropriateness and sufficiency of the modeling approach. First, the equations underlying the HUDTOX and

FISHRAND models were not presented, nor were the modeling inputs, concepts, assumptions and rationales adequately described. I understand that other reports and peer review panels have or will deal with this issue, but more could have been presented so that the ERA peer reviewers could be in a position to properly evaluate the models. As an example, we are told that the FISHRAND model is a probabilistic model (e.g., pages 44 and 46) that predicts 25th, 50th and 95th percentiles. Yet, no equations or input distributions are described. How can we evaluate this probabilistic model without this information. The authors should consult the *Guiding Principles for Monte Carlo Analysis* (U.S. EPA 1997) which describes reporting requirements for probabilistic analyses. My second frustration with this chapter is that little or no information was provided on sample designs and sample sizes for the chemical monitoring studies that were undertaken. How are we to judge the credibility of the various measures of centrality and variance without this information?

Egg concentrations in piscivorous receptors were estimated by applying a biomagnification factor from the literature (28 for total PCBs, 19 for TEQ-based concentrations). I would guess that this number would vary depending on species and species condition, congener composition (for total PCBs), and environmental conditions. It would be useful to provide information on the expected variability (e.g., standard deviation, range) of parameters that are crucial to the exposure calculations. Better still, would be to conduct probabilistic analyses so that the impacts of variability and incertitude in the input parameters on predicted exposures can be determined.

On page 40, the report states that the TEQ congener distribution was assumed to be constant from year to year in the FISHRAND bioaccumulation model. Based on statements elsewhere in the report that lower chlorinated PCBs with chlorines in the ortho position degrade faster than higher chlorinated PCBs with chlorines in the meta and para positions, constant congener composition over time seems unlikely.

Total PCBs concentrations in water, sediments, benthic invertebrates and fish are described as "averages" (arithmetic mean or geometric mean???) and 95% upper confidence limits (UCL) on the mean. The rationale for using 95% UCL on the mean in exposure

calculations is not clear. It was argued in the report that predators tend to "average" their exposures over time and space (e.g., sometimes eating more contaminated fish, other times less contaminated fish). For predators, the issue is to determine what the "average" is and its associated uncertainty. Confidence intervals about the mean are the appropriate measure of this uncertainty (although lower confidence limits should also be calculated and used to bracket the quotient calculations). This rationale does not apply for all assessment endpoints. For example, non-motile invertebrates (e.g., clams) and plants cannot spatially average their exposures over the river segments that were the basis for estimating exposures. For these assessment endpoints, a better representation of exposure variability would be to estimate the 5th and 95th percentiles of the lognormal distribution, which will be much wider than the 5th and 95th confidence limits on the mean. Gilbert (1987) provides the formula for calculating 5th and 95th percentiles (and confidence limits about these percentiles) for parameters that are lognormally distributed.

For those fish species in which a fillet to whole fish conversion factor for lipid content was unavailable, fillet concentrations were used instead of whole body concentrations. The net result of this decision is that fish concentrations for white and yellow perch were underestimated by roughly a factor of two. A more defensible decision would have been to use a range of conversion factors based on factors observed in other fish species (weighted towards more similar species).

Although not my area of expertise, it is my understanding that toxicity equivalency factors are only roughly known for particular fish, bird and mammal species (e.g., within an order of magnitude of reported values). Treating TEFs as point estimates ignores this uncertainty. Further, assuming that BZ #126 was at the reported detection limit when it occurred at levels below this value seems to have little scientific justification. A better approach would have been to use distributional techniques to extrapolate to levels below the reported detection limit. If this technique is infeasible (because of few positive detections), then a range of approaches should be used and the results compared (e.g., assume detection limit, half detection limit, zero).

- (5) The exposure equations for the wildlife receptors are well described and, for the most part, the inputs have been adequately specified and justified. Most of the issues described below are fairly minor in nature. The only major problem I have with the exposure approach for wildlife was the continued reliance on conservative point estimates, rather than use of distributions. In my introductory comments, I pointed out that there are published case studies and Superfund guidance available that describe how to conduct probabilistic exposure modelling for wildlife.

In several places in the exposure chapter, water or food ingestion rates are labelled as "normalized". The units provided (e.g., L/day), however, indicate that the rates are not normalized to body weight (i.e., L/kg body weight/day). The exposure equations also show body weight in the denominator, which negates the need to normalize the ingestion rates in the numerator. I believe the ingestion rates therefore are not normalized rates. If they are, then a major error has occurred because the units would not cancel out to mg/kg/day for average daily dosage.

The wildlife exposure equations are used to estimate both mean average daily dose and 95th UCL average daily dose. Only the input variables for concentration (e.g., water, sediment, diet), however, are treated as having variability. Unless other important input variables (e.g., ingestion rates, foraging effort, dietary composition, etc) are treated as distributions, the 95th UCL average daily dose has little meaning because major sources of variability are being ignored. Note that 95th UCL outputs cannot be calculated by combining 95th UCL inputs according to the exposure equation (when a series of 95th percentiles are multiplied together the result will be a percentile >>> 95th percentile in the output distribution). Uncertainty propagation techniques such as first order error propagation or Monte Carlo analysis are required for this computation.

The exposure analyses all assumed that the wildlife assessment endpoints forage exclusively in the Hudson River year round (i.e., all modifying factors = 1). This may be a reasonable assumption for non-migratory species with small home ranges (e.g., kingfishers), but seems grossly conservative for species that migrate, have large home ranges, or forage in upland

areas less affected by PCB contamination (e.g., bald eagles, raccoons).

The total daily ingestion rate for mink was based on a study by Bleavins and Aulerich (1981). This study was a pen study and it seems likely that total daily ingestion rate would be much higher for wild mink because they must expend more energy foraging for food, defending territories, etc. Food ingestion rate also varies with food quality (ingestion rate increases as gross energy of diet items decreases). An alternate approach that takes account of these and other factors is described in Moore et al. (1997, 1999a).

The mink diet for the Hudson River was assumed to consist of 34% fish and 16.5% invertebrates (the remainder was not specified). Mink are, however, opportunistic carnivores with highly variable diets. Studies cited in the Wildlife Exposure Factors Handbook (U.S. EPA 1993) indicate that fish composition in the diet may vary from 0 to 75%, while muskrats and other small mammals may be insignificant or major components of the mink diet. For the exposure analyses for mink, a range of different possible diets should have been explored to determine the consequences on estimated average daily dose.

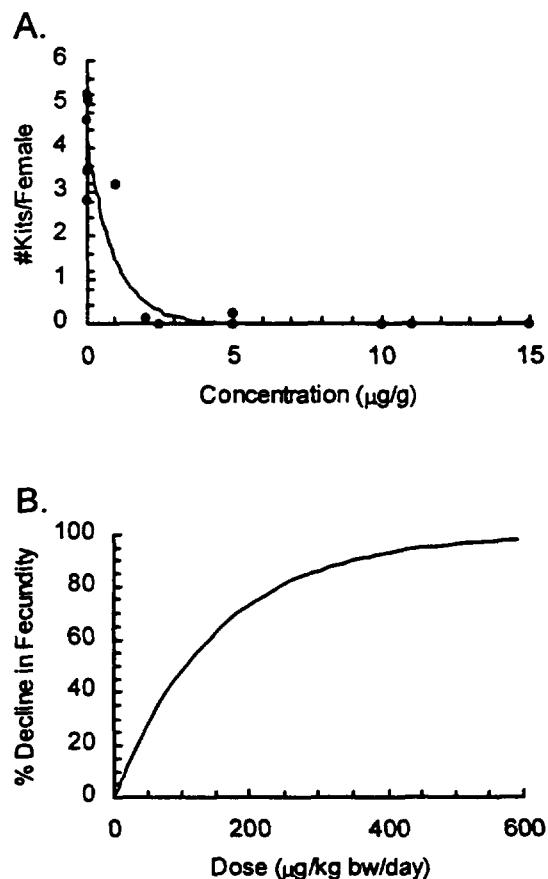
Effects Assessment

- (6) While there are some advantages to deriving field-based NOAELs (e.g., avoiding lab-to-field and interspecies extrapolations), I do not support their use in this assessment for three reasons: (1) the field-based NOAEL is unbounded because no corresponding LOAEL was derived, (2) the methodology for deriving the field-based NOAEL has not been sufficiently developed and validated (see, for example, the extensive database, methods development and validation efforts that have taken place for developing sediment effects concentrations – MacDonald et al. 2000), and (3) there is general accord that NOAELs (and LOAELs) are poor choices for estimating low toxic effects in ecological risk assessment (Stephan and Rogers 1985; Suter 1996; Moore and Caux 1997; OECD 1998; Environment Canada 1999; many others). Using generic order-of-magnitude uncertainty factors to derive TRVs from laboratory- or field-derived NOAELs or LOAELs is perhaps an acceptable approach in a screening level ERA (i.e., to identify risk scenarios in need of further analysis). The TRVs

approach, however, should not be the basis of an effects assessment in a higher tier ERA such as the baseline Hudson River PCBs ERA. The TRV approach is deficient in many ways including: (1) multiplying ten-fold safety factors for each of several extrapolations (e.g., LOAEL to NOAEL, interspecies, etc) results in hyperconservative threshold estimates of toxicity, (2) use of one NOAEL (or LOAEL) result ignores much of the available information from other studies (laboratory and field) or from other treatments within the same study, (3) the ten-fold safety factors ignore much of the available information that could be used to develop empirical safety factors (Chapman et al. 1998), and (4) TRVs provide little information to risk managers about the potential magnitude of effects that may be occurring if they are exceeded. A far superior approach would be to develop concentration- or dose-response relationships (based on one or multiple studies) for each assessment endpoint. Moore et al. (1999a) used this approach to develop dose-response curves for mink exposed to mercury and to PCBs (see text box 1 for an example). Additional comments on the effects assessment follow.

The wide range of NOAELs and LOAELs for body burdens is used as a rationale for not developing

Text Box 1. (A) Log Poisson regression model for combined results from 3 long-term PCBs feeding studies to mink. (B) Estimated dose-response curve.



body burden TRVs for benthic invertebrates. This is an unfair summary of the data because toxicity results from a wide range of congeners and Aroclors are being lumped together. The ranges would narrow considerably if only appropriate Aroclor mixtures (e.g., 1242 or 1248) were considered, although the available data are somewhat limited.

There appears to have been no attempt to evaluate the quality of the toxicity studies before selecting the key studies used to derive the TRVs. I would have expected acceptability criteria to have been developed (e.g., for control responses, use of appropriate protocols and statistics, etc) against which each toxicity study would be judged. TRVs should not be based on studies that are not of acceptable quality. If studies were evaluated prior to deriving TRVs, this should be indicated in the ERA along with the acceptability criteria used to evaluate the studies.

The same toxicity studies tended to be used over and over again to derive TRVs for the various fish, bird and mammal assessment endpoints. This indicates to me, that the ERA lacks the capability to separately assess risks to different fish species, different bird species or different mammal species. Had specific bioassays been performed for each of the assessment endpoints (or closely related surrogates), there would be justification for developing separate TRVs for each endpoint. This was not the case. Instead of pretending to have the capability to assess effects and risks to each of a large number of fish, bird and mammal species, the authors should be more forthright and admit that, at most, this assessment can only assess risks to fish, bird and mammal species for which toxicity data are available or are available for close surrogates (e.g., spottail shiners, mallards, mink). Alternatively, TRVs could be developed from species sensitivity distributions that would be protective of, for example, 95% of fish, birds and mammals. In the latter case, the list of assessment endpoints would be reduced to three generic ones – protection and maintenance of fish, bird and mammals. This approach would also make for a far less repetitious ERA.

The implicit assumption in using an interspecies extrapolation uncertainty factor to derive a TRV is that the assessment endpoint is always more sensitive than the test species (by 10-

fold!). There is equal probability, however, that the assessment endpoint is less sensitive than the test species. Had NOAELs and LOAELs been multiplied by a factor of ten, instead of divided by ten, most of the toxicity quotients would go below one. That is, uncertainty cuts both ways. It would be a far more intellectually honest exercise to develop bounds or, better still, a distribution for TRVs. This would facilitate development of bounded or probabilistic quotients (see Bartell 1996). Then risk managers would have a proper perspective on which to judge the credibility of the risk estimates.

The LOAEL for pheasants on page 95 appears to be off by two orders of magnitude.

Risk Characterization/Uncertainty Analysis

- (7) The question posed here on “whether the methodologies used in calculating ... TQs are adequately protective of ... receptors” is a misleading one. The objective of an ecological risk assessment is not to be “protective” but to estimate and characterize risks to biota. It is then up to risk managers, with input from stakeholders and the public, to decide what remedial actions are required to ensure protection for receptors of concern. It is an easy exercise to design toxicity quotients that are “protective”. Simply pile on the safety factors and conservative assumptions and you have “protective” quotients. The approach, however, lacks credibility. To take a well worn analogy – if weather forecasters predict rain every day (to be protective), then eventually people will start ignoring the forecasts because they have no credibility. Thus, a forecast of “it will very likely rain” when rain is highly unlikely is not helpful; rather we would like to know the true odds, and act according to our attitude toward risk.
- (8) The risk characterization does not adequately characterize risks posed by PCBs in the Hudson River to receptors of concern. Risk describes the relationship between probability and magnitude of effect (Warren-Hicks and Moore 1998). The TQs that were by far the dominant line of evidence in the risk characterization chapter do not address probability or magnitude of effect. Further, the consequences of any effects that could occur to populations or communities were not explored with ecological models or other techniques.

Specific comments on the risk characterization chapter follow.

The field evidence for effects of PCBs to benthic organisms is very weak. Although there may be a relationship between PCBs concentrations and some of the benthic community metrics, this relationship is confounded by differences in the sediments between sampling locations. In fact, "when PCBs concentrations were normalized to TOC [a more accurate indication of bioavailable PCBs], there were no significant differences between stations" [page 120]. Perhaps a more sophisticated multivariate technique would have provided stronger evidence of a relationship between PCBs concentrations and benthic community structure. Non-metric clustering and association analysis, for example, can identify clusters based on community composition and ranks variables (e.g., TOC, sediment grain size, metals concentrations, PCBs concentrations, etc) in order of importance for distinguishing the observed clusters (see Landis et al. 1996 for an example). This technique is more sophisticated than the crude and insensitive approach described in chapter 5 of using ANOVAs to tests for differences in community indices between locations. Nevertheless, the evidence as presented in chapter 5 gives little indication that PCBs have caused effects to benthic community structure in the Hudson River. By the conclusions chapter (chapter 7), however, the field evidence is seen in a somewhat different light – "The analysis shows a reduced macroinvertebrate community ... [and] All three lines of evidence [of which the field study is one] suggest an adverse effect of PCBs on benthic invertebrate populations ...". Uncertainty in the analysis is further stated as being low. In my opinion, this conclusion is not supported and further suggests that the assessment is biased towards finding risks even when this is not warranted on appeal to the available evidence. Similar biases are evident elsewhere in chapter 5 and the conclusions chapter (e.g., interpretation of the tree swallows field study, discounting of evidence of healthy fish, kingfisher and waterfowl populations).

Often the same line of evidence was used repeatedly as an indicator of risk to assessment endpoints. For example, concentrations of PCBs in water were compared to ambient water quality criteria for the assessment endpoints involving benthic community structure, fish, bird and mammal populations. The connection between this line of evidence and, for example, risks to tree swallows seems very tenuous indeed. Similarly, many of the TRVs derived for different fish, bird and mammal species are based on the same toxicity studies.

In the end, I do not believe that the ERA has taken a weight-of-evidence approach, primarily because most of the so-called lines of evidence are slight variations on the same theme – comparing observed or predicted concentrations to generic TRVs. With the exception of the field studies for benthic community structure and tree swallows, little field evidence is available to support the risk characterization (anecdotal evidence from a few individuals is of little use). No ambient or *in situ* toxicity tests were conducted to support the ERA.

In several places in chapters 5 and 7, statements that “true risks are likely underestimated” appear. Ignoring for the moment the difficulties with the notion of “true risks”, the statements ignore the obvious conservatism that was built into the TQ calculations. For example, the comparison of Tri+ PCB concentrations in water to water quality criteria is stated as underestimating risk to fish because the criteria are based on the sum of all congeners (page 127). The PCBs criterion, however, is a conservative threshold based on concern for protection of wildlife. Because the same concentration of PCBs in water leads to higher exposures in top food chain species than in fish species, it seems likely that a PCBs criterion for wildlife will be highly conservative when applied to fish. Thus, I doubt very much that “true risks” are being underestimated in this or any other risk scenario.

- (9) Many of the important sources of uncertainty in this ERA were identified and discussed in chapter 6. Obviously, I would have preferred that quantitative uncertainty analyses be conducted. Nevertheless, a qualitative discussion of uncertainties is an important exercise in ecological risk assessment, and the discussion in chapter 6 is reasonably comprehensive. Perhaps more discussion of the uncertainties about TRVs for individual assessment endpoints should be added (because assessment endpoints may also be less sensitive than test species). Also, the influence of assumptions about diet for mink, and foraging behaviour of species with large home ranges should have been explored.

