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09/16/2009 10:33 AM

To Docket ORD@EPA

cc Cynthia Dougherty/DC/USEPA/US@EPA, Pamela Barr/DC/USEPA/US@EPA, Stig Regli/DC/USEPA/US@EPA

bcc

Subject Probabilistic Risk Assessment White Papers, Docket ID No. EPA-HQ-ORD-2009-0645

Docket ID No. EPA-HW-ORD-2009-0645

Enclosed are the comments of the American Water Works Association (AWWA) on the two White Papers on Using Probabilistic Methods to Enhance Role of Risk Analysis in Decision-Making.

If you have any questions about these comments, please feel free to call or email. Alan

J. Alan Roberson, P.E.

Director of Security and Regulatory Affairs

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- PRA White Papers Comments Final 91609.pdf

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September 16, 2009

U.S. Environmental Protection Agency
Office of Research and Development
1200 Pennsylvania Avenue NW
Washington, DC

RE: Probabilistic Risk Assessment (PRA) White Papers
Docket No. EPA-HQ-ORD-2009-0645

Dear Docket:

The American Water Works Association (AWWA) is pleased to have the opportunity to comment on the above referenced white papers, as these are important issues in the regulatory development process. AWWA is an international, nonprofit, scientific and educational society dedicated to the improvement of drinking water quality and supply. Founded in 1881, the Association is the largest organization of water supply professionals in the world. Our 57,000 members represent the full spectrum of the drinking water community: treatment plant operators and managers, environmental advocates, scientists, academicians, and others who hold a genuine interest in water supply and public health. Our membership includes more than 4,600 utilities that supply roughly 80 percent of the nation's drinking water.

AWWA and its member utilities are dedicated to providing safe drinking water to the American public, and the drinking water community recognizes the importance of setting health-based standards that are balanced against the need to keep drinking water affordable. AWWA commends EPA for the development of these two white papers and its flexible approach in the use of probabilistic risk assessment (PRA) in the regulatory development process. AWWA has previously supported the use of PRA in our comments on several proposed national primary drinking water regulations (NPDWRs) in the past several years, and there is no need to restate detailed comments that can be found in those rulemaking dockets. In those comments, AWWA, building on recommendations from EPA’s Science Advisory Board (SAB), has supported the incorporation of probabilistic methods into EPA’s decision making process. These tools provide the Agency’s decision makers with a more realistic view of a range of the risks, as opposed to a single, central point estimate. A single point estimate can be appealing to decision makers, as a single number tends to imply clear outcomes, i.e., a single point estimate of risk reduction benefits is either unambiguously greater than or less than the estimated cost of compliance.

In a 2002 report, *Quantifying Public Health Risk Reduction Benefits*, published by the Water Research Foundation (formerly AwwaRF), researchers discussed a range of measures and decision criteria that should be explored and considered as the PRA is used in a probabilistic benefit-cost analysis. This report presented a case study on portraying probabilistic benefit-cost results for MTBE.

In addition, the use of PRA helps avoid the persistent problem whereby risk assessments are driven by a sequence of several conservative assumptions, each of which is intended to ensure that the risk is not under-estimated. While the use of some conservative assumptions in risk assessment can be justified in the spirit of erring on the side of caution when being protective of public health, the typical consequence of the current process is a large degree of compounded over-statement of the risk because of the multiplicative manner in which the assumptions interact. The draft PRA white papers need some more detailed guidance on how assumptions or sequences of assumptions should be handled, i.e., whether they are dependent or independent of each other.

PRA does not result in a single final answer in which the conservative assumptions seen in traditional risk assessments have been compounded. Additionally, PRA results in a range as opposed to traditional risk assessment single point value that can be skewed by the use of upper bounds rather than being more informed by probability distributions. Another study by the same researchers found that the degree to which risk reduction benefits (if at all) will be overstated varies considerably from rule to rule. The illustrative examples in that study indicate that benefits derived using precautionary assumptions may be 10, 20, 100, or even more times higher than one would expect at the mean or media of the benefits distribution. That study can be found at http://www.nrwa.org/whitepapers/conserve/conserve02/conserve02.doc . The well-managed application of PRA will be of considerable value where it replaces the traditional use of single point benefits in which the cascading sequences of precautionary assumptions mask the degree of variability and uncertainty therein.