The sensitivity analysis described in section 6.5.2 is of no use. No information is provided on input parameters, nor were rationales provided. Exposure parameters were all apparently assigned triangular distributions, yet this distribution has no theoretical plausibility for any stochastic environmental variable I can think of (see Seiler and Alvarez 1996). TRVs were

assigned uniform distributions that spanned an order of magnitude. However, since assessment endpoints have an equal probability of being more or less sensitive than the chosen test species, the appropriate range should have been two orders of magnitude (more if additional safety factors were used). A uniform distribution also assumes that all possible values for sensitivity are equiprobable. This will not be the case – very few species are highly sensitive or highly tolerant. The appropriate distribution would likely be the log-logistic or lognormal distributions for TRVs. As a result of these shortcomings, I have no faith in the results of the sensitivity analyses, nor do I believe “the output distributions of toxicity quotients generated by this Monte Carlo analysis represent population heterogeneity”. Again, the authors should refer to Agency guidance (U.S. EPA 1997, 1999) for reporting the results of a Monte Carlo analysis.

General Questions

- (1) For many of the reasons stated in the preceding responses, I do not believe that the Hudson River PCBs ERA adequately characterizes risks to sensitive populations. Although the assessment is reasonably clear (except for the missing information describing the equations, inputs and their rationales for the HUDTOX and FISHRAND models), consistent and transparent, it fails the criterion on being “reasonable”. In my opinion, the assessment is excessively conservative and superficial. In the end, I have no idea of the seriousness of the risks posed by PCBs to Hudson River biota. What are the probabilities of effects of differing magnitudes? What are the ecological consequences of any effects that do occur? How will proposed remedial actions reduce risks? What are the countervailing risks introduced by the remedial actions? The ERA provides little information to help answer these and other important questions. Without this information, I do not see how effective environmental decision making can take place.
- (2) I think I have said enough (too much, more likely).

Conclusion

In its current form, I do not believe this ERA is acceptable. To make it acceptable, ambient and in situ tests and field studies are required, new analyses (e.g., probabilistic risk analyses, ecological modeling), and a major re-write are required. Whether this will occur, I cannot say. Thus, I am unsure whether to choose the "acceptable with major revisions" option, or the "not acceptable" option.

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Ross Norstrom

**Hudson River PCBs Site Reassessment RI/FS
Risk Assessments
Peer Review 4**

Charge for Peer Review 4

The peer review for the Human Health Risk Assessment and the Ecological Risk Assessment is the fourth and final peer review that the U.S. Environmental Protection Agency (USEPA) is convening for the major scientific and technical work products prepared for the Hudson River PCBs site Reassessment Remedial Investigation and Feasibility Study (RI/FS). USEPA previously has peer reviewed the modeling approach (Peer Review 1) and the geochemistry studies (Peer Review 2). The peer review for the computer models of fate, transport, and bioaccumulation of PCBs (Peer Review 3) will conclude on March 28, 2000.

This peer review is comprised of two panels of independent experts: one for the Human Health Risk Assessment and one for the Ecological Risk Assessment. The reviewers are asked to determine whether the risk assessment they review is technically adequate, competently performed, properly documented, satisfies established quality requirements, and yields scientifically valid and credible conclusions. The reviewers are not being asked to determine whether they would have conducted the work in a similar manner.

In making its remedial decision for the PCB-contaminated sediments in the Upper Hudson River, USEPA will answer the three principal study questions that are a focus of the Reassessment RI/FS:

1. When will PCB levels in fish meet human health and ecological risk criteria under continued No Action?
2. Can remedies other than No Action significantly shorten the time required to achieve acceptable risk levels?
3. Could a flood scour sediment, exposing and redistributing buried contamination?

The risk assessments will be used to help address the first two questions. Specifically, the risk assessments will be used in the Feasibility Study to back-calculate to appropriate levels of PCBs in fish to compare various remedial alternatives, including the No Action alternative (i.e., baseline conditions) required by federal Superfund law.

Ecological Risk Assessment

The goal of the Ecological Risk Assessment is to evaluate the risks to ecological receptors associated with exposure to PCBs in the Hudson River in the absence of remedial action of the PCB-contaminated sediments (i.e., under baseline conditions). The following documents will be provided to the peer reviewers:

Primary

- Baseline Ecological Risk Assessment, August 1999
- Responsiveness Summary for the Baseline Ecological Risk Assessment, March 2000

References

- Ecological Risk Assessment Scope of Work, September 1998
- Responsiveness Summary for Ecological Risk Assessment Scope of Work, April 1999
- Executive Summary for the Baseline Ecological Risk Assessment for Future Risks in the Lower Hudson River, December 1999
- Executive Summary for the Human Health Risk Assessment, Upper Hudson River, August 1999
- Executive Summary for the Human Health Risk Assessment, Mid-Hudson River, December 1999
- Executive Summary for the Revised Baseline Modeling Report, January 2000
- Suggested charge questions from the public for the ERA, February 2000

The reference documents listed above are being provided to the reviewers as background information, and may be read at the discretion of the reviewers as time allows. The reviewers are not being asked to conduct a review of any of the background information.

Additional Reassessment RI/FS documents are available on USEPA's website (www.epa.gov/hudson) and/or by request. Additional documents include the following:

- Hudson River Reassessment RI/FS Database, August 1998
- Executive Summaries for other USEPA Reassessment RI/FS Reports
- Peer Review Reports from first two peer reviews
- Responsiveness Summary for first peer review

Specific Questions

Problem Formulation/Conceptual Model

- 1) *Consistent with USEPA guidance on conducting ecological risk assessments (USEPA, 1997), the problem formulation step establishes the goals, breadth, and focus of the assessment. As part of the problem formulation step in the ERA, a site conceptual model was developed (Chapter 2.3, pp. 11-19). Please comment on whether the conceptual model adequately describes the different exposure pathways by which ecological receptors could be exposed to PCBs in the Hudson River. Was sufficient information provided on the Hudson River ecosystems so that appropriate receptor species could be selected for exposure modeling?*

As a general comment, I confess to an intense dislike for the jargon that frequently accompanies these exercises. Perhaps the legal wording behind the process requires that terms like 'receptors' be used instead of potential species at risk, or some other descriptive term. Receptor used in this way is very non-standard terminology, and would never be used in the wildlife toxicology scientific literature. A receptor, biochemically, is a specific protein that has a site with an affinity for binding a particular chemical or group of chemicals. The Oxford Standard dictionary refers to an organ or cell that responds to an external stimulus and transmits a signal to a sensory organ, or a region of a tissue or molecule in a cell membrane, etc. which responds specifically to a substance. None of these definitions even remotely encompass a species, population or community. That does not mean the new meanings should not be assigned when they help to clarify, but these terms only obfuscate, in my opinion. Another grand word for which confuse could be substituted!

As pointed out in the life history and ecology of the various species in the appendices, very few of the chosen terrestrial organisms can be classified as exclusively piscivorous without supporting field evidence, and the one species for which this could safely have been assumed, the osprey, was not included for some inexplicable reason. It may be that bald eagles *are* primarily piscivorous on the upper Hudson, but they are certainly known to eat birds and scavenge carcasses of dead animals in other areas, and there was no particular attempt to verify the feeding habits of Hudson River bald eagles as part of this ERA, as far I could figure out. Therefore, it may be that mink, bald eagles, and kingfishers should be classified in the far right box – as consuming a variety of prey although primarily piscivorous. The other main criticism I have with the conceptual model is that the exposure of amphibians and reptiles is only via flood plain soils. What about amphibians that are almost exclusively aquatic throughout their life phases, and furthermore may form an important food source for reptiles, providing a direct aquatic link? Herptiles are ultimately eliminated from the ERA process because of a paucity of information, but honestly, the quality of information on a lot of the other species is not much better, and the very large worldwide concern for decline in amphibian populations would seem to me to be enough reason to have attempted to include them. They are likely to be more endangered than the great blue herons that eat some of them, in any case.

I am fully in support of the criticisms that the conceptual model should have been constructed on the basis of field surveys. For example, what is the status of the mink population in the Hudson river ecosystem (as opposed to tributaries) at this time? Are there any at all? The only reference that is provided in this regard is Foley et al. (1988) which is hardly up-to-date nor comprehensive in the first place. In this survey, insufficient detail is given to be sure that any mink were taken on the Hudson River proper. The data were grouped by large areas including several counties on either side of the Hudson River. My suspicion is that most animals were not from near the river, since PCB concentrations in the Upper and Lower Hudson River area mink were a factor of two or less higher than most other areas of the state, which makes no sense if they were eating Hudson River fish. A statement is made that, "Collection of animals near bodies of water known to be contaminated with PCBs, including the Hudson River and Lake Ontario, required more intensive efforts than in other areas of the state." Absence of mink along the shores of the Great Lakes is widely considered to be indicative of the effects of PCBs on reproduction. Although this is difficult to prove, the very extensive literature on the sensitivity of the species, plus assessment of available habitat, makes a cause-effect relationship, in this case absence of the species, much easier to establish than is the case for many of your other chosen species.

The evidence is stronger from the Foley et al. (1988) study that river otter were taken from the Hudson River proper, which is now further defined as 'valley'. Concentrations in Hudson River valley Otter were much higher than in other areas of the state. Assuming that river otters still do occupy the river, consideration should be given to assessing their reproductive status, and if trapping is done, comparing data on baculum length in males with the studies carried out by Henny on the Columbia River. Incidentally, none of Henny's work, which is the most extensive available on river otter, was used in this assessment. The work of Harding et al. (Environ. Health Persp. 107:141-147, 1999) on correlation of reproductive and morphological condition in mink and river otter in relation to organochlorine contamination has also been ignored.

Pg. 12, 2nd para. The description of the SARs that determine which PCBs are more readily metabolized and excreted is far too simplistic. First of all, the comments should be placed into taxonomic context. Fish and invertebrates are poor metabolizers of PCBs, although lower chlorinated congeners may be excreted back to water unchanged as was pointed out. However, birds, mammals and at least some reptiles, metabolize PCBs according to quite well-defined rules in which substitution pattern of the chlorines, degree of induction of enzymes, etc. is more important than degree of chlorination, although the latter also has some influence. Thus, PCBs with no chlorine at a *m-p* position on at least one ring are much more readily metabolized than those that are substituted at both *p,p'* positions. Birds and mammals that are exposed to dioxin-like (Ah receptor active) compounds may have sufficiently induced enzymes to metabolize them. This is especially the case for BZ#77, but has also been shown to occur for BZ#118 and possibly BZ#105 in man, seals and polar bears. In fact, polar bears are efficient metabolizers of even BZ#126. Thus, generalizations such as are made in para. 2, page 13 about hexachlorobiphenyls taking a long time to reach equilibrium cannot be made. Some of them are metabolized quite quickly by birds and mammals, although most are not. It is even more unconscionable to generalize to Aroclor 1254, which contains several congeners which are easily metabolized by birds and mammals. Aroclor 1242, the major Aroclor of concern in the Hudson river, has an even higher percentage of metabolizable congeners.

Assessment and Measurement Endpoints

- 2) *Assessment endpoints specify the valued ecological resources to be protected, such as local fish populations. They focus the risk assessment on particular components of the ecosystem that could be adversely affected by contaminants from the site. Please comment on whether the assessment endpoints selected (pp. 19-20) adequately protect the important ecological resources of the Hudson River. Are major feeding groups and sensitive species sufficiently covered by the selected assessment endpoints?*

The aquatic endpoints appear to have been adequately chosen, but after reading through the life histories of the various species in the appendices, I began to wonder why so many terrestrial species need to be included. Not only is there a fair degree of uncertainty in feeding ecology in many cases, there is little (no?) comparable information on PCB levels from other areas (e.g., raccoons and bats), and therefore no field studies that might give a hint as to possible toxic effects.

This, combined with TRVs derived from rats, makes the risk assessment process more like guess work than science. This is especially true for TEQs (see below for comment on TEFs and TEQs).

The WHO TEFs are an improvement over the largely rodent-based values which were in

common use until recently. However, it must be understood that there is still a large variability in species sensitivity within each group. This has been adequately demonstrated for fish and birds, but there is still very little information on mammals. *In vitro* studies with bird hepatocytes, which has also been ignored in this assessment, indicate that there is likely to be considerable variability in sensitivity to specific congeners as well (i.e., variable TEFs among species within a group). I therefore believe the study should have been restricted to terrestrial species for which we have site-specific information, extensive data from other areas, or assessments (laboratory or field) indicating that the species is sensitive: mink, otter, bald eagle, tree swallow and great blue heron. Given what we know about the sensitivity of mink, if action or no-action is protective of this species, it will be for the others as well. We do not even need to do a formal risk assessment to reach this conclusion.

I initially believed osprey might have been a better choice than bald eagles because of their exclusive piscivory, although when I reached chapter 5 I discovered that anecdotal information indicates osprey they are rare in the upper Hudson River, and probably breeding on nearby lakes when present. However, bald eagles seem to be similarly scarce, so it is moot which species would have been the better choice.

- 3) *Measurement endpoints were used to provide the actual measurements used to estimate risk. Please comment on whether the combination of measured, modeled, guideline, and observational measurement endpoints used in the ERA (pp. 20-29) supports the weight of evidence approach used in the ERA.*

In all of the Assessment Endpoints, I consider gathering actual field data, and making comparisons of species abundance, diversity and reproductive endpoints in a similar, uncontaminated riverine ecosystem, to be the most important and accurate approach as to assessing whether there is current harm. Because concentrations of PCBs are not predicted to increase, evidence (or lack thereof) of effects under current conditions is by far the best predictor of potential future risk. This was quite eloquently defended on page 29, but then criticized as not being sensitive, confounded by other stressors, etc. I find most of these arguments untenable. Can observational data be any less useful in assigning cause-effect relationships than not knowing if there are even any effects present (even if the species is present) and then using a water-quality guideline to estimate probable risk? I think not. While it is true that observational data may require time to gather, and is best done over a period of time to look at trends, it is also true that EPA has had ample time to have been gathering data over past years, and chose not to take that approach. Having not done so is no excuse for not doing it in the future before decisions are made on action/no-action.

Various water and sediment water quality guidelines, while useful in the absence of other methods, may seriously over- or underestimate risk, given the lack of adequate experimental data to support relative sensitivity of many of the species under consideration, and other large uncertainties involved in their derivation. I found the repetition of these guidelines (and other endpoints) under virtually every heading unnecessary. Why could they not have been discussed once, if they have such general applicability? I do not think TRVs derived this way add much to weight of evidence.

The wording of the benthic community endpoint strikes me as odd. Was the purpose solely to preserve structure as a food source for fish? What about the importance of maintaining the

benthic community for its own sake? Note the transposition of the Endpoints 2 and 3.

I question how accurately TEQ values can be calculated for protection and maintenance of fish, given the quality of the data base that is available. In fact, I am not so sure about total PCBs either. I assume Endpoint 3 for fish refers to sufficient loss of benthic species that it would affect the food supply of fish dependent on this community? This appears to overlap somewhat the statement above.

In general, the multiplicity of endpoints, provided the data to support them have a scientifically sound basis, which is well beyond the purview of this process, and therefore cannot be assessed, will tend to support the weight of evidence, but this will undoubtedly vary considerably in quality and certainty among the various groups of animals. TRVs based on estimated exposure rates are the most problematical to deal with, especially for mammals (and especially for TEQs – see later). Body burdens/concentrations will be very difficult to estimate from exposure alone, especially for a mammal like a raccoon, for which there is no experimental data, including its capability to metabolize PCBs, and for which the diet is uncertain. BMFs in bird eggs are fairly well established, and there is a considerable body of information on embryotoxicity related egg to concentrations, so this likely to be one of the stronger endpoints. However, uncertainty in diet composition (and substitution of a few representative species, as surrogates for those actually eaten), is a problem even here.

Furthermore the available data in the literature has not been fully explored as to its applicability to the Hudson River exposure situation (or toxicity for that matter). Having references spread through several different volumes and places made it difficult to assess what may or may not have been taken into consideration. However, it appears that one reference with which I am very familiar, Braune and Norstrom (1989), which provides forage fish/herring gull whole body, liver and egg biomagnification factors for a range of PCB congeners, as well as PCDDs, PCDFs, and other OCs has not been used. It would have been a much better reference for estimating body burdens and egg residues for species like great blue heron, bald eagle and kingfisher which are also primarily piscivorous and probably accumulating residues over a much longer period of time, than a passerine insectivore like the tree swallow or a generic BMFs for PCBs in birds of 28. The assumption that patterns of exposure in prey species are going to be highly conserved is probably not true for any bird or mammal other than tree swallows (see discussion below). The relatively large amount of data for tree swallows and the reliance on it has therefore hindered, rather than enhanced the ERA, in my opinion.

Characterization of risk for sensitive species can only be poorly understood because of lack of detailed information on food web structure, and inherent patchiness of feeding ecology of upper trophic level species. The conclusions that can be reached are only as strong as the data base available for the assessment. This is not intended to be a criticism. Riverine ecosystems are actually much more complicated than large lake ecosystems, such as Lake Ontario, which we are only beginning to understand after many years of study and a much bigger investment in time and resources. The list of species in the Hudson River in the Appendices is adequate demonstration that this is not a simple system.

I believe that the authors of the conceptual model and providers of background ecological information have done an admirable job of attempting to distill the complexity of the Hudson River

ecosystem and how it relates to the PCB contamination into something manageable, although I think too many terrestrial species were included, as indicated above. A more open-minded and pragmatic approach, e.g. use of *in vitro* studies, more extrapolation from field BMFs in other species, etc. may not have yielded answers which are much more concrete than the ones which were reached. That is difficult to prejudge, but my opinion is that a better feeling for the probability of impact on various species would have been achieved if the process had been more transparent and clear. On this point, I am in agreement with GE that there is a unnecessary degree of complexity and opacity in the process which is not fully penetrable with the resources (information and time) allotted to this peer review. There were many times when I found myself wondering where I had seen this table or that, and in which volume it resided. Sometimes I just gave up trying to figure it out.

I disagree completely with the philosophy stated in the middle of page 29 that, "because the receptor may be affected by a variety of other factors unrelated to the stress of interest," observational studies are not sensitive. If a species is already under stress from another source, for example, a poor food supply (mentioned as cause of mortality in juvenile raccoons), mercury, habitat destruction, etc., then protection of the species from effects of PCBs may be either moot, or it may be the deciding factor in survival. For example, resident adult bald eagles in coastal areas of British Columbia appear to resort to eating seabirds outside of the breeding season, and consequently some individuals have very high levels of PCBs, even though their chicks, which are being fed local fish, may have quite low concentrations. I simply do not accept the single chemical approach to risk assessment. Identical exposures, body burdens, whatever approach is taken, may have different outcomes in different areas if the health of the whole ecosystem, including exposure to other chemicals, is not taken into account.

I found Appendix J to be almost unfathomable.

Exposure Assessment

- 4) *USEPA used several avian and mammalian exposure models to evaluate the potential risks due to PCBs (see, ERA, pp. 37-71). Sampling data from USEPA, NOAA, NYSDEC, and USFWS collected from 1992-1996 were used to estimate current fish body burdens and dietary doses to avian and mammalian receptors. Future concentrations of PCBs were derived from USEPA's fate, transport, and bioaccumulation models, which are the subject of a separate peer review. Concentrations of PCBs in piscivorous bird eggs were estimated by applying a biomagnification factor from the literature. Please comment on the appropriateness and sufficiency of this approach to estimate ecological exposure to PCBs.*

I found the methodology for determining the non-ortho PCBs, especially BZ#126, suspect and inadequate, and consequently the calculation of TEQs unacceptable. The only data indicating what the actual relative concentrations of toxic minor components, such as BZ#126, were to total PCBs are in the largely unreadable Figure K-43. Using a ruler and a magnifying glass, I estimated the mass fraction of BZ3126 to be ca. 10^{-4} , which is approximately what I would have expected. However, there are no data given for its mass fraction in Aroclors for comparison, even though this information was published several years ago by at least two groups I am aware of. It is standard practice in all of the laboratories I know of to do a prior separation of non-ortho PCBs on a carbon column, usually along with PCDDs and PCDFs, add ^{13}C -labelled internal standards for the target analytes and use mass spectrometry detection. There are three very good reasons for this. Sensitivity

is improved because the sample can be taken to much lower volume if the mega PCBs are first removed (purely a chromatographic reason), accuracy and precision are significantly enhanced by isotope dilution calculations, and the chances of false positives are virtually eliminated. Apparently the BZ#126 data passed the qualification test, but it is not at all clear what level was spiked to reach this conclusion. If the spike was at a substantially higher concentration than the native concentrations in the samples, then the precision and accuracy of the BZ#126 data may be much poorer than assumed.

Because an insensitive method was used, a high proportion of the samples had undetectable BZ#126. Given my concerns about how well the qualification was carried out for this congener, there may also be concerns about the accuracy of the data that were above detection. The decision to use the detection limit as a surrogate for the real concentration is completely unacceptable in a risk analysis. For purposes of statistics, sometimes a half-detection limit is used, and sometimes it is desirable to assign random numbers from zero to the detection limit for multivariate analysis. But when BZ#126 is estimated to represent 33-85% of the fish-based TEQs (Table 3-1), then real numbers are required. Incidentally, the heading and overall description of what is actually being presented in Table 3-1 leave a lot to be desired. Upper River mean in what? Egg and Chick of what species? There is no question that the TEQs will be overestimated by this procedure, but by how much is impossible to say. As far as I am concerned, calculation of TEQs should not even have been attempted for those samples with non-detect BZ#126. On page 40, Book 1 of 3, it is stated that the error is "likely within an order of magnitude at most". Is this degree of uncertainty acceptable? It is quite unnecessary to have lived-with if up-to-date analytical procedures had been followed. This inadequacy compromises the TEQ-based assessment.

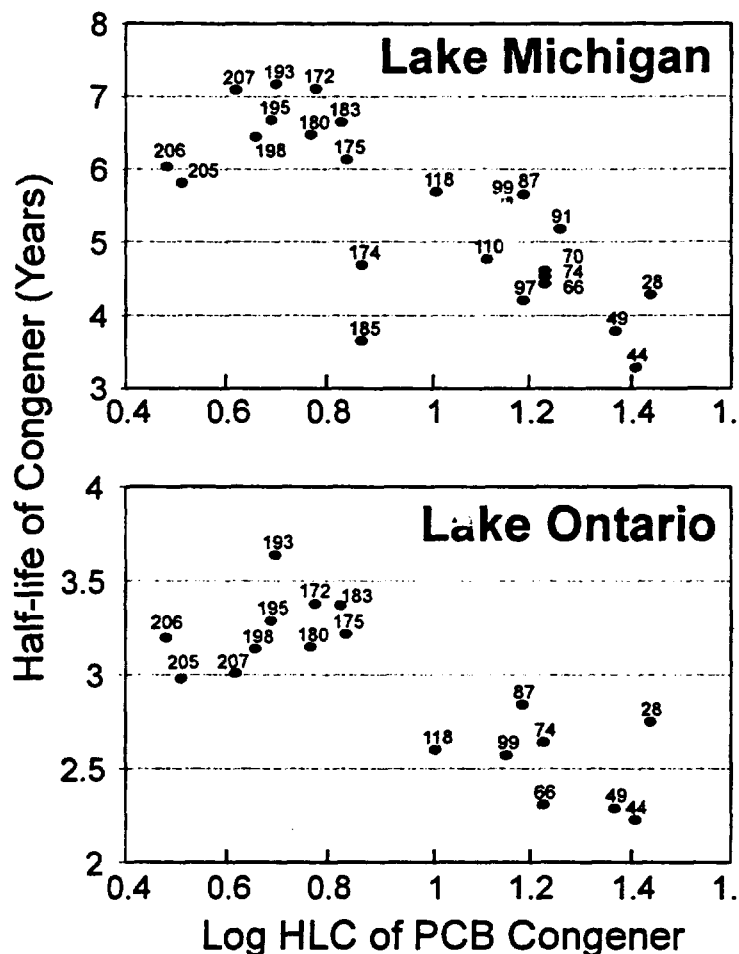
While I agree in general with procedure used to develop TEFs for Tri+ PCB concentrations, subject to concerns about BZ#126 above, I think it is a very large assumption that congener distribution will remain relatively consistent from year to year. It was one of my major recommendations in the BMR review that HUDTOX and FISHRAND be calibrated for a small number of specific congeners, then run into the future to validate whether the assumption of unchanging congener composition is valid.

In figure 1, the half life of various congeners in Lake Michigan and Lake Ontario over a 10 year period is shown to be dependent on air/water partitioning (HLC). Some of the changes in Lake Ontario may be due to differences in Aroclor loading patterns from the Niagara River in the early time period, but the changes in the Lake Michigan data (which is influenced by Aroclor 1242 from the Fox River, and therefore similar in some respect to the Hudson River) are thought to be largely due to evaporative losses. Although the half lives are different in the two lakes, the change in rate of decrease with HLC (slope) is similar in the two lakes: a two-fold difference between trichloro and octa-/nonachloro congeners. These data illustrate that considerable alteration in congener composition may occur over the time frame that HUDTOX and FISHRAND are intended to operate, and such a possibility should be included in the HUDTOX model.

I also objected in my review of BMR to the approach of estimating distributions of concentrations in fish based on Bayesian optimization of distributions of parameters with already known distributions, such as log K_{ow} and lipid percentages fish. It was my opinion that modeling specific congeners as suggested above would provide a more rigorous calibration of the model because these parameters would no longer be available for adjustment. It was also my feeling that

the model should predict distribution of concentrations according to size of fish, especially for large species like large-mouth bass, not just an overall population distribution. That way, size preferences of the various species could be factored into the analysis. Given the other imponderables, such as actual composition of the diet of piscivorous species, this may not be as important as I thought at the time, but it is still by far the most scientifically valid approach. FISHRAND, despite its purported mechanistic approach (and that is somewhat debatable), becomes a statistical empirical model the

Ecological Half-life Determined by Volatility?



way it is applied. Although it appears quite successful at predicting Tri+ PCB concentrations this is accomplished to a degree by artificially altering the distribution of log K_{ow} values. Is this an indication that congener distributions were changing over time the model was calibrated?

Figure 1. Ecological half life of PCB congeners in herring gull eggs from Northern Lake Michigan (mouth of Green Bay) and Scotch Bonnett Island Lake Ontario, 1971-1981 vs. Henry's Law Constant

One of the biggest problems that I have with the Exposure assessment relates in various ways to what I have already alluded to above – unrealistic extrapolation and assumptions. The data base is so limited and incomplete that this has to be done, unfortunately, but it could be done with a lot

more circumspection and awareness of how biological differences affect bioaccumulation.

For example, what is the point of calculating avian and mammalian TEQs in water, sediment, invertebrates and forage fish (Tables 3-3 to 3-6 - note that the heading should say TEQs based on TEFs)? This might be useful for piscivorous fish, which do not metabolize PCBs to any extent. But since a large proportion of TEQs is contained in BZ#77, which is quite rapidly metabolized by both birds and mammals, and an indeterminate amount of the BZ#126 values are not real numbers to start with, an exposure assessment based on these values in the absence of some biological and kinetic considerations is quite meaningless.

TEQs should not ever be used for exposure assessment. They are only useful in the context of converting measured concentrations in the species being studied into a better measure of possible effects due to Ah-receptor mediated toxicity. TEQs are not bioaccumulated!

I am assuming from Tables J-2 and J-3 that BZ#77 was an important contributor (concentrations as well as TEQs) in tree swallows. That is probably because the females are deriving a large proportion of egg lipid from their diet of highly contaminated emergent insects, as opposed to lipid reserves. This is frequently the case for passerine birds, which lay a high proportion of their body weight in eggs. If the diet during rapid yolk deposition happens to be highly contaminated with PCBs, as is the case here, then there is little opportunity for metabolism to occur. In migratory species that probably are much less exposed most of the rest of the year, but use exogenous sources for egg production, local diet has a bigger influence on residue levels. However, this will probably not be the case at all for any of the mammals, and probably not also for species like the bald eagle, which lay a smaller percentage of their body weight as eggs, are not so dependent on lipid from endogenous resources, and may be resident and therefore exposed over a longer period of time. Another case in point are mallards. I do not know off-hand what strategies mallards have for obtaining lipid sources deposited in eggs, but geese and ducks lay a high proportion of their body weight in eggs, and some species (e.g., the snow goose) rely entirely on endogenous resources for lipids. In this case, the concentrations in eggs reflect what is retained by the female from exposure over the previous year (more or less, depending on metabolism of the congener). Therefore, amount

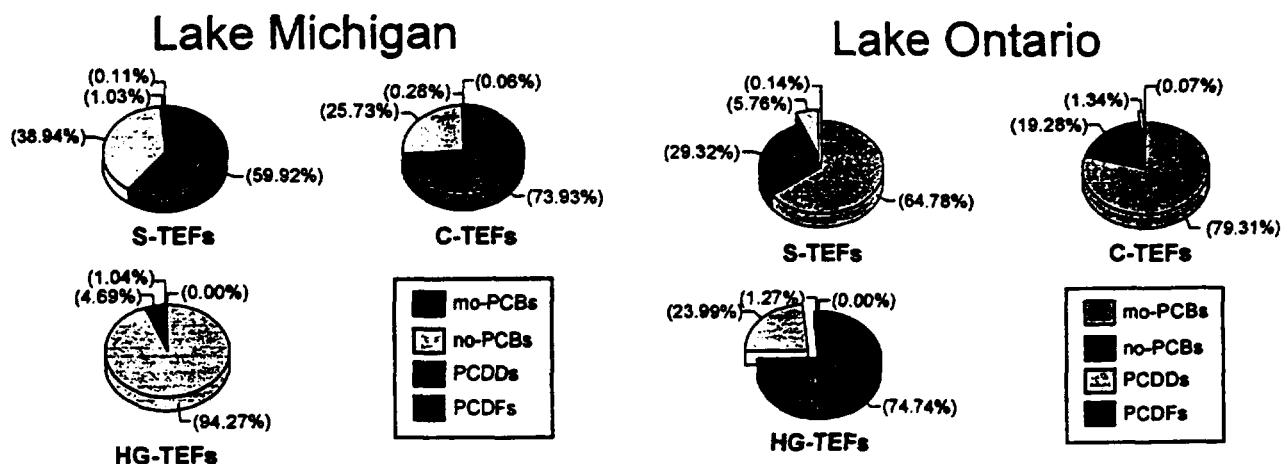


Figure 2. Calculated proportion of contribution to TEQs from mono-ortho and non-ortho PCBs (all of the congeners listed in Table 4-2, Book 2 of 3 in the ERA) in herring gull eggs

from northern Lake Michigan (mouth of Green Bay) and Scotch Bonnett Island Lake Ontario, 1971-1981. S-TEFs are based on those derived by Safe et al. C-TEFs and HG-TEFs are chicken and herring gull based values taken from studies on EROD induction in cultures of primary hepatocytes of embryos of these species by Kennedy and coworkers.

of PCB deposited to eggs may have little to do with local conditions, unless the individual is resident.

To state it simply, the relative contribution of BZ#77 (and probably also BZ#81), cannot be assumed to remain the same in birds and mammals as it does in water, sediments, invertebrates or fish. This another case where only real measurements will tell the story. From my experience, there is ample evidence that BZ#77 accumulation is much lower in the majority of birds (and their eggs) than BZ#126 if exposure to PCBs occurs over a relatively long period of time. Incidentally, this applies to an even greater extent to 2,3,7,8-TCDF. If there is high exposure during yolk formation in some species, TCDF is found in bird eggs, but if exposure is spread out over a long period of time, it is metabolized so rapidly it seldom shows up at all.

It is not exactly pertinent to this charge question, but the contribution of PCDFs to any assessment of exceedance of TEQ-based TRVs is something that cannot be ignored. Although not listed in Table 4-2, it is very clear from the complete table in van den Berg et al. (1998) that TCDF is very toxic to birds (although not readily bioaccumulated), for example. PCDD/Fs are much more important in fish than PCBs as well. Since 2,3,7,8-TCDF and 2,3,4,7-PnCDF are important contaminants in Aroclors, and are closely associated with them, it is unconscionable that they were completely ignored just because they happen to have a structure that is a little different from PCBs. PCDF data should have been obtained.

To illustrate the difficulties in use of TEQs, Figure 2 shows application of three different TEF indices to the same data set from two colonies of herring gulls in the Great Lakes. At the time of this analysis, avian WHO values were not generated, but if they were applied, the proportion of TEQs would appear similar to those based on HG-TEFs. The important thing to note is that the contribution of mono-ortho PCBs is close to 80% of the total if chickens are used as the reference species. They are still a significant proportion if rats are the reference species, but their importance disappears completely if the herring gull-specific values are applied. Herring gulls simply do not respond to BZ#118 or BZ#105, and that is likely true for most wild birds. Wild gallinaceous species have shown greater sensitivity than others in both *in vivo* (pheasants) and *in vitro* (turkey) tests, but in no case does the absolute or relative sensitivity approach that of the chicken. It is one of those amazing things that happens every now and then in science. The sky appears to be falling, when it isn't (reference to the story of Chicken-Little for those of you with a children's literature bent). While there is always the possibility that EROD-based TEFs derived in this manner are not reflective of embryotoxicity, the evidence accumulated from studies by Brunström's research on the effect of injection of PCBs (primarily BZ#77) into eggs of various species of birds strongly suggests that there is a rank-order correlation of embryo LD₅₀s and EROD induction.

The take-home message is that BZ#126 is very important to Ah-receptor mediated toxicity in birds, and failure to come up with an adequate assessment of exposure/bioaccumulation of this congener negates the TEQ approach to TRVs, in my opinion. Another take-home message from

Figure 2 may be that PCDFs are not all that important. That is probably true for most species which are continuously exposed, but it would also be a mistake for some species. TCDF has a very high TEF in avians. For tree swallows (*vide infra*), which may deposit TCDF into eggs before they have a chance to metabolize it because of utilization of exogenous resources for lipids, it could be a more significant compound than the PCBs themselves.

Because birds do not metabolize BZ#118 or BZ#105, but do metabolize BZ#77 (Norstrom, R.J. 1988. Patterns and trends of PCB contamination in Canadian wildlife. In: Hazards, Decontamination and Replacement of PCB, J.-P. Crine, ed., Plenum Publ. Corp., New York, pp. 85-100.), it is also important to understand the kinetic implications of the bioaccumulation of the BZ#77 in eggs, as indicated earlier.

Note the publication by Froese et al. (ET&C, 17:484-492, 1998), another one of the many relevant references that are not were not used in this assessment (it was published 2 years ago). They state in the abstract that, "Our results indicate that patterns of relative concentrations of PCB congeners change with trophic level, specifically from sediment to invertebrates and from tree swallow eggs to nestlings." This is very true, and the substance of my criticism of the use of TEQs as an exposure TRV. They recommend a TRV of 0.015 TEQ/g total organic carbon in sediment as protective of sensitive avian species. While I do not agree with this conclusion, why was this reference was ignored as part of this assessment.