To illustrate this point, we would like to offer our point of view regarding one of the Group 3 case studies in Appendix D. AWWA did not end up with the same perception of the economic analysis and the underlying statistical model that is discussed in the case study on the *Cryptosporidium* concentrations used for Long-Term 2 Enhanced Surface Water Treatment Rule (LT2ESWTR). The Bayesian analysis used to generate these concentrations, from our point of view, was a case of "smoke and mirrors" particularly in how they were presented to the Federal Advisory Committee involved in the negotiations. The graphical results of the Bayesian analysis that were presented to the Committee were difficult to follow and/or understand, and there was not a consensus on the assumptions being used in this analysis. The case study summary implies that the stakeholders were in agreement with the occurrence and dose-response components of the risk analysis model, and that is simply not the case. While these issues did not impact our ultimate agreement with this rulemaking and our ongoing commitment to its successful implementation, our comments on the proposed LT2ESWTR that are in that rulemaking docket reflected our serious concerns with the economic analysis and the underlying model.

One issue not discussed in the white papers is the source of data for the distributions used in a PRA. AWWA was very involved in negotiations for a Revised Total Coliform Rule (RTCR) that culminated in an Agreement-In-Principle (AIP) being signed in September 2008. Section 4.1.c of this AIP details the need for information and data collection to better inform the benefit-cost analysis (BCA). The PRA white papers should clearly acknowledge the need for quality control and quality assurance practices to be in place prior to the collection of any data used to build the PRA distributions. Furthermore, the PRA white papers should also provide a list of clear and simple recommendations that outlines minimum data quality for attaining these objectives.

If you have any questions about these comments, please feel free to call me or Alan Roberson in our Washington Office.

Yours Sincerely,

Thomas W. Curtis
Deputy Executive Director

cc: Cynthia Dougherty—USEPA OGWDW
Pam Barr—USEPA OGWDW
Stig Regli—USEPA OGWDW

CRD 2009 p.0645

UNITED STATES
NUCLEAR REGULATORY COMMISSION
WASHINGTON, D.C. 20555-0001
October 1, 2009

OCT 06 2009

Dr. Gary Banks
ORD Docket
Environmental Protection Agency
Mailcode 28221T
1200 Pennsylvania Avenue, NW
Washington DC 20460

SUBJECT: COMMENTS ON US EPA WHITE PAPER, "USING PROBABILISTIC METHODS TO ENHANCE THE ROLE OF RISK ANALYSIS IN DECISION-MAKING WITH CASE STUDY EXAMPLES"

Dear Dr. Banks:

The United States Nuclear Regulatory Commission (NRC) has reviewed and is providing comments on the United States Environmental Protection Agency's (EPA's) White Paper "Using Probabilistic Methods to Enhance the Role of Risk Analysis in Decision-Making With Case Study Examples" made available for public comment via *Federal Register* notice (74 FR 41695) dated August 18, 2009. The NRC supports the use of risk-informed and performance-based regulation. A key tenet of this regulatory philosophy is the use of risk-insights (that may be generated from probabilistic analysis) and other information to focus more licensee and regulatory attention on those issues most important to health and safety. The Commission's commitment to risk-informed regulation was formalized in a Policy Statement (60 FR 42622) issued on August 16, 1995, to expand the use of probabilistic risk assessment (PRA) "in all regulatory matters to the extent supported by the state of the art in PRA methods and data, and in a manner that complements the NRC's deterministic approach and supports the NRC's traditional defense-in-depth philosophy." The NRC continues to develop guidance, fund research, develop tools, and collect data to support the use of PRA in agency decision-making and appreciates the efforts of and challenges faced by EPA in similarly advancing the use of probabilistic risk assessment in its regulatory matters.

The NRC looks forward to continuing to review EPA documentation and sharing information related to use of PRA as both agencies continue to develop the tools and informational resources needed to support and advance the use of PRA methods and analysis to inform agency decision making.

In accordance with 10 CFR 2.390 of the NRC's "Rules of Practice for Domestic Licensing Proceedings and Issuance of Orders," a copy of this letter will be available electronically for public inspection in the NRC Public Document Room or from the Publicly Available Records component of NRC's Agencywide Documents Access and Management System (ADAMS). ADAMS is accessible from the NRC Web site at http://www.nrc.gov/reading-rm/adams.html.

OCT 02 2009

If you have any questions regarding our comments, please feel free to contact me at 301-415-6663 or Christepher.Mckenney@nrc.gov.