- 5) *Have the exposure assumptions (ERA, pp. 46-66 and Appendices D, E, and F) for each fish and wildlife receptor been adequately described and appropriately selected? Please discuss in detail.*

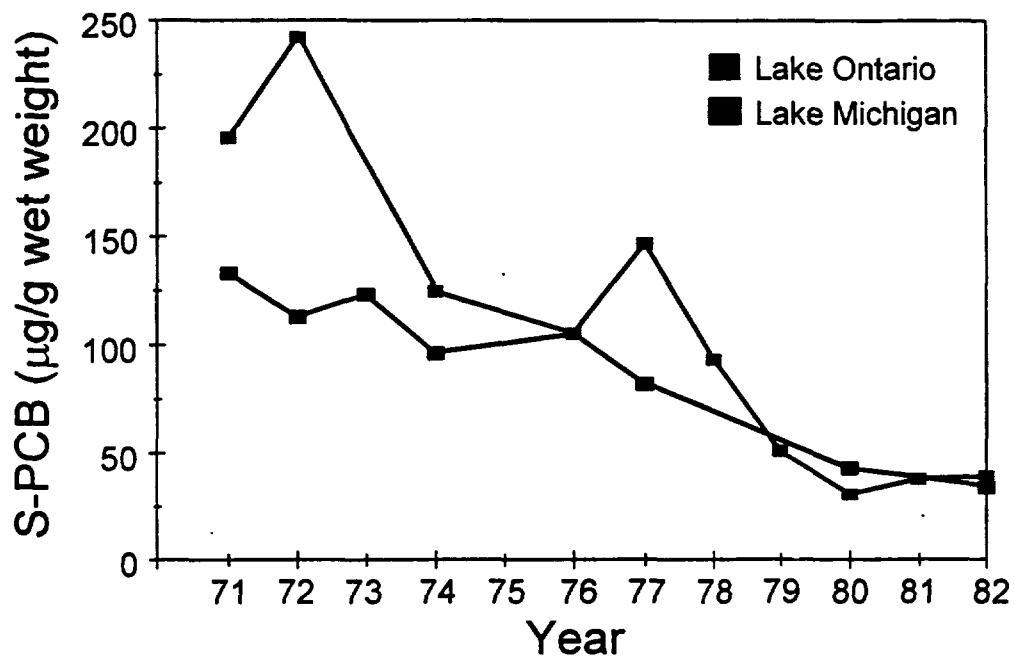
The feeding rates and diet composition assumptions have been addressed as well as possible under the circumstances. However there need not have been so many assumptions if more field data had been obtained. I have only one major difficulty, which is the treatment of biomagnification factors in eggs (3.4.3.5). Egg BMFs of 28-30 for total PCBs is a reasonable value based on a continuous exposure scenario. However, as pointed out below, I do not think it is feasible to discuss BMFs of TEQs. The composition of congeners contributing to TEQs is expected to vary between fish and birds/mammals due to metabolism, especially of BZ#77. TEQs should only be used for tissue concentration-based TRVs. The very low BMFs of 2 for tree swallows and 3 from one mallard and two wood duck samples are undoubtedly a reflection of the fact that the diet being used for this calculation is an overestimate of the true exposure (much lower) of the species to PCBs prior to egg formation. Although this is probably a fair representation of reality, it must be remembered that the actual source of PCBs in eggs of these species may not be the Hudson River, or if it is, a considerable proportion may have been retention from previous years' exposure, allowing time for metabolism of congeners such as BZ#77 to occur. Under these circumstances, calculation of BMFs is not valid -- comparison of apples and oranges.

Effects Assessment

- 6) *For field-based toxicity studies, only a NOAEL toxicity reference value (TRV) was developed because other contaminants or stressors may be contributing to observed effects. Please comment on the validity of this approach. Also, please comment on whether the general approach of using uncertainty factors (interspecies, LOAEL-to-NOAEL, and subchronic-to-*

chronic) is appropriate in developing TRVs that are protective of Hudson River receptor species.

Sum PCB Concentrations in Herring Gull Eggs



OC and TEQ Levels Relative to 1982, Lake Ontario: Correlation with Reproductive Success

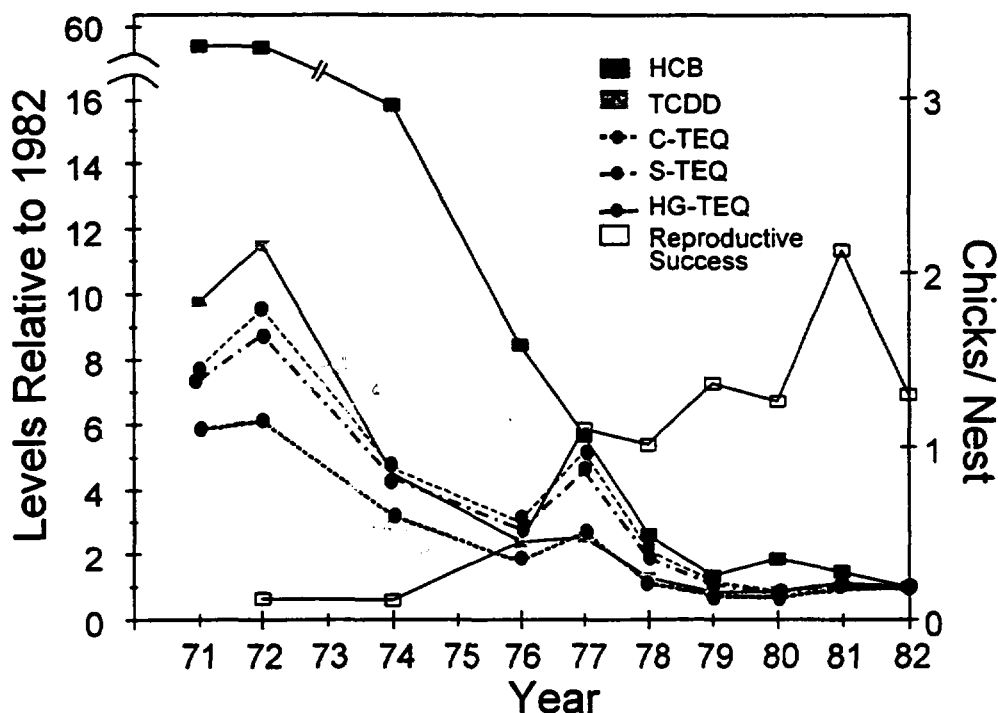


Figure 3: Sum of PCB concentrations and TEQs ion eggs, and reproductive success of herring gulls from northern Lake Michigan (mouth of Green Bay) and Scotch Bonnett Island Lake Ontario, 1971-1981

The whole idea of doing a risk assessment is to protect an ecosystem/species. How is this possible if multiple stressors are not figured-in? I grant you that using most-sensitive-species like chickens or mink birds will be protective. But is it real? It will probably be overprotective for most species. On the other hand, the lack of an ERA which integrates exposure to all of the potential stressors (esp. PCDFs), may miss real effects that are there.

I have little doubt that mink, and possibly river otter, will be affected by the present and near-future levels of PCBs in the Hudson River. All the other assessments pale by comparison.

As shown in Figure 3, concentrations of PCBs in herring gull eggs were similar in Lake Ontario and northern Lake Michigan in the early 1970s and declined steadily to the early 1980s. Although herring gulls were experiencing reproductive failure in the late 1960s in Lake Michigan, this may have been due to DDE-induced eggshell thinning rather than embryotoxicity of PCBs. During the period represented in Figure 3, reproductive success was (anecdotally) normal in Lake Michigan, despite similar concentrations of PCBs as in colonies in Lake Ontario that were experiencing essentially zero reproductive success (note that adults of both populations are resident, so there is no confusion introduced by migration). Note also that during the fairly rapid increase in reproductive success of herring gulls in Lake Ontario between 1974-77, there was no significant change in TEQs, TCDD (a significant contributor to TEQs in Lake Ontario – see Fig. 2), or total

PCBs (not shown). HCB concentrations were in excess of the LD₅₀ in the early 1970s, and its decline correlates much more strongly with improvement in reproductive success than other OCs. Spearman Rank Order Correlation tests of reproductive success vs. chemical concentrations is given below. HCB wins, but TCDD and HG-TEQs are also in the race. Chorostyrenes are functionally correlated to HCB because the source of both was carbon electrodes used in chlorine production. S-PCBs do not seem to be much more important than many other OCs, despite concentrations over 200 ppm in the early 1970s.

Chemical/ Class	p-level
HCB	0.0009
TCDD	0.0072
HG-TEQ	0.0072
S-PCDD	0.0072
S-CStyr	0.0125
S-PCB	0.0199
S-DDT	0.0298
C-TEQ	0.0298
S-TEQ	0.0358
S-Mirex	0.0424
Dieldrin	0.0610
S-PCDF	0.1544
b-HCH	0.2351

I do not wish this analysis to be perceived as underestimating the significance of PCB contamination. There is no question that reproduction of mustelids is sensitive to PCBs, and that alone is sufficient to be concerned about PCBs in the Hudson River. Within the avian world, there is some indication that terns are more sensitive than gulls, but they are not part of the assessment. And, we have no information at all for many species, especially mammals. Nevertheless, it is important to use the best scientific information available and realize when we are barking up the wrong tree. Herring gulls can quite clearly sustain normal reproduction with PCB concentrations exceeding 200 mg/kg in their eggs. That does not mean that their immune systems were not compromised, or that behavioral problems did not exist. But if these effects were present, they did not affect recolonization of Lake Ontario by herring gulls in the late 1970s. If anything, forage fish community structure changes were more important. In Lake Erie, the invasion of zebra mussels has been the single most important factor in exposure of piscivorous avians to contaminants.

Kannan et al. (Hum. Ecol. Risk Assess. 6:181-201, 2000) point out that NOAELs and LOAELs may be artifacts of the study design, and may not reflect the specific point of the dose-response relationship. Because they felt NOAELs were overprotective, and LOAELs were underprotective, they chose a mean of the two as the likely threshold at which effects would occur. Although this approach is a debatable one, it certainly is worth having the debate, and seeing if a

consensus could be reached on its utility.

Risk Characterization/Uncertainty Analysis

- 7) USEPA calculated toxicity quotients (TQs) for all receptors of concern on both a total PCB and dioxin-like PCB (TEQ) basis. Please comment on whether the methodologies used in calculating these TQs are adequately protective of these receptors.

They are probably over-protective for birds in many cases, see the example of the herring gull above. Peterson et al. (1993, Critical Reviews in Toxicology 23:283-335) provides LOAELs, NOAELs and LD₅₀s for embryotoxicity of TCDD in birds. The LOAEL and the LD₅₀ for pheasant was in the 1-2 mg/kg range, and the LOAEL for the eastern bluebird was 10 mg/kg. Given the lower sensitivity of birds to PCB-based TEQs than mammals, and the apparently overall low sensitivity to Ah-receptor based toxic effects, I believe the TQs to be considerably overprotective for birds. Note that Elliott et al. (1996) proposed a LOAEL of 210 ng/kg TEQs for CYP1A induction in bald eagle eggs, whereas this ERA came up with a TRV of between 10-20 ng/kg (pg. 106). The Elliott et al. estimate is arguably not a toxic effect, rather an indication of biochemical sensitivity to exposure. Concentrations of total PCBs in yolk sacs were in the order of 200-400 ng/kg lipid, and there was no indication of concentration-related effects for morphological, physiological or histological parameters. The true LOAEL for significant toxic effects may therefore be much higher than 210 ng/kg. However, since TQs using average and upper confidence limits were frequently in the 100 range for bald eagles using the low TRV values, they would likely be greater than 1 even if the higher values were used.

Kannan et al. recently published a thorough analysis of the derivation of TRVs for aquatic mammals (Kannan et al. 2000. Toxicity reference values for the toxic effects of polychlorinated biphenyls to aquatic mammals. Human and Ecol. Risk Assessment 6:181-201.) This paper was published subsequently to the preparation of this report, but must be considered in the final analysis.

I am not in any way endorsing the conclusions of the publication, since there was insufficient time to assess it. However, it must be taken into account in the final revision of the ERA, and appears to have been a very reasoned analysis of aquatic mammalian TRVs. Most of the conclusions are based on studies of mink and European otter. Note that they are in agreement with my assessment that dietary BMFs of TEQs are precluded as an approach to developing TRVs, "due to site-specific differences in congener composition and species-specific differences in toxicokinetic and toxicodynamics,"

The conclusion was that threshold concentrations for effects were preferable to NOAELs or LOAELs. Maximum allowable toxicant concentrations (MATCs) were defined as the toxicant concentration in mammalian 'receptor' species that is likely to be without appreciable risk of deleterious effects, calculated as the geometric mean of NOAEL and LOAEL. The values were 9 mg/kg total PCBs and 520 pg/g TEQs in lipid in liver. Dietary exposure TRVs were expressed as a range of values: 0.01-0.15 mg/kg of total PCBs and 1.4 to 1.9 pg/g TEQs, wet weight. The method of derivation of TEQs was not assessed, so these values would be subject to re-analysis before being accepted.

- 8) *The risk characterization section of the ERA (Chapter 5, pp. 117-151) summarizes current*

and future risks to fish and wildlife that may be exposed to PCBs in the Upper Hudson River and current risks to fish and wildlife in the Lower Hudson River. Please comment on whether the risk characterization adequately characterizes the relative risks to ecological receptors (e.g., piscivores, insectivores) posed by PCBs in the Hudson River.

As discussed earlier, the risk to piscivorous birds is probably overestimated, but by how much can only be estimated with any degree of certainty for bald eagle. In this case, I believe the TRV to be at least an order of magnitude too low, and perhaps two orders of magnitude. If this is the case, the risks are likely to be borderline to this species. However, the risk to mink and otter is high as determined. There is no real basis to determine the validity of the risk assessment for raccoons and bats, because of the paucity of information on these species.

Note that the reference to Table 5-84 on page 146 should read Table 5-85.

The anecdotal information in Table 5-85 that mink numbers are large and increasing and there are 'quite a few otters' needs to be verified, especially as to the exact location of these populations. Are the mink on the main stem, or on tributaries and other areas nearby, and therefore not necessarily exposed to PCBs. If, indeed, there are successfully breeding mink populations on the mainstem, this would suggest that the risks are being overestimated.

- 9) *The uncertainty analysis is presented in Chapter 6 of the ERA (pp. 153-165). Have the major uncertainties in the ERA been identified? Please comment on whether the uncertainties (and their effects on conclusions) in the exposure and effects characterization are adequately described.*

See discussion above about the problems of calculating TEQs when there are so many non-detects for BZ#126, and the lack of consideration of metabolism of BZ#77. These are major sources of uncertainty which are glossed over in the analysis.

Toxicological uncertainties are discussed adequately. However, I feel that better use could have been made of *in vitro* comparative toxicology studies in birds to reduce the level of uncertainty.

Uncertainty in long-range extrapolation of exposure due to congener composition changes over time is not dealt with.

General Questions

- 1) *A goal for Superfund risk assessments is that they be clear, consistent, reasonable and transparent and adequately characterize risks to sensitive populations (e.g., threatened and endangered species). Based on your review, how adequate are the ERA and the Responsiveness Summary when measured against these criteria?*

The fragmented way in which the whole process is being conducted, with baseline modeling,

ecological and human health assessments essentially divorced from each other, makes it very difficult for peer reviewers to obtain a 'big picture'. I was fortunate to have participated in both the BMR and ERA, so I had some continuity of perspective in how concentrations were derived. In fact, I requested to be involved in both for that reason. Perhaps it will not turn out to have been as important as I imagined, but my opinions of BMR certainly influenced how I viewed how I approached this review of the ERA.

I found the volume of material, and the requirement to jump around between three volumes of material in order to make sense of statements was very time consuming, and probably contributed to me missing several points that I should have addressed. I therefore have to give the risk assessment a low grade on clarity and transparency.

I did not have adequate time to cover the responsiveness Summary at this writing, however, I will have done so prior to the peer review meeting and provide comments at that time.

- 2) *Please provide any other comments or concerns, both strengths and weaknesses, with the ERA not covered by the charge questions, above.*

Recommendations

Based on your review of the information provided, please select your overall recommendation for the ERA and explain why.

1. *Acceptable with major revision (as outlined)*

I would like to see the results of individual congener modeling from HUDTOX and FISHRAND before making final conclusions as to the acceptability of using the long-term predictions of Tri+ PCB concentrations in the risk analysis, especially the stability of the ratio of TEQs/Tri+ PCBs.

If possible, the approach to calculating TEQs based on non-detectable BZ#126 numbers should be re-visited.

The use of TEQ BMFs should be reassessed, and probably eliminated.

Much better use needs to be made of the available literature. I did not have time to assemble a list, but there are many relevant studies that were not considered at all, and may have considerable influence on the TRV values that were used.

The literature on bald eagles suggests that the TRVs used for this species are too high. Note that early attempts to ascribe bald eagle reproductive failure in the Great Lakes to PCBs was confounded by high levels of DDE, and it was probably the latter compound which was responsible (egg shell thinning). Another source of information which is highly relevant, but not readily accessible, are studies on white-tailed sea eagle reproductive success in the Baltic Sea (Helander et al., 1999, White-tailed sea eagles *Haliaeetus albicilla* in Sweden: reproduction in relation to DDE, PCB, coplanar PCB and eggshell parameters; manuscript in thesis of Anders Olsson, University of Stockholm). I will bring a copy of the thesis with me to the meeting.

Although some attempts were made to obtain anecdotal information on presence of species, this effort was wholly inadequate to provide appropriate observational-based assessments. This should be addressed in any follow-up studies, especially the status of mink and otter populations.

See other comments above.