Sincerely,

Chris McKenney

Christepher McKenney, Branch Chief
Performance Assessment Branch
Environmental Protection
and Performance Assessment Branch
Division of Waste Management
and Environmental Protection
Office of Federal and State Materials
and Environmental Management Programs

DOCKET ID NO. EPA-HQ-ORD-2009-NNN

Enclosure: Comments on US EPA White Paper

Comments on the U.S. Environmental Protection Agency's White Paper "Using Probabilistic Methods to Enhance the Role of Risk Analysis in Decision-Making with Case Study Examples"

General

1. Given the large range of decisions that could be made across the Environmental Protection Agency (EPA) offices and programs, it would be helpful to include in introductory chapters a general description of the types of decisions that are being made at EPA that could benefit from a Probabilistic Risk Assessment (PRA) (e.g., to perform cost-benefit analyses for rulemaking; to compare alternatives for remediation; to evaluate compliance against regulatory standards; or to optimize sample collection) and to discuss whether PRA may be more or less suited for these various applications (see last bullet on the bottom of page 21). A similar situation exists for NRC, the agency recognizes that the state of the art in PRA methods is more advanced and/or more amenable to certain regulatory programs (e.g., nuclear reactors and high-level waste disposal). Thus, when the Commission issued its Policy Statement on PRA in 1995, it expected that the transition to a risk-informed regulatory framework across all regulatory programs would be incremental and that the extent to which PRA would be used in various regulatory programs variable.

2. There appears to be a lack of specificity regarding how statistical measures of uncertainty should be considered in making decisions (e.g., evaluating compliance against a regulatory limit). While lack of specification provides more flexibility to the regulator and regulated, it could also lead to the inequitable treatment of risk information from site to site and undermine confidence in agency decision making. For example, the U.S. Nuclear Regulatory Commission (NRC) guidance advocates use of the peak of the mean dose generated from probabilistic risk assessment for decommissioning dose modeling analyses, as well as consideration of the 95th percentile dose for low-level waste disposal facilities when evaluating compliance with regulatory criteria (NRC, 2000; and NRC, 2006). Having recommended metrics in guidance makes it easier for the decision maker and the regulated community to more consistently apply risk information obtained from PRA, providing stability in the regulatory process.

The NRC recognizes differences in the manner in which EPA and NRC regulate (e.g., use of a risk range versus use of a specific dose criterion) which may lead to significant differences in the way PRA is used to make certain decisions. In fact, in several instances the White Paper implies that there is some flexibility in establishing a metric and variability between programs with respect to the usefulness of PRA and how the results are to be interpreted.

- Page 6, Section 1.3, second paragraph, Statements are made that in the face of uncertainty decision-making is determined not only by science but by Agency policy and where not prohibited by statute, the relative costs and benefits of regulatory alternatives may be considered in making decisions. But it is not clear what policies and statutes may limit the utility of PRA or cost/benefit analysis. Examples could be provided.

- Page 7, Section 1.4, sixth bullet, a statement is made that a decision maker often asks, "What is the percentile of the population to be protected?" It is not clear if there are specific recommendations regarding who is to be protected in EPA guidance or regulations.

- Page 13, Section 2.2, bullet 4, Statements are made that PRA might be most useful "When significant equity issues are raised by inter-individual variability." However, it is again not clear what individuals are to be protected.

- Page 16, Section 2.8, States that binary decisions may be perceived to be more readily answered with deterministic analysis as opposed to PRA that present a range of uncertainty. This statement recognizes the difficulty in making decisions when a range of possible outcomes is presented but offers no specific details on how this information should be synthesized or interpreted to make a decision.

- Page 25, fourth paragraph, Discusses evaluation of central tendency and reasonable upper bounds of exposures, effects, and risk estimates, such that the estimates could be for an actual individual in the population of interest rather than a hypothetical Maximally Exposed Individual (MEI). This discussion seems to imply that there is some regulatory flexibility in evaluating risks to potential receptors (does not necessarily have to be the MEI) but introduces ambiguity in the regulatory and decision criteria that might be used in its stead.

- Page 32, "Resolution" definition, Discusses an example when an evaluation of upper-bound risks may necessitate a geographical-information-system-based modeling framework precise enough to model exposures to individual receptors. One might argue that the necessary level or scale of the analysis would be dictated by regulatory criteria (e.g., maximally exposed individual) and depending on the endpoint, one form of analysis may be more appropriate then another (i.e., deterministic bounding versus probabilistic analysis) and/or would dictate what metrics would be used if a PRA is selected. It is not clear to what extent regulatory criteria (and modeling objectives) are prescribed or if there is some flexibility in evaluating individual risk.