Timothy Thompson

May 11, 2000

John Wilhelmi
Eastern Research Group
110 Hartwell Avenue
Lexington, MA 02421

Re: Hudson River Ecological Risk Assessment Review

Dear Mr. Wilhelmi

Thank you for this opportunity to review the report entitled *Baseline Ecological Risk Assessment; Hudson River PCBs Reassessment RI/FS*. As will be reflected in my comments below, I believe that the document authors did a good job following the EPA guidance on risk assessment for Superfund, and that the document is generally well-written. I believe that the conclusions concerning overall baseline environmental risk within at least the upper Hudson are probably correct, but it is my recommendation for the ERA that EPA consider some major revisions before it should be released as final. The basis for my concern fall into three major categories as follow:

- **Transparency.** I am concerned that the ERA does not meet the basic requirement for clarity and transparency defined in the *Ecological Risk Assessment Guidance for Superfund, Process for Designing and Conducting Ecological Risk Assessments* (USEPA 1997). Clear documentation and communication is paramount to evaluating the need for risk reduction through remediation. I found that document(s) difficult to follow and logic paths not always laid out in a way that myself as an experienced environmental scientist could understand the decisions made.

There is a great deal of background information on that is simply referenced in other documents, that are not clear to the reader unless he/she consults and reads the entire document. For example, what was the determination about existing data that lead to sampling additional stations in 1993, and what was the logic that supported using only eight samples to characterize risk for the Thompson Island Pool, two for Stillwater and one for the Federal Dam reaches of the River. In addition, decisions are made on data inclusion/exclusion without presenting to the reader a satisfactory explanation as to why they were included/excluded (e.g., previous sediment sampling done by NYSDEC, or the NYSDOH 1976 - 1985 benthic macroinvertebrate study). Another example is the apparent dropping

of the 95% UCL calculation from FISHRAND projections in the *Responsiveness Summary* (see Tables 3-10 and 5-9 in that document), without any discussion. There are very likely good reasons for all of the above, but they are not readily reflected in the *Scope of Work*, *ERA*, or the *Responsiveness Summary*.

- **Technical Deficiencies.** There is a tremendous amount of good data that has been compiled into the Hudson River Database (HRDB), that was never used in the ERA. My own opinion is that an ERA should be conducted in a way that allows for the evaluation of remedial alternatives. This apparently is also what the authors of EPA's *Ecological Risk Assessment Guidance for Superfund* intended as well, since they cite OSWER Directive 9285.7-17 in noting that the objective of the ecological risk assessment process is "(1) to identify and characterize the current and potential threats to the environment from a hazardous substance release; and (2) to identify cleanup levels that would protect those natural resources from risk".

Much of the sediment data in the available in the HRDB could be utilized to strengthen the risk characterization, and produce a document upon which a feasibility study could be constructed. For example, the NYSDEC sediment data could be incorporated with EPA's 1993 data to produce PCB-contaminant isopleths for the River. Such maps would help communicate that there are PCB exposure levels over an entire reach (e.g., Thompson Island Pool), and avoid the criticism that reach wide risks in the ERA have been characterized using too few stations that do not reflect all conditions within a reach. I also offer the recommendation that the more recent datasets for sediments or benthic infauna be incorporated into the ERA (e.g., the 1998 Exponent benthic infaunal analyses, the 1998 surface sediment data collected by GE — incorporated into the model calibration, but apparently not into the baseline ERA). If there are very good technical or data quality assurance reasons why those should not be, then that needs to be better communicated in the ERA.

- **Organization.** While the writing is good in this document, it is difficult to track information over multiple documents to evaluate statements or decisions made in the ERA. There are four separate documents comprising the ERA, and an additional five to seven additional documents one must access in order to have a complete picture of all the elements that comprise this ERA. Perhaps this reflects a personal preference, but I believe that an ERA should be a stand-alone document that would include the following elements:

Section 1: Introduction. Fairly similar to the introduction section provided in the ERA now, including purpose and organization

Section 2: Background. A more complete background section that could be something as simple as the executive summary and relative figures from a remedial

investigation. This section should lay out the description of the entire Hudson River as relevant to the ERA. This should include not only a description of the PCBs in the system, but also other sources of potential chemicals of potential concern. It would include a better description of the PCB distribution in the depositional zones (currently only identified as "hot spots" in the ERA figures. Finally, a complete description of the fate and transport processes from a hydrodynamics perspective (e.g., depositional vs. scour zones) is a precursor to understanding the model dynamics, as well as understanding important fish habitats within the River.

Section 3: Data. Inclusion of a summary of the three reports that comprise the data used in the ERA; the 1995 *Database Report*; the 1997 *Data Evaluation and Interpretation Report*, and the *Low Resolution Sediment Coring Report*. In addition, the fish and bird data collected by NYSDEC, NOAA, US Fish and Wildlife, and General Electric. A description of the data sets, and especially the relevant quality assurance determination (e.g., are the data compliant with National Contingency Program standards, or are they supportive data from the standpoint of a defensible ERA) is important for the reader to understand as we evaluate the findings. While this was done for the congener data used in this ERA, as a reader I need to understand the data useability for all facets of the ERA.

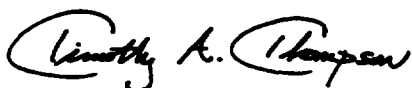
In addition, this section should cover the statistical treatment of any data. For example, the current ERA does discuss calculation of a 95% upper confidence limit on the mean (95% UCL), but omits a discussion of what happens when the 95% UCL exceeds the maximum concentration (see for example Table 3-7 where 95% UCLs for Brown Bullhead and Largemouth Bass exceed the maximum measured concentration). This section should also include rules for determining percentiles (which the current ERA omits). For example, what is the minimum number of samples needed to determine a percentile ($n > 10$), and what value is used when "n" data are not available.

Section 4: HUDTOX/FISHRAND. A more complete description of the HUDTOX and FISHRAND models. This is instrumental in understanding the prediction of future risks, and deserves a more complete description within the ERA. The presentation material given by Ed Garvey at our meeting in March would make an excellent chapter. While the reviewers have the benefit of his presentation by slide and video, an external reader will not. This relates back to the issue of transparency — the tools and decisions used in the ERA must be clear to the reader. I note that limitations and uncertainties to the predicted model results are not discussed in Section 6 of the ERA — something that clearly needs to be included. This proposed Section 4 should discuss the strengths, and limitations of the models; which should then be reflected in the Uncertainty Section of the ERA.

The subsequent chapters would then focus on the problem formulation, exposure characterization, assessment, risk characterization and uncertainty, as previously discussed.

The attachment that follows focuses on the specific questions given to the reviewers to answer. I trust that my responses will be helpful in assisting EPA in crafting a document that assists in developing risk management decisions. Should you have any questions, please feel free to contact me at 206 624 9349.

Sincerely,



Tim Thompson
Senior Environmental Scientist
ThermoRetec Corporation

Specific Questions

Problem Formulation/Conceptual Model

- 1) *Consistent with USEPA guidance on conducting ecological risk assessments (USEPA, 1997), the problem formulation step establishes the goals, breadth, and focus of the assessment. As part of the problem formulation step in the ERA, a site conceptual model was developed (Chapter 2.3, pp. 11-19). Please comment on whether the conceptual model adequately describes the different exposure pathways by which ecological receptors could be exposed to PCBs in the Hudson River. Was sufficient information provided on the Hudson River ecosystems so that appropriate receptor species could be selected for exposure modeling?*

In the narrow sense of the question posed, the conceptual model for biological fate and transport within the Hudson River is adequate for the purposes stated. However, from a risk communication standpoint, I recommend that EPA consider adding or supplementing the existing ERA in order to help the reader understand the overall environmental system and the compilation of data used in the ERA. Specific examples are provided below.

P. 3. Sect. 1.3. Site Investigation and Hudson River Data Sources

This is very high of level overview of the data collected and used in the ERA. Without accessing and reading the data management reports, there is no ability to assess whether information collected met the requirements for useability under the National Contingency Program; i.e., whether the data from each of the data sets listed could be fully validated, or whether it could be listed only as supporting information for the purposes of a Superfund assessment.

Much of the sediment and water information is available through the TAMS *Database Report*, in the *Low Sediment Coring Report* (December 1988), and the February 1999 *Addendum*. However, there does not appear to be a single source of information that describes the validation for each of the data sets, unless that would be the *Data Evaluation and Interpretation Report*, which is was not available to me, nor is a copy at EPA's Hudson website.

Again for transparency purposes, it would be useful to summarize that within a separate section of the RA.

P. 10. Sect. 2.2 Contaminants of Concern

While it is acknowledged that PCBs are the focus of the re-assessment for the Hudson River, it would be very useful to include a discussion of other chemicals of potential concern (COPC) known to exist in the River is necessary in order to put into perspective potential risks from PCBs. For example, metals as COPCs and their potential effect on benthic populations appear in Appendix H.

In Table H-10, the levels of lead, chromium, and mercury are at levels that exceed several different sediment benchmarks, including both the threshold effects, and the probable effects concentrations, of the consensus-based sediment quality guidelines for freshwater ecosystems (MacDonald et al, *in press*). While the ERA needn't assess risk for these other COPC, at least their effects on the eco-receptors (especially the benthic infauna) needs to be accounted for in the uncertainty section.

P. 11. Sect. 2.3.1, Exposure Pathways in the Hudson River Ecosystem

A fundamental question not defined in this ERA is the nature and extent of PCB contamination throughout the Hudson River. The ERA cited three documents that discussed in detail the magnitude and extent of contamination (*Baseline Modeling Report, Data Evaluation and Interpretation Report, Low Resolution Coring Report*). However, I did not find that those documents (I did not have the *Data Evaluation and Interpretation Report*) laid out the data in a way that at least the spatial distributions could be understood. The type of PCB-distribution maps put together apparently with the 1984 NYSDEC data (as cited in the Executive Summary for the *Data Evaluation and Interpretation Report*) would be very useful. It is this reviewers opinion that a knowledge of the contaminant distribution is necessary in order to evaluate whether the 10 sampling locations used in 1993 are representative of the entire PCB distribution in the upper Hudson River.

The remainder of section 2.3 is well-written and adequately covers the conceptual site model, as shown in Figure 2-4.

Assessment and Measurement Endpoints

- 2) *Assessment endpoints specify the valued ecological resources to be protected, such as local fish populations. They focus the risk assessment on particular components of the ecosystem that could be adversely affected by contaminants from the site. Please comment on whether the assessment endpoints selected (pp. 19-20) adequately protect the important ecological resources of the Hudson River. Are major feeding groups and sensitive species sufficiently covered by the selected assessment endpoints?*

With the following two suggestions, the assessment endpoints listed in the ERA appear to follow guidance and are adequate to assess risk to the important receptors of the Hudson River.

P. 20. Sect. 2.4, Assessment Endpoints

Phytoplankton are an important primary producer, particularly in ponded (i.e., with dams or wiers) sections of many riverine systems. Phytoplankton are identified as an important component of the FISHRAND food web -- as a principle food source for Spottail Shiner -- but are not identified as an important assessment endpoint in the ERA. If phytoplankton communities are not an important endpoint, then why they are excluded should be carefully defined.

Protection of significant habitats as an assessment (and measurement) endpoint is poorly defined. There is never a definition given on what constitutes a "significant" habitat, and how PCBs might affect those. This is a common critic in the comments to the ERA, and it could use some reconsideration. For example, in the *Responsiveness Summary*, Page 25 in response to EF 1.4 and EP 2.1, the comment is made that the significant habitats were not mapped due to the length of the Hudson (200 miles). The *Remedial Investigation for the Lower Fox River and Green Bay* has prepared habitat maps for all of the River and both shores of Green Bay using information compiled from federal (e.g., USFWS, NOAA), state (Wisconsin and Michigan Departments of Natural Resources), consultant reports (Exponent on behalf of the Fox River Group), and several commercial sources that sell maps for Geographic Information Systems. I believe the relationships between exposure to PCBs and species/habitats would be strengthened by this type of presentation.

- 3) *Measurement endpoints were used to provide the actual measurements used to estimate risk. Please comment on whether the combination of measured, modeled, guideline, and observational measurement endpoints used in the ERA (pp. 20-29) supports the weight of evidence approach used in the ERA.*

The measurement endpoints, as defined, are generally adequate to support the ERA. The exception may be the measurement endpoints selected to judge effects on significant habitats. The connection between "significant habitats" and surface water or sediment PCB concentration is ambiguous and tenuous at best. I would also note that in the ERA, body burdens relative to TRVs are discussed in Section 5.8 (Evaluation of Assessment Endpoint" Protection of Significant Habitats) as being indicative of risks to significant habitats, but that these are not listed as measurement endpoints. This is reiterated by EPA on page 21 of the *Responsiveness Summary* (Response to EL-1.2, EL-1.6, EL-1.7, and EG 1.3, third paragraph) where the exposure of fish, avian, and mammalian receptors is held up as a potential threat to significant habitats. If that case is to be made, then those should be listed as measurement endpoints earlier in the document.

As I recommended above, mapping of the significant habitats along the Hudson River would be an excellent way to communicate where those habitats exist, and how they are important to the overall assessment of risk.

I would recommend that the revised ERA include a better definition of when percentiles are employed, versus the use of the upper confidence limit on the mean. This is to some degree clarified in the *Responsiveness Summary* (see Page 21, Response to EF-1.9), but it again reflects the burden placed on the reader to read all of the associated documents in order to understand the analysis of ecological risks.

Available field observations of the presence and abundance of specific receptors is an important consideration for an ERA. The measurement endpoints used in the Hudson River ERA rightly acknowledge that fact. As I will discuss further below, I believe this ERA needs to include what data

are available on receptor populations in the Hudson River Valley in a weight-of-evidence approach. The discussion on Page 29 of the ERA argues that the major weakness of observational studies is that the a variable, and may not account for larger differences over time. Cause and effect relationships are important, and there are uncertainties associated with using those data. But those are equally (if not more) effective than using results from lab-gavaged chickens to assess risks to Belted Kingfishers.

Exposure Assessment

- 4) *USEPA used several avian and mammalian exposure models to evaluate the potential risks due to PCBs (see, ERA, pp. 37-71). Sampling data from USEPA, NOAA, NYSDEC, and USFWS collected from 1992-1996 were used to estimate current fish body burdens and dietary doses to avian and mammalian receptors. Future concentrations of PCBs were derived from USEPA's fate, transport, and bioaccumulation models, which are the subject of a separate peer review. Concentrations of PCBs in piscivorous bird eggs were estimated by applying a biomagnification factor from the literature. Please comment on the appropriateness and sufficiency of this approach to estimate ecological exposure to PCBs.*

The question, as stated, requests that we focus principally on fish, avian, and mammalian species — and specifically the modeling parameters used to estimate current and future risks. In that regard I offer the following:

- *Avian and Mammalian Exposure Modeling Approach.* The oral dose models developed and applied within this ERA are consistent with current practice. Model parameterization — including the area use factors — are appropriate for the assessment of avian and mammalian risks along the Hudson River. Presentation of the oral dose models was well written and clear.
- *Projection of Future Risk Using HUDTOX/FISHRAND.* Fate and transport modeling is an important tool in evaluating the effects of remedial alternatives. While I not specifically reviewed the model documentation for HUDTOX, FISHRAND is based on the algorithms developed by Frank Gobas (1993), which have previously been applied to in the Great Lakes Water Quality Initiative, the Lower Fox River RI/FS, and for the Sheboygan River, WI Ecological Risk Assessment with good predictive success.
- *Appropriateness of Biomagnification Factors derived from the scientific literature.* In the absence of site specific information, using bioaccumulation or biomagnification factors derived from refereed scientific journals is appropriate, and consistent with current ERA practice.

Beyond these narrow questions, I offer the following observations and recommendations concerning the Section 3 — Exposure Assessment.

Page 40. Sect. 3.1.2 Estimating Future baseline Conditions

I understand the need for, and generally concur with the methodology presented in this section for estimating future TEQ risks from the FISHRAND output. What is not clear in this section was whether the mean, median, 90th percentile, or 95% UCLM was utilized for estimating the future TEQs. There is no discussion of whether there were sufficient data points to make this calculation sufficiently robust to be confident in the projections. Given that there are likely limited congener data, then I believe that the future risk analysis should be strengthened by estimating the probability distribution around the TEQ estimate, and calculating the future hazard quotients around that distribution.

Table 3.2, cited in this section, is difficult to understand. What is Value 1 fish vs. Value 2 fish, mammals, avians? Do these represent different trophic levels?

Page 40. Section 3.2 Observed Exposure Concentration

The ERA uses mean, median, 90th percentile, and 95% Upper Confidence Limit on the Mean for different endpoints. While this is clarified to some degree in the *Responsiveness Summary* (page 21, response to EF 1.9), there are still some outstanding questions in reviewing the exposure concentrations.

For example, were distributions for PCBs assumed to be lognormal, or were normality tests applied? The current ERA does discuss calculation of a 95% upper confidence limit on the mean (95% UCL), but omits a discussion of what happens when the 95% UCL exceeds the maximum concentration (see for example Table 3-7 where 95% UCLs for Brown Bullhead and Largemouth Bass exceed the maximum measured concentration). Was the maximum, or 95% UCL used for estimation of exposure in these situations? This section should also include rules for determining percentiles (which the current ERA omits). For example, what is the minimum number of samples needed to determine a percentile ($n > 10$), and what value is used when “n” data are not available.

Pages 41 - 44. Sects. 3.2.1 - 3.2.6. Observed Concentrations and associated Tables.

Table summarizing exposure concentrations based on measured observations currently only present the mean and an associated percentile or UCL. It would be useful to include in the tabular presentation of the number of samples (N), the minimum, the maximum value, and whether risk characterization is based upon the 95% UCL or maximum value.

Table 3-7 provides an example of the confusion over which values are used for risk characterization. For example, for Brown Bullhead at River Mile 189 (Federal Dam), the 95% UCL for 1993 exceeds the maximum wet weight value. This is also true for the 1993 lipid-normalized concentration for Largemouth Bass at RM 113, the 1993 and 1994 wet weight concentrations for White Perch, and the 1996 lipid-normalized white perch and the 1993 lipid-normalized yellow perch concentrations. Furthermore, for the purpose of clarity, it would be useful to have the percent lipids used in Table 3-7. It is not clear if the average lipid is divided into the average wet-weight PCBs (which I assume must be the case).

Finally, for fish exposure the ERA uses fillet PCB concentrations for estimating whole body risks. First, clarification would be useful to know if this is skin-on, or skin-off filets. Secondly, use of filets likely underestimates risks, as correctly pointed out in the EKA and the Uncertainty Section. Fillet-whole-body ratios for several fish species, including bluegill and largemouth bass, were used as part of the Clinch River Operable Unit Ecological Risk Assessment at Oak Ridge Tennessee, and were published in 1996. I would recommend that EPA consider looking at the values in the following reference for any future assessments at the Hudson:

Bevelhimer, MS, BE Sample, GR Southworth, JJ Beauchamp, and MJ Peterson. 1996.
Estimation of whole-fish contaminant concentrations from fish fillet data.
EA/ER/TM202. Oak Ridge National Laboratory, Oak Ridge, TN.

Table 3-10 of the ERA, and Table 3-10 of the *Responsiveness Summary*, reflect the transparency issue I have discussed previously. In ERA Table 3-10, there are projected values for whole water average and 95% UCL concentrations for 1993 through 2018. The difference between the predicted average water and the 95% UCL is negligible, but not discussed in the ERA. For the Thompson Island Pool, the concentrations in 1993 are identical, and only differ by 2×10^{-7} in 2018. For the Stillwater Reach, there are no differences. The same is fairly well true in Table 3-11 for sediments.

In Table 3-10 of the *Responsiveness Summary*, the 95% UCL calculations are dropped. There is no reflection of this in the text.

- 5) *Have the exposure assumptions (ERA, pp. 46-66 and Appendices D, E, and F) for each fish and wildlife receptor been adequately described and appropriately selected? Please discuss in detail.*

These were well written. I wish to commend the authors for Appendices C through G. I thought the life history sections were well researched and presented, and found myself enjoying reading those. I have no further comments here.

Effects Assessment

6) *For field-based toxicity studies, only a NOAEL toxicity reference value (TRV) was developed because other contaminants or stressors may be contributing to observed effects. Please comment on the validity of this approach. Also, please comment on whether the general approach of using uncertainty factors (interspecies, LOAEL-to-NOAEL, and subchronic-to-chronic) is appropriate in developing TRVs that are protective of Hudson River receptor species.*

- *Field-based NOAELs.* In general I concur that for most studies and receptor species, developing field-based NOAELs is appropriate. There are some exceptions, however, worth noting. Giesy et al (1994), and Tillitt et al (1992) developed effects-based regressions for piscivorous bird fecundity based upon field observations of TCDD-Eq and field effects using double crested cormorants and Caspian Terns from field data collected in the Great Lakes. These equations can be used to estimate a 20% or 30% field-based effect, as opposed to strictly a NOAEL.

Giesy, J, J Ludwig, and D Tillitt. 1994. Deformities in birds of the Great Lakes Region: Assigning causality. *Environ Sci. Technol.* 28: 128 - 135.

Tillitt, D., et al 1992. Polychlorinated biphenyl residues and egg mortality in double-crested cormorants from the Great Lakes. *Environ. Toxicol. Chem.* 11: 1281 - 1288.

- *Application of Uncertainty Factors.* This is a hotly debated topic within the risk community, and one that cannot be borne up by science, *per se*, but only by the respective opinions of the policy makers and risk managers at a site. If the narrow question is asked, "Are UFs common practice in establishing TRVs for ecological risk assessments?", the answer is yes. This includes interspecies UFs (that may range from 10 to 1,000), LOELs to NOELs, and subchronic to chronic. Complete discussions of the history and application of uncertainty factors may be found in Calabrese and Baldwin (1993), and in Chapman et al (1998).

I would caveat that, however, that in my own experience I have seen this done only for screening level ERAs, not baseline ERAs. My own opinion regarding the use of UFs for a baseline ERA is that they are not appropriate. I share the opinion expressed by Chapman *et al* that a UF applied to derive an NOAEL from an LOAEL is not appropriate for application to decisions that effect remedial alternative decisions. If the UFs are to be applied or TRVs for that matter), then they must be appropriately bracketed and placed in context.

For example, the ERA develops LOAEL and NOAEL TRVs for total PCBs in eggs of the Great Blue Heron by using the chicken fecundity value of Scott (1977). Those values are:

LOAEL	2.21 mg PCBs/kg egg
NOAEL	0.33 mg PCB/kg egg

In this case, interspecies UF or subchronic to chronic UFs were not applied, based on the known documentation of gallinaceous birds. The ERA states that there were no field studies that examined effects of PCBs to eggs of the great blue heron or birds of related taxonomy. Thus, no context was provided in the ERA.

Custer et al (1997) examined the effects of organochlorines (including PCBs), mercury and selenium on pipping of great blue heron eggs collected from 10 colonies on the upper Mississippi. These eggs were field collected, incubated in the laboratory, and a comprehensive set of chemical, EROD, egg-shell thinning, and hatching data were collected. While having a geometric mean PCB concentration of 2.9 mg/kg egg, the PCB concentrations in the embryos were too low to induced EROD activity, and those authors concluded that PCBs (and the other studied COPCs) did not "seem to be a serious threat to nesting GBHs" on the upper Mississippi. While other contaminants are involved in this study, context is applied in the sense that a more appropriate NOAEL might be closer to 2 mg/kg egg — which is the LOAEL proposed for the Hudson ERA.

Likewise, Halbrook et al (1999) measured PCB concentrations in field collected Great Blue Heron eggs from four colonies as part of the Clinch River ERA at the Oak Ridge, TN. While concentrations of Arochlor 1260 were measured at a mean of 2 mg/kg ww, there were no statistical differences in the number of chicks fledged per nest or in the mean weight of eggs or shell thickness between site, and reference site collections. Again, when placed in context with this study, the appropriate NOAEL for GBH along the Hudson River might be closer to 2 mg/kg ww.

I would point out that the Custer et al (1997) work supports the LOAEL/NOAEL for field-based TEQs in the GBH. The geometric mean TEQ for PCB congeners and PCDDs/PCDFs from the GBH eggs collected was 0.551 ug/kg TEQ (using the Kennedy et al chicken TEFs). This compares well with the values selected of 0.5 ug TEQ/kg egg, and 0.3 TEQ/kg egg, respectively.

Calabrese, E., and L. Baldwin. 1993. *Performing Ecological Risk Assessments*. Lewis Publishers. Chelsea, MI.

Chapman, P., A. Fairbrother, and D. Brown. 1998. A critical evaluation of safety (uncertainty) factors for ecological risk assessment. *Env. Toxicol. Chem* 17:99 - 108.

Custer, T.W., et al 1997. Contaminant concentrations and biomarker response in Great Blue Heron eggs from 10 colonies on the Upper Mississippi River, USA. *Env. Tox. Chem.* 16: 260 - 271.

Halbrook, R., L. Rober, and D. Buehler. 1999. Ecological risk assessment in a large river-reservoir: 7. Environmental contaminant accumulation and effects in great blue heron. *Env. Tox. Chem.* 18: 641 - 648.

Risk Characterization/Uncertainty Analysis

- 7) *USEPA calculated toxicity quotients (TQs) for all receptors of concern on both a total PCB and dioxin-like PCB (TEQ) basis. Please comment on whether the methodologies used in calculating these TQs are adequately protective of these receptors.*

The methodology of calculating toxicity quotients is consistent with current practice. There is a substantive body of evidence that support the used of the Toxicity Equivalent Quotient in evaluating risks to birds and mammals. As noted above, it is not the determination of the TEQ exposure concentration that effects the assessment of risk, it is the selection and defense of the appropriate Toxicity Reference Value that most greatly influences the process.

- 8) *The risk characterization section of the ERA (Chapter 5, pp. 117-151) summarizes current and future risks to fish and wildlife that may be exposed to PCBs in the Upper Hudson River and current risks to fish and wildlife in the Lower Hudson River. Please comment on whether the risk characterization adequately characterizes the relative risks to ecological receptors (e.g., piscivores, insectivores) posed by PCBs in the Hudson River.*

Benthic community structure

The assessment of baseline PCB risks as assessed in the ERA for benthic community structure are hampered by (1) the inconclusive results of infaunal community analysis, (2) omission of a discussion of the other COPCs, and (3) the lack of a complete presentation of the spatial extent of PCB levels in the non-1993 sampled sections of the Hudson River. As noted previously, the levels of lead, chromium, and mercury at the infaunal stations sampled are at levels that exceed several different sediment benchmarks, including both the threshold effects, and the probable effects concentrations, of the consensus-based sediment quality guidelines for freshwater ecosystems (MacDonald et al, *in press*). While I concur that the HQs using the sediment quality thresholds for infauna do indeed suggest a level of risk for infauna, the conclusion of risk is hampered by the fact that those HQs are for 19 stations over 200 miles of river.

Health and maintenance of local fish populations

This is an excellent data set, and the conclusions of potential risk appear to be supported by the data. The relative magnitude of the calculated HQs for baseline, and future risks, for most species appears to fairly low ($< \text{HQ of } 10$ for most species by the year 2018) — given the conservative assumptions built into this ERA. I would interpret the results to be consistent with the conclusion that the current lines of evidence indicate that the current and future PCB exposures are not of sufficient magnitude to prevent reproduction or recruitment.

I would still recommend, however, that the ERA incorporate what existing fish population data is available. For example, striped bass measured HQs (Table 5-36 of the *Responsiveness Summary*) are relatively low (at or near 1). Data are apparently available on striped bass populations; these should be used to examine the effects at least of these species.

Health and maintenance of insectivorous bird populations

Here to the data set is strong and supports the general conclusion that the lines of evidence indicate that current and future concentrations of PCBs are not of a sufficient magnitude to prevent reproduction of insectivorous bird species, especially as they are represented by tree swallows. While the debate may rage for years on just what does anomalous behavior in nesting birds mean from the standpoint of population effects, the conclusions of the ERA appear to be supported.

Health and maintenance of local waterfowl

These lines of evidence relies extensively on the use of modeled uptake, bioaccumulation factors derived from the scientific literature, conservative toxicity reference values, and predicts high TEQ-HQs for feeding female mallards and eggs throughout the modeling period. The use of any available field-population data would benefit the determination that mallards remain at risk currently, and throughout the modeling period.

Health and maintenance of local piscivorous bird populations

These lines of evidence relies extensively on the use of modeled uptake, bioaccumulation factors derived from the scientific literature, conservative toxicity reference values, and predicts high TEQ-HQs for both kingfishers and great blue herons throughout the modeling period. For GBH, the ERA should look again at the available scientific literature of toxicity reference values. Never-the-less, even using the NOAEL suggested in this review would still result in unacceptable risks (expressed as high HQs) currently, and throughout the modeling period.

Health and maintenance of endangered species

Like piscivorous birds, the lines of evidence for bald eagles relies extensively on the use of modeled uptake, bioaccumulation factors derived from the scientific literature, conservative toxicity reference values, and predicts high TEQ-HQs throughout the modeling period. The ERA should make better use of the excellent plasma data and population data provided in the *Responsiveness Summary*, pages 88 and 89. This is an excellent argument, with good field data, that should be placed directly into the risk assessment revision, or if the ERA is to be recompiled, directly into the exposure assessment.

Health and maintenance of local wildlife

The same arguments made previously could be applied here. Risks to mink and otter are probably supported by the existing data, but should be buttressed by any available habitat and population information.

Protection of Significant Habitats

This characterization is the hardest to define and defend. Significant habitats were never completely defined, and the use of sediment concentrations (from 19 stations over 200 miles of River) to suggest that these habitats are at risk is difficult to support. It is this reviewer's recommendation that the significant habitats of the Hudson River be mapped, that a definition of what is significant habitat be developed, and a set of measurement and endpoints be developed beyond the current two to evaluate this.

- 9) *The uncertainty analysis is presented in Chapter 6 of the ERA (pp. 153-165). Have the major uncertainties in the ERA been identified? Please comment on whether the uncertainties (and their effects on conclusions) in the exposure and effects characterization are adequately described.*

In general, the uncertainties for the data and TRVs used in this ERA are characterized in Chapter 6. However, given that there is no clear view in the reader's mind as to what the spatial extent of contamination is over 200 miles of river, there is considerable uncertainty unaccounted for in using sediment and benthic infaunal data from only 19 stations.

General Questions

- 1) *A goal for Superfund risk assessments is that they be clear, consistent, reasonable and transparent and adequately characterize risks to sensitive populations (e.g., threatened and*

endangered species). Based on your review, how adequate are the ERA and the Responsiveness Summary when measured against these criteria?

Please see previous comments on clarity and transparency.

- 1) *Please provide any other comments or concerns, both strengths and weaknesses, with the ERA not covered by the charge questions, above.*

Please see previous comments on report organization.

Recommendations

Based on your review of the information provided, please select your overall recommendation for the ERA and explain why.

1. *Acceptable as is*
 2. *Acceptable with minor revision (as indicated)*
 3. *Acceptable with major revision (as outlined)*
 4. *Not acceptable (under any circumstance).*
-

John Toll

Hudson River PCBs Site Reassessment RI/FS Ecological Risk Assessment Peer Review

Written Comments

John Toll

May 5, 2000

Specific Questions

Problem Formulation/Conceptual Model

- 1. Consistent with USEPA guidance on conducting ecological risk assessments (USEPA, 1997), the problem formulation step establishes the goals, breadth, and focus of the assessment. As part of the problem formulation step in the ERA, a site conceptual model was developed (Chapter 2.3, pp. 11-19). Please comment on whether the conceptual model adequately describes the different exposure pathways by which ecological receptors could be exposed to PCBs in the Hudson River. Was sufficient information provided on the Hudson River ecosystems so that appropriate receptor species could be selected for exposure modeling?*

Response to Conceptual Model Question

The conceptual model presented in Figure 2-4 of the Baseline Ecological Risk Assessment (BERA) adequately describes the different pathways by which ecological receptors could be exposed to PCBs in the Hudson River from the GE facilities, but it does not adequately describe the different pathways by which ecological receptors could be exposed to PCBs in the Hudson River from non-point sources. The conceptual model should include sources other than the GE facility, even if they are arguably insignificant. The model could be revised to show (qualitatively or quantitatively) the relative significance of different sources and exposure pathways, but should not exclude minor sources or pathways. This becomes an issue, for example, for the mink, because the BERA exposure assessment assumes the mink gets about half its food from non-river related sources.

Consistent with this last point, the conceptual model could be improved by adding sediment, water column and lower trophic level compartments that are not connected to the GE Facilities PCB source. This would better represent the possibility of non-river related diet sources. Again, this seems appropriate because one receptor (mink) has a significant non-river related diet source (49.5%) in its nominal exposure assumptions (Table 3-24).

Response to Receptor Species Question

I found the information presented in Section 2.6 of the BERA report, and Appendices C – F sufficient to demonstrate that the receptor species selected are sensible representatives of their respective trophic levels in the Hudson River ecosystem. What was less clear to me was the process whereby the assessment endpoints were selected that led to these receptors.

The selection of receptor species is in part a value decision that should flow from the assessment endpoints. Assessment endpoints should represent the values to be protected using information from the risk assessment. The values to be protected should reflect the views of USEPA, technical team members and interested and affected organizations, groups and individuals. USEPA and the technical team prepared the BERA. Therefore, I focused my review as it pertained to this question on how the views of interested and affected organizations, groups and individuals were solicited and incorporated into the receptor species selected for exposure modeling.

The BERA does a good job of identifying the interested and affected organizations, groups and individuals, how their views were solicited and incorporated into the problem formulation. Chapter 1 identifies the interested and affected organizations that were consulted with regard to the problem formulation. The second paragraph of chapter 2 describes the process by which the problem formulation was completed. In particular I noted the statement that most of the issues considered in the problem formulation were discussed with the interested and affected organizations during a number of technical and public meetings. It was not clear to what extent other interested and affected groups and individuals had the opportunity to observe these discussions because there was no statement about the content of the discussions at technical versus public meetings. Therefore, I do not have sufficient information to evaluate whether information provided on the Hudson River ecosystems during the problem formulation was sufficient for selecting appropriate receptor species.

Next I turned my attention to the BERA report itself, and evaluated whether the information provided *a posteriori*, in the report, was sufficient for selecting appropriate receptor species. The pertinent data for evaluating this are the public comments on the BERA and responses to these comments. I found the BERA Responsiveness Summary to be well organized and helpful for this evaluation. I found no comments suggesting that appropriate receptor species were excluded from the risk assessment.

In light of all these factors, it's my conclusion that sufficient information has been provided on the Hudson River ecosystems so that appropriate receptor species could be selected for exposure modeling.