- Page 58, Case Study 4, second paragraph, Discusses use of the 95th percentile regulation for lower tiers that do not include percent crop treated to use of the 99.9th percentile for the more refined assessments which would include percent of crop treated information, but the basis for these percentiles is not provided and it is not clear if these percentiles are specified in regulation or guidance.

- Page 60, Case Study 5, first paragraph, Discusses results of deterministic analysis that exceeds regulatory benchmarks but does not indicate what these benchmarks are or provide information on whether these benchmarks constitute regulatory requirements. The "Results of the Analysis" section presents information on the central tendency individual and reasonable maximum exposure individual, but it is not clear if decisions are based on protection of one or both of these individuals and at what percentage of the PRA distribution of results are these individuals expected to be evaluated.

Although there are obvious differences in the manner in which various agencies regulate (and even intra-agency program differences), it would help to clarify if evaluation of specific PRA metrics are recommended in EPA guidance (e.g., percentile of the population to be protected; mean or 95th percentile of exposure distribution) when making certain regulatory decisions (e.g., making cost-benefit decisions, or decisions regarding regulatory compliance); if certain constraints (agency policy or statute) limit the use of PRA in EPA decision making; and if there is some flexibility in applying specific metrics to certain problems.

3. It appears that the focus of most of the PRA case studies presented in the EPA White Paper is on a subset of the components of an environmental risk assessment. Consideration of uncertainty and variability in all aspects of the assessment (i.e., initiating events/release, fate and transport, exposure, and consequences) may reveal that the risk is dominated by certain components of the environmental risk assessment model that may not be immediately intuitive. Factors such as uncertainty in scenarios being evaluated; features, events and processes that may occur in the future; and fate and transport of contaminants in the environment may have relatively large uncertainties. While uncertainty in these components of the environmental risk assessment is recognized (see bulleted examples below), more emphasis appears to be placed on exposure and consequence modeling in the textual examples and Case Studies.

- Page 23, bullet four, States that "There may be mismatches in the temporal and spatial resolution of each model, which confound the ability to propagate variability, and uncertainty from one model to another," recognizing the challenges of integration, and coupling of models.
- Page 27, Section A.3.1, "Structural Uncertainty in Scenarios" Section, Discusses important components of environmental risk assessment models (e.g., source definition, transport, exposure routes, etc.) that constitute forms of structural uncertainty but states that there are no formalized methodologies for dealing with uncertainty and variability (and that qualitative approaches to addressing these uncertainties are common).
- Page 27, Section A.3.1, "Coupled Models," Discusses components of an environmental risk assessment model that have varying spatial and temporal scales that are difficult to integrate and can introduce a significant source of structural uncertainty but presents no clear path on how this uncertainty can be addressed.
- Page 60, Case Study 5, "Probabilistic Analysis" Section, last sentence, Discusses the use of mathematical models of environmental fate, transport, and bioaccumulation of polychlorinated biphenyls (PCBs) in the Hudson River to forecast changes in PCB concentrations over time. However, it is not clear that uncertainty in PCB concentrations were propagated over time and how the uncertainty in PCB concentrations compares to uncertainty in angling duration and other exposure factors which appear to be the focus of the Case Study.

If sensitivity of model results to uncertainty and variability inherent in all aspects of environmental risk assessment modeling is not adequately studied, sensitivity analysis results may be misleading and PRA results of limited utility. Because the nature EPA decision making may differ markedly from other agencies (due to, for example, programmatic differences in risk assessment methods or end points evaluated), the apparent emphasis on certain aspects of environmental risk assessment modeling may be warranted. Additional discussion regarding the relative uncertainties expected to be introduced by various components of the environmental risk assessment model for various EPA applications and/or an explanation on why emphasis seems to be placed in particular areas of the risk assessment would be beneficial.