Assessment and Measurement Endpoints

2. *Assessment endpoints specify the valued ecological resources to be protected, such as local fish populations. They focus the risk assessment on particular components of the ecosystem that could be adversely affected by contaminants from the site. Please comment on whether the assessment endpoints selected (pp. 19-20) adequately protect the important ecological resources of the Hudson River. Are major feeding groups and sensitive species sufficiently covered by the selected assessment endpoints?*

Please see my response to the receptor species portion of question #1.

3. *Please comment on whether the combination of measured, modeled, guideline, and observational measurement endpoints used in the ERA (pp. 20-29) supports the weight of evidence approach used in the ERA.*

While the weight-of-evidence concept is described at the beginning of Section 2.5, I did not find a description of the approach. Clearly, one element of the weight-of-evidence approach is the use of multiple, independent measurement endpoints (multiple lines of evidence) to evaluate assessment endpoints. The multiple measurement endpoints described in Section 2.5 really fell into two, more or less independent groups. Group 1 involves measured or predicted exposures (doses or concentrations, measured in total PCB or TEQ-based units) that were compared to effect thresholds by a quotient approach. Group 2 is comprised of the field observations on presence and relative abundance of receptor populations. This second group was not well enough defined for me to evaluate their suitability for use in the risk assessment. For example, there was no discussion of observational interpretation methods, of conditioning variables or of methods for accounting for confounding factors (see comments in Table 1). In the absence of further information about the Group 2 measurement endpoints, I have reservations about the ability to use the combination of measurement endpoints in a weight-of-evidence approach.

General comment – the definitions of measurement endpoints (measures of exposure and measures of effect) should be sufficiently specific for a reviewer, when provided with the raw data used by the risk assessor, to reproduce the exposure estimates and effect thresholds. Some of the measurement endpoints defined in Section 2.5 did not.

Specific comments on the measurement endpoints are provided in Table 1 (attached).

Exposure Assessment

4. *USEPA used several exposure models to evaluate the potential risks due to PCBs (see, ERA, pp. 37-71). Sampling data from USEPA, NOAA, NYSDEC, and USFWS collected from 1992-1996 were used to estimate current fish body burdens and dietary doses to avian and mammalian receptors. Future concentrations of PCBs were derived from USEPA's fate, transport, and bioaccumulation models, which are the subject of a separate peer review. Concentrations of PCBs in bird eggs were estimated by applying a biomagnification factor from the literature. Please comment on the appropriateness and sufficiency of this approach to estimate ecological exposure to PCBs.*

General Comments

I frequently found myself wanting more detail about the exposure analysis. For example, as I write this I'm looking at the introduction to Section 3.2, where it would have been very helpful to see the normality test results for the various subsets of the PCB concentration data used in the exposure assessment. These results may be contained in the baseline modeling report. I was expecting to receive a copy of that report but haven't yet; I apologize for any

oversight on my part that may have occurred. I would still like to see the baseline modeling report before the ecological risk assessment peer review meetings. (Please see also Response to Modeling Question below.)

Response to Sampling Data Question

It always seems to be true that reviewers come up with many specific questions about ecological risk assessment databases, and this one is no exception. Of course hindsight is always 20/20. Having said that, for the most part I found the sampling data to be appropriate and sufficient.

A specific area where I do have questions about the sufficiency of the data is in the mallard exposure assessment. Specifically, site-specific mallard diet information, and measurements of PCB concentrations in vegetation types consumed by mallards on the Hudson River seemingly would have been appropriate. This is in light of the relatively high contribution of vegetation to the mallard ADD (as reflected in Tables 3-30 and 3-31).

Response to Modeling Question

Estimating the PCB concentrations that were used to compute average daily doses is a fundamental element of the exposure assessment that is not covered in baseline modeling report, rather than the BERA report. Reviewing these estimates takes on greater significance because the sensitivity analysis for risk models (Section 6.5.2) identifies uncertainties in the PCB concentrations used to compute average daily doses as the most sensitive inputs to the toxicity quotient equations, for all avian and mammalian receptors.

A discussion of the ADD estimation results would have been helpful. All I found were summary tables of numerical results. The discussion could be placed in Chapter 3 (Exposure Assessment), Chapter 6 (Uncertainty Analysis) or a new exposure assessment appendix. Chapter 6 might be the best place for the discussion. Currently, the sensitivity and uncertainty analyses do not address exposure modeling in sufficient detail. For example, the sensitivity analysis for exposure models is lumped into the brief (three-paragraph) section on sensitivity analysis for risk models (Section 6.5.2).

I found contradictory statements about the assumed dietary composition for mallards. On page 54, end of the first paragraph, the diet is described as 50% aquatic invertebrates and 50% vegetation. On page 162, in the last sentence of the second-to-last paragraph, the diet is described as 70% aquatic invertebrates.

Response to Biomagnification Factor Question

The decision to use the BMF published by Giesy *et al.* (1995) may be defensible, but it has not been defended in the BERA report. BMFs are empirical constants, so it is important that any time a non-site specific literature value is used, that use be defended. Issues that should be considered include:

- similarity of the PCB mixtures in the literature study and at the site,
- similarity of avian species (Giesy *et al.* is a bald eagle study),
- similarity of fish PCB body burdens in the literature study and at the site,
- similar quality of dietary exposure concentration estimates in the literature study and at the site,
- similarity of exposure levels from other significant exposure pathways,
- availability of other literature BMFs (subject to the same sort of evaluation).

Even limited corroborative evidence – in the form of paired site-specific dietary exposure and egg concentration data – would be very useful for evaluating the appropriateness and sufficiency of the biomagnification factors used in the BERA.

5. *Have the exposure assumptions (ERA, pp. 46-66 and Appendices D, E, and F) for each fish and wildlife receptor been adequately described and appropriately selected? Please discuss in detail.*

Modeled Water Concentrations

Specific HUDTOX modeling assumptions and parameters are not presented in the BERA report, so I cannot comment on whether they were appropriately selected. I hope to be able to review the baseline modeling report as it pertains to the BERA peer review questions before the peer review meetings.

Modeled Sediment Concentrations

Specific HUDTOX modeling assumptions and parameters are not presented in the BERA report, so I cannot comment on whether they were appropriately selected. I hope to be able to review the baseline modeling report as it pertains to the BERA peer review questions before the peer review meetings.

Modeled Benthic Invertebrate Concentrations

Sections 3.3.1.3 and 3.4.1 indicate that invertebrate PCB concentrations were estimated by the product of sediment concentration and a biota-sediment accumulation factor (BSAF). This approach is adequate (i.e., it's a reasonable conceptual approach), but I cannot comment on whether the BSAF was selected appropriately because I did not find a description of the specific modeling assumptions in the BERA.

Detailed documentation should be added describing how BSAF was estimated from sediment and invertebrate PCB concentration data. Presumably BSAF was derived using co-located

data from the ecological sampling stations, and used to predict aquatic invertebrate concentrations in other locations. Plots of BSAF versus sediment concentration and versus sampling station location (river mile or segment) would be useful for evaluating the BSAF selected.

Modeled Fish Concentrations

Specific FISHRAND modeling assumptions and parameters are not presented in the BERA report, so I cannot comment on whether they were appropriately selected. I hope to be able to review the baseline modeling report as it pertains to the BERA peer review questions before the peer review meetings.

Benthic Exposure Pathways

See comments under the heading "Modeled Benthic Invertebrate Concentrations."

Sections 3.3.1.3 and 3.4.1 are redundant and I recommend they be merged.

Fish Exposure to Surface Water Sources of PCBs

See comments under the heading "Modeled Fish Concentrations."

I would merge Section 3.4.2 with Section 3.3.1.4.

The last sentence in the first paragraph of Section 3.4.2.1 seems tautological in that slower depuration that uptake is necessary for bioaccumulation to occur.

Fish Exposure to Sediment Sources of PCBs

See comments under the heading "Fish Exposure to Surface Water Sources of PCBs."

Avian Surface Water Ingestion

Three avian parameters are introduced in Section 3.4.3.1: normalized water ingestion rate (*NWI*), areal forage effort (*FE*) and body weight (*BW*):

- *NWI*, calculated using an equation from the USEPA *Wildlife Exposure Factors Handbook*, is adequately described and appropriately selected.
- *FE* is adequately described. Setting *FE* = 1 is arguably appropriate, although one could also argue that some portion of avian receptor populations' diets and ingested waters come from upstream of Hudson Falls or from surface waters off the main stem of the Hudson River. A discussion of this assumption (*FE* = 1) should be added to Chapter 6 (Uncertainty Analysis).
- *BW* is adequately described in Appendix E and appropriately selected.

The model for average daily dosage from surface water ingestion (equation 3-5) is adequately described and appropriately selected.

Avian Incidental Sediment Ingestion

Two avian parameters are introduced in Section 3.4.3.2: fraction of abiotic media in the diet (*FS*) and total food ingestion rate (*NIR*):

- *FS* is adequately described and appropriately selected.
- *NIR*, calculated using an equation from the USEPA *Wildlife Exposure Factors Handbook*, is adequately described and appropriately selected. The switch in nomenclature from *NIR* to *FI* (top of page 50) is a little bit confusing.

The model for average daily dosage from incidental sediment ingestion (equation 3-7) is adequately described and appropriately selected.

Avian Dietary Exposure

Several avian parameters are introduced in Section 3.4.3.3:

- The first three: normalized field metabolic rate (*NFMR*), metabolizable energy (*ME*) and gross energy content of dietary component (*GE*), all based on the USEPA *Wildlife Exposure Factors Handbook*, are adequately described and appropriately selected.
- Assimilation efficiency (*AE*) often is the most sensitive parameter in bioaccumulation models, so greater justification for the selected values is appropriate. A section on assimilation efficiency uncertainty should be added to Chapter 6 (Uncertainty Analysis).
- Dietary fractions (*PD*), derived from the scientific literature and from consultations with NYSDEC and USFWS staff, also are adequately described and appropriately selected.
- Dietary fractions for the tree swallow are based on the work of McCarty and Winkler (in press). Some version of that work should be made available for peer review, since it is the basis for exposure assumptions used in the BERA.

The models for average daily dosage from fish consumption (equation 3-11) and invertebrate consumption (equation 3-12) are adequately described and appropriately selected.

The use of the model for PCB concentration in macrophytes (equation 3-13) was the subject of comments on the exposure assessment, under the heading "Response to Sampling Data Question." At a minimum, it would be useful to obtain a small amount of coincident PCB concentration data in water (dissolved PCB concentration) and plants consumed by waterfowl on the Hudson River, to corroborate the predictions of equation 3-13. Better yet would be to obtain site-specific mallard diet information, and measurements of PCB

concentrations in vegetation types consumed by mallards on the Hudson River.

Avian Behavioral and Temporal Modifying Factors Relating to Exposure

See comments on areal forage effort (*FE*) under the heading "Avian Surface Water Ingestion."

Biomagnification Factors for Predicting Egg Concentrations

See comments on this topic under the heading "Response to Biomagnification Factor Question."

Mammalian Surface Water Ingestion

Same comments as for avian surface water ingestion.

Mammalian Incidental Sediment Ingestion

Same comments as for avian incidental sediment ingestion.

Mammalian Dietary Exposure

Several avian parameters are introduced in Section 3.4.4.3:

- The first three: normalized field metabolic rate (*NFMR*), metabolizable energy (*ME*) and gross energy content of dietary component (*GE*), all based on the USEPA *Wildlife Exposure Factors Handbook*, are adequately described and appropriately selected.
- Assimilation efficiency (*AE*) often is the most sensitive parameter in bioaccumulation models, so greater justification for the selected values is appropriate. A section on assimilation efficiency uncertainty should be added to Chapter 6 (Uncertainty Analysis).
- Dietary fractions (*PD*), derived from the scientific literature and from consultations with NYSDEC and USFWS staff, also are adequately described and appropriately selected.

The models for average daily dosage from fish consumption (equation 3-22) and invertebrate consumption (equation 3-23) are adequately described and appropriately selected.

Mammalian Behavioral and Temporal Modifying Factors Relating to Exposure

See comments on areal forage effort (*FE*) under the heading "Avian Surface Water Ingestion."

Effects Assessment

6. For field-based toxicity studies, only a *NOAEL* toxicity reference value (*TRV*) was developed

because other contaminants or stressors may be contributing to observed effects. Please comment on the validity of this approach. Also, please comment on whether the general approach of using uncertainty factors (interspecies, LOAEL-to-NOAEL, and subchronic-to-chronic) is appropriate in developing TRVs that are protective of Hudson River receptor species.

Question on Use of Field-Based Toxicity Studies

In general, I would not use NOAELs generated from field-based toxicity studies to derive TRVs if factors that may be confounding the measurement of a PCB dose-response relationship cannot be controlled. A better choice is to use laboratory-based toxicity studies to derive TRVs, and use the field data (without censoring the observed effects portion of the database) in a weight of-evidence approach.

Question on Use of Toxicological Uncertainty Factors

I will in defer to the peer review team's wildlife toxicologists on this question.

Risk Characterization/Uncertainty Analysis

- 7 *USEPA calculated toxicity quotients (TQs) for all receptors of concern on both a total PCB and dioxin-like PCB (TEQ) basis. Please comment on whether the methodologies used in calculating these TQs are adequately protective of these receptors.*

The methodologies used in calculating the TQs appear to have sufficient uncertainty factors built in (both for exposure and toxicity) to ensure that the probability of a false negative ($TQ < 1$ when risk from PCB exposures is present) is low.

The BERA only looks at baseline risks, so the question of risks to ecological receptors from remedial actions driven by false positives ($TQ > 1$ when risk from PCB exposures is absent) does not apply in Phase 2, although it will apply in Phase 3, where, as stated in the charge to the risk assessment peer reviewers:

"the risk assessments will be used in the Feasibility Study to back-calculate to appropriate levels of PCBs in fish to compare various remedial alternatives, including the No Action alternative (i.e., baseline conditions) required by federal Superfund law."

Therefore, when this risk assessment is used in Phase 3, it will be important to evaluate both false positive and false negative probabilities, as well as the potential consequences of false positive and false negative results on the remedial action decision.

8. *Please comment on whether the risk characterization adequately characterizes the relative risks to ecological receptors (e.g., piscivores, insectivores) posed by PCBs in the Hudson River.*

It is likely that the risk characterization adequately characterizes the relative risks to different trophic level receptors (e.g., piscivores, insectivores) because of the bioaccumulative nature of PCBs.

9. *The uncertainty analysis is presented in Chapter 6 of the ERA (pp. 153-165). Have the major uncertainties in the ERA been identified? Please comment on whether the uncertainties (and their effects on conclusions) in the exposure and effects characterization are adequately described.*

I am not confident that the major uncertainties in the BERA have been identified. I would like to have seen much greater discussion of model error (Section 6.5.3). In particular, the statement at the top of page 165, "(i)n this assessment, model error is probably not a significant source of uncertainty" is a sweeping statement that needs to be substantiated. Also, Section 6.5.3.1 provides a very brief summary of the uncertainty analysis for FISHRAND model predictions. I was surprised not to find an equivalent section for the HUDTOX uncertainty analysis.

I would have liked to have seen much more extensive sensitivity and uncertainty analyses, although it is really in Phase 3 of the Reassessment RI/FS that they will be needed. For Phase 2, the uncertainties and their effects on conclusions arguably are adequately described, though the uncertainty analysis is minimal. The sensitivity and uncertainty analyses I would like to have seen, and that I think will be needed in Phase 3, would systematically review each data set and model that feeds into the ecological risk estimates, including HUDTOX and FISHRAND. Each review would explicitly answer the following questions:

How well do the estimators derived from the (data set or model) represent the intended parameter needed for the risk assessment?

For those estimators with significant uncertainties, what is the cause of the uncertainty and how could it be reduced?

The uncertainty analysis results then would be rolled up to produce probability distributions on levels of PCBs in fish (for baseline conditions in Phase 2, for each remedial alternative in Phase 3), from which the probability of exceeding appropriate levels of PCBs in fish could be calculated.

General Questions

1. *A goal for Superfund risk assessments is that they be clear, consistent, reasonable and transparent and adequately characterize risks to sensitive populations (e.g., threatened and endangered species). Based on your review, how adequate are the ERA and the responsiveness summary when measured against these criteria?*

The BERA is comparable on these criteria to other ecological risk assessments I have reviewed.

2. *Please provide any other comments or concerns, both strengths and weaknesses, with the ERA not covered by the charge questions, above.*

No additional comments.

Recommendations

1. *Based on your review of the information provided, please select (from among the following) your overall recommendation for the ERA and explain why (this is your overall recommendation):*

- *acceptable as is*
- *acceptable with minor revision (as indicated)*
- *acceptable with major revision (as outlined)*
- *not acceptable (under any circumstance).*

My overall recommendation for the Phase 2 Baseline Ecological Risk Assessment is that it is acceptable with minor revisions (as indicated in my comments), with the caveat that major revisions will likely be needed before the risk assessment can be used for the purposes of Phase 3, as it is explained in the third paragraph of the Charge for Peer Review 4.

Table 1

Assessment Endpoint	Measurement Endpoint	Comments
Benthic community structure as a food source for local fish and wildlife	1a Field observations of benthic community abundance in relation to measured PCB concentrations and habitat characteristics	The measures of community abundance and habitat characteristics are undefined.
	1b Field observations of benthic community composition in relation to measured PCB concentrations and habitat characteristics	This is a general comment that applies to many of the measurement endpoints. The measure of PCB concentrations isn't clearly stated in Section 2.5. My assumption is that it's tri-phenyl concentration (unless the measurement endpoint specifically says TEQ-based concentration), but I don't think these concepts are introduced until Chapter 3.
	2 Measured and modeled average and 95% UCL PCB concentrations in water (fresh and saline) compared to NYSAWQC for the protection of benthic aquatic life or wildlife	The measures of community composition and habitat characteristics are undefined.
	3 Measured and modeled average and 95th percentile UCL PCB concentrations in sediment compared to sediment benchmarks such as NOAA sediment effect concentrations for PCBs in the Hudson River, NYSDC Technical Guidance for Screening Contaminated Sediments, Perreud et al. (1993), Ingerson et al. (1999), Washington Department of Ecology (1997), and Jones et al. (1997) for protection of aquatic life	This is a general comment that applies to many of the measurement endpoints. Is this the 95% UCL on the mean or on some other population statistic?
		Is this by river segment by year? What level of spatial and temporal averaging of the water concentration data?
		Are the AWQCs both acute and chronic? Are the water concentration averaging periods different for comparing to acute and chronic AWQC?
		Are the PCB water concentrations water column or pore water?
		Why is comparison of water concentrations to wildlife AWQC included under this assessment endpoint?
		Should be specific about the sediment benchmarks to which concentrations are compared. Is this the list or are these just possible examples. Also, do you compare to all the benchmarks, or are they ranked in terms of applicability and the most applicable benchmark used?
		Washington State sediment management standards are for Puget Sound sediments and wouldn't be applicable to the Hudson River.

Assessment Endpoint Protection and maintenance (i.e., survival, growth and reproduction) of local fish populations (forage, omnivorous, piscivorous)	Measurement Endpoint 1 Measured and modeled median and 95th percentile PCB body burdens in fish for each river segment over 25 years to determine exceedence of effect-level thresholds based on TRVs derived in Chapter 4 2 Measured and modeled TEQ-based median and 95th percentile PCB body burdens in fish for each river segment over 25 years to determine exceedence of effect-level thresholds based on TRVs derived in Chapter 4 3 Measured and modeled TEQ-based median and 95th percentile PCB concentrations in water (fresh and saline) compared to NYSAWQC for the protection of benthic aquatic life 4 Measured and modeled average and 95th percentile UCL PCB concentrations in sediment compared to sediment benchmarks such as NOAA sediment effect concentrations for PCBs in the Hudson River, NYSDC Technical Guidance for Screening Contaminated Sediments, Persaud <i>et al.</i> (1993), Ingersoll <i>et al.</i> (1996), Washington Department of Ecology (1997), and Jones <i>et al.</i> (1997) for protection of aquatic life 5 Available field observations on the presence and relative abundance of fish species within the Hudson River for each river segment as an indication of the ability of the species to maintain populations	Comments Are the data stratified by age or size class? Whole body or fillet body burdens? Wet weight or dry weight? same as above Why compared to criteria for protection of benthos? Is this by river segment by year? What level of spatial and temporal averaging of the water concentration data? Are the AWQCs both acute and chronic? Are the water concentration averaging periods different for comparing to acute and chronic A/V/QC? Which river segments are treated as saline? Filtered or unfiltered water? repeat; see above For each species, what's the threshold for ability or inability to maintain a population? Are there any conditioning variables on the threshold for ability or inability to maintain a population (e.g., time of year, carrying capacity)? Any confounding factors (e.g., stocking, habitat quality, habitat abundance, presence/absence of predators or competing species, etc.)?
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Table 1

Assessment Endpoint Protection and maintenance (i.e., survival, growth and reproduction) of local insectivorous birds	Measurement Endpoint 1. Modeled total average and 95% UCL PCB dietary doses to the free swallow to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	Comments The measure of exposure is expressed as a dose. Presumably the TRV is expressed as an egg concentration. Both should be in the same units (i.e., both concentrations or both doses, both wet weight or both dry weight etc.). Where can the reader find the model for predicting egg concentration from dose? Is exposure during the nesting season by year for 25 years, by river segment? Does total PCB mean fit? 95% UCL on what (e.g., mean, median, 95th %ile, etc.)? What's the benchmark for adverse effects (e.g., EC10, EC50, LOEC, NOEC)? How does this differ from the first measurement endpoint for protection and maintenance of local insectivorous birds? The numerator (measure of exposure) and denominator (effect-level threshold) must be in the same units (dose or concentration); and converting between dose and concentration won't change the ratio. 95% UCL on what (e.g., mean, median, 95th %ile, etc.)? What's the benchmark for adverse effects (e.g., EC10, EC50, LOEC, NOEC)? How does this differ from the first measurement endpoint for protection and maintenance of local insectivorous birds? How does this differ from the second measurement endpoint for protection and maintenance of local insectivorous birds? 4D24 Just the receptor (free swallow) or other species as well? For each species, what's the threshold for ability or inability to maintain a population? Are there any confounding variables on the threshold for ability or inability to maintain a population (e.g., time of year, carrying capacity)? Any confounding factors (e.g., quality or abundance of habitat, presence/absence of predators or competing species, etc.)?
	2. Modeled TEQ-based average and 95% UCL PCB dietary doses to the free swallow for each river segment over 25 years to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	The measure of exposure is expressed as a dose. Presumably the TRV is expressed as an egg concentration. Both should be in the same units (i.e., both concentrations or both doses, both wet weight or both dry weight etc.). Where can the reader find the model for predicting egg concentration from dose?
	3. Modeled total average and 95% UCL PCB concentrations in free swallow eggs to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	How does this differ from the first measurement endpoint for protection and maintenance of local insectivorous birds? The numerator (measure of exposure) and denominator (effect-level threshold) must be in the same units (dose or concentration); and converting between dose and concentration won't change the ratio.
	4. Modeled TEQ-based average and 95% UCL PCB dietary doses concentrations in free swallow eggs for each river segment over 25 years to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	How does this differ from the second measurement endpoint for protection and maintenance of local insectivorous birds?
	5. Modeled and measured average and 95% UCL PCB concentrations in water (freshwater and saline) compared to NYSAWQC for the protection of wildlife	4D24 Just the receptor (free swallow) or other species as well? For each species, what's the threshold for ability or inability to maintain a population? Are there any confounding variables on the threshold for ability or inability to maintain a population (e.g., time of year, carrying capacity)? Any confounding factors (e.g., quality or abundance of habitat, presence/absence of predators or competing species, etc.)?
	6. Available field observations on the presence and relative abundance of insectivorous bird species within the Hudson River for each river segment as an indication of the ability of the species to maintain populations	

Assessment Endpoint Protection and maintenance (i.e., survival, growth and reproduction) of local waterfowl	Measurement Endpoint 1 Modeled total average and 95% UCL PCB dietary doses to the mallard duck to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	Comments The measure of exposure is expressed as a dose. Presumably the TRV is expressed as an egg concentration. Both should be in the same units (i.e., both concentrations or both doses, both wet weight or both dry weight etc.). Where can the reader find the model for predicting egg concentration from dose? Is exposure by year for 25 years, by river segment? Is it cumulative or nesting season? Does total PCB mean fit? 95% UCL on what (e.g., mean, median, 95th %ile, etc.)? What's the benchmark for adverse effects (e.g., EC10, EC50, LOEC, NOEC)? The measure of exposure is expressed as a dose. Presumably the TRV is expressed as an egg concentration. Both should be in the same units (i.e., both concentrations or both doses, both wet weight or both dry weight etc.). Where can the reader find the model for predicting egg concentration from dose? 95% UCL on what (e.g., mean, median, 95th %ile, etc.)? What's the benchmark for adverse effects (e.g., EC10, EC50, LOEC, NOEC)? How does this differ from the first measurement endpoint for protection and maintenance of local waterfowl? The numerator (measure of exposure) and denominator (effect-level threshold) must be in the same units (dose or concentration); and converting between dose and concentration won't change the ratio. How does this differ from the second measurement endpoint for protection and maintenance of local waterfowl? Which river segments are treated as saline? Does whole water mean unfiltered? Just the receptor (mallard) or other species as well? For each species, what's the threshold for ability or inability to maintain a population? Does presence indicate the ability to maintain a population? How are non-resident waterfowl accounted for? Are there any conditioning variables on the threshold for ability or inability to maintain a population (e.g., time of year, carrying capacity)? Any confounding factors (e.g., quality or abundance of habitat, presence/absence of predators or competing species, etc.)?
	2 Modeled TEQ-based average and 95% UCL PCB dietary doses to the mallard duck for each river segment over 25 years to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	
	3 Modeled total average and 95% UCL PCB concentrations in mallard duck eggs to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	
	4 Modeled TEQ-based average and 95% UCL PCB dietary doses concentrations in mallard duck eggs for each river segment over 25 years to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	
	5 Modeled and measured average and 95% UCL PCB concentrations in whole water (freshwater and saline) compared to NYSAWQC for the protection of wildlife	
	6 Available field observations on the presence and relative abundance of duck species within the Hudson River for each river segment as an indication of the ability of the species to maintain populations	

Table 1

Assessment Endpoint Protection and maintenance (i.e., survival, growth and reproduction) of local piscivorous birds	Measurement Endpoint	Comments
1 Modeled total average and 95% UCL PCB dietary doses to the belted lingfisher, great blue heron and bald eagle to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	1 Modeled total average and 95% UCL PCB dietary doses to the belted lingfisher, great blue heron and bald eagle to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	The measure of exposure is expressed as a dose. Presumably the TRV is expressed as an egg concentration. Both should be in the same units (i.e., both concentrations or both doses, both wet weight or both dry weight etc.). Where can the reader find the model for predicting egg concentration from dose? Is exposure by year for 25 years, by river segment? Is it cumulative or nesting season? Does total PCB mean fit? 95% UCL on what (e.g., mean, median, 95th %ile, etc.)? What's the benchmark for adverse effects (e.g., EC10, EC50, LOEC, NOEC)?
2 Modeled TEQ-based average and 95% UCL PCB dietary doses to the belted lingfisher, great blue heron and bald eagle for each river segment over 25 years to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	2 Modeled TEQ-based average and 95% UCL PCB dietary doses to the belted lingfisher, great blue heron and bald eagle for each river segment over 25 years to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	The measure of exposure is expressed as a dose. Presumably the TRV is expressed as an egg concentration. Both should be in the same units (i.e., both concentrations or both doses, both wet weight or both dry weight etc.). Where can the reader find the model for predicting egg concentration from dose? 95% UCL on what (e.g., mean, median, 95th %ile, etc.)? What's the benchmark for adverse effects (e.g., EC10, EC50, LOEC, NOEC)?
3 Modeled total average and 95% UCL PCB concentrations in belted lingfisher, great blue heron and bald eagle eggs to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	3 Modeled total average and 95% UCL PCB concentrations in belted lingfisher, great blue heron and bald eagle eggs to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	How does this differ from the first measurement endpoint for protection and maintenance of local piscivorous birds? The numerator (measure of exposure) and denominator (effect-level threshold) must be in the same units (dose or concentration); and converting between dose and concentration won't change the ratio.
4 Modeled TEQ-based average and 95% UCL PCB dietary doses concentrations in belted lingfisher, great blue heron and bald eagle eggs for each river segment over 25 years to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	4 Modeled TEQ-based average and 95% UCL PCB dietary doses concentrations in belted lingfisher, great blue heron and bald eagle eggs for each river segment over 25 years to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	How does this differ from the second measurement endpoint for protection and maintenance of local piscivorous birds?
5 Modeled and measured average and 95% UCL PCB concentrations in water (freshwater and saline) compared to NYSAWQC for the protection of wildlife	5 Modeled and measured average and 95% UCL PCB concentrations in water (freshwater and saline) compared to NYSAWQC for the protection of wildlife	Which river segments are treated as saline? +D24
6 Available field observations on the presence and relative abundance of piscivorous bird species within the Hudson River for each river segment as an indication of the ability of the species to maintain populations	6 Available field observations on the presence and relative abundance of piscivorous bird species within the Hudson River for each river segment as an indication of the ability of the species to maintain populations	Just the receptors (belled lingfisher, great blue heron and bald eagle) or other species as well? For each species, what's the threshold for ability or inability to maintain a population? For example, does absence of a bald eagle (or nesting pair) in a river segment indicate an inability to maintain a population? Are there any conditioning variables on the threshold for ability or inability to maintain a population (e.g., time of year, carrying capacity)? Any confounding factors (e.g., quality or abundance of habitat, presence/absence of predators or competing species, etc.)?

Table 1

Assessment Endpoint	Measurement Endpoint	Comments
Protection and maintenance (i.e., survival, growth and reproduction) of local wildlife	1 Modeled total average and 95% UCL PCB dietary doses to the little brown bat, racoon, mink and otter to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	<p>The measure of exposure is expressed as a dose. Presumably the TRV is expressed as an body burden. Both should be in the same units (i.e., both concentrations or both doses, both wet weight or both dry weight etc.). Should indicate where can the reader find the model for predicting body burden from dose.</p> <p>Is exposure by year for 25 years, by river segment? For reproductive endpoints, is exposure cumulative or during the breeding season?</p> <p>Does total PCB mean tri+?</p> <p>95% UCL on what (e.g., mean, median, 95th %ile, etc.)?</p> <p>What's the benchmark for adverse effects (e.g., EC10, EC50, LOEC, NOEC)?</p>
	2 Modeled TEQ-based average and 95% UCL PCB dietary doses to the little brown bat, racoon, mink and otter for each river segment over 25 years to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	<p>The measure of exposure is expressed as a dose. Presumably the TRV is expressed as an body burden. Both should be in the same units (i.e., both concentrations or both doses, both wet weight or both dry weight etc.). Should indicate where can the reader find the model for predicting body burden from dose.</p> <p>95% UCL on what (e.g., mean, median, 95th %ile, etc.)?</p> <p>What's the benchmark for adverse effects (e.g., EC10, EC50, LOEC, NOEC)?</p>
	3 Measured total PCB concentrations in the liver of mink and otter	<p>Why no effect-level threshold?</p> <p>Wet or dry weight?</p>
	4 Modeled and measured average and 95% UCL PCB concentrations in water (freshwater and saline) compared to NYSAWQC for the protection of wildlife	<p>Which river segments are treated as saline?</p> <p>Filtered or unfiltered water?</p>
	5 Available field observations on the presence and relative abundance of wildlife species within the Hudson River for each river segment as an indication of the ability of the species to maintain populations	<p>Just the receptors (little brown bat, racoon, mink and otter) or other species as well?</p> <p>For each species, what's the threshold for ability or inability to maintain a population? Does absence from a river segment indicate inability to maintain a population?</p> <p>Are there any conditioning variables on the threshold for ability or inability to maintain a population (e.g., time of year, carrying capacity)?</p> <p>Any confounding factors (e.g., quality or abundance of habitat, presence/absence of predators or competing species, etc.)?</p>

Table 1

Assessment Endpoint Protection of threatened and endangered species	Measurement Endpoint	Comments
Protection of threatened and endangered species	1 Modeled total median and 95th %ile PCB body burdens in shortnose sturgeon using surrogate upper trophic level fish species to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	Is exposure by year for 25 years, by river segment? Does total PCB mean fit?
		95% UCL on what (e.g., mean, median, 95th %ile, etc.)?
		What's the benchmark for adverse effects (e.g., EC10, EC50, LOEC, NOEC)?
	2 Modeled TEQ-based median and 95th %ile PCB body burdens in shortnose sturgeon using surrogate upper trophic level fish species to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	Is exposure by year for 25 years, by river segment?
		95% UCL on what (e.g., mean, median, 95th %ile, etc.)?
		What's the benchmark for adverse effects (e.g., EC10, EC50, LOEC, NOEC)?
	3 Modeled total PCB dietary doses to the bald eagle to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	Subset of the first measurement endpoint for piscivorous birds; see comments above
	4 Modeled TEQ-based PCB dietary doses to the bald eagle to determine exceedance of effect-level thresholds based on TRVs derived in Chapter 4	Subset of the second measurement endpoint for piscivorous birds; see comments above
	5 Modeled and measured average and 95th UCL PCB concentrations in water (freshwater and saline) compared to NYSAWQC for the protection of wildlife	Same as fourth measurement endpoint for piscivorous birds; see comments above
	6 Measured and modeled average and 95th percentile UCL PCB concentrations in sediment compared to sediment benchmarks such as NOAA sediment effect concentrations for PCBs in the Hudson River, NYDEC Technical Guidance for Screening Contaminated Sediments, Penseud <i>et al.</i> (1993), Ingersoll <i>et al.</i> (1996), Washington Department of Ecology (1997), and Jones <i>et al.</i> (1997) for protection of aquatic life	repeat; see above

Table 1

Assessment Endpoint Protection of significant habitats	Measurement Endpoint 1 Measured and modeled PCB concentrations in water (freshwater and saline) compared to NYSAWQC for the protection of benthic aquatic life or wildlife	Comments What statistics on water concentrations? Which river segments are treated as saline? Filtered or unfiltered water?
	2 Measured and modeled PCB concentrations in sediment compared to applicable sediment benchmarks such as NOAA sediment effect concentrations for PCBs in the Hudson River, NYSDEC Technical Guidance for Screening Contaminated Sediments, Pensaud <i>et al.</i> (1993), Ingersoll <i>et al.</i> (1996), Washington Department of Ecology (1997), and Jones <i>et al.</i> (1997) for protection of aquatic life	What statistics on sediment concentrations? Should be specific about the sediment benchmarks to which concentrations are compared. Is this the list or are these just possible examples. Also, do you compare to all the benchmarks, or are they ranked in terms of applicability and the most applicable benchmark used? Washington State sediment management standards are for Puget Sound sediments and wouldn't be applicable to the Hudson River.