4. In many cases, in order to adequately perform PRA to inform decision making, sufficient resources are needed to (i) analyze and synthesize data into forms that are of use to a risk analyst and (ii) create an infrastructure to more efficiently implement PRAs. In fact, these limitations and challenges to the use of PRA are recognized by EPA. For example, it was noted in the EPA Policy for Use of Probabilistic Analysis in Risk Assessment (1997) that additional study was needed to evaluate uncertainty in dose response for human health risk assessments. The White Paper discusses the upfront increase in resources needed to perform PRA (bottom of page 16), but recognizes the longer-term benefit of development of standardized approaches and/or methods that can lead to the routine incorporation of PRA in Agency matters.

The NRC has faced similar challenges in developing a framework and tools to increase the use of PRA in its regulatory programs. For example, NRC contracted Sandia National Laboratories and Argonne National Laboratory to provide probabilistic capabilities in the DandD and RESRAD decommissioning codes, respectively, including development of associated parameter distributions in the late 1990s (SNL, 1999; ANL, 2000).

A summary listing or examples of the types of data that are routinely collected or are planned to be collected and analyzed by the EPA for inclusion in environmental risk assessments; and the types of tools and other resources that are currently being developed to aid in implementation of PRA analyses in the future would be beneficial.

5. A stated goal of the EPA White Paper is to explain how EPA can achieve a broader use of probabilistic methods and address uncertainty and variability by capitalizing on the wide array of *tools* and methods that comprise PRA. However, while significant information is provided on methods that comprise PRA, less information is provided on the tools of PRA (e.g., off-the shelf software for performing PRA or EPA sponsored codes specifically designed to execute probabilistic analysis). While this omission may have been intentional, it would be helpful if additional examples of "off-the shelf" software and additional details on other tools being used to perform these types of analyses is included.

<u>Specific</u>

6. Page 9, Section 1.7, second paragraph, A statement is made that deterministic risk assessments provide estimations of exposures and resulting risks that address uncertainty and variability in a qualitative manner. Deterministic analyses can, to a certain degree, study uncertainty and variability in model results in a quantitative fashion, although there are obvious limitations to this approach. For example, robust sensitivity analysis can be conducted and a conscious decision made to select a certain parameter value that is expected to represent central-tendency or pessimistic estimates of risk in a deterministic assessment. Statements in the text regarding the limitations of deterministic analysis and implications that these types of analyses are not science based should be checked and carefully worded (see also page 16, Section 2.8, statement that decisions should be based on the best available science). As discussed in the White Paper, in some cases deterministic analyses are adequate and while they may not necessarily reflect the best available science (because best available science is not warranted), they may still be technically defensible and scientific in their approaches.

7. Page 12, Section 2.1.1, While the 1997 EPA Policy on Probabilistic Analysis in Risk Assessment (EPA, 1997) states that additional study is needed to apply PRA to dose effects (and this policy does not appear to be superseded), several examples are included in this paragraph regarding use of PRA in this manner. Please clarify EPA's current policy and progress in this area.

8. Page 13, first paragraph on "Model Uncertainty" and page 23, Section 3.4, bullet 2, These sections of the White Paper provide information about model uncertainty and challenges faced in evaluating this type of uncertainty. It should be noted that while model uncertainty and abstraction are important components of uncertainty that should be evaluated, it would be preferable to spend adequate resources up front making sure that the models used to perform the risk assessment are appropriate for their intended application (e.g., that the appropriate level of complexity is captured in the models) and that uncertainty is propagated in the appropriately-selected model. However, in the case of complex systems that must necessarily be simplified and that are difficult to validate using existing data, use of multiple models or scenarios may be warranted and uncertainty in these models should be addressed. While evaluation of model uncertainty is an area in need of continued research, guidance is currently available on the treatment of model uncertainty that can be referenced in this section (e.g., see for example Meyer, 2004).

9. Page 13, Section 2.2, Suggest adding a bullet regarding the utility of PRA when model complexity makes it difficult to assess the conservatism of a particular selection of parameter values in a deterministic assessment.

10. Page 21, Section 3.1, Statements are made regarding the utility of PRA in evaluating various risk management strategies and alternatives; and that sensitivity analyses can be used to identify influential knowledge gaps. However, no information is provided on how probabilistic analyses are superior in these areas compared to deterministic analyses and/or when it might be appropriate to use deterministic analyses.

11. Page 24, second paragraph, last sentence, Provide an example of when it would be appropriate to refine an assessment objective depending on the availability of information. It would seem that the assessment objective would be based on some regulatory metric and not necessarily the availability of information. If insufficient information is available one might not be able to make a decision or might manage uncertainty with conservative assumptions.

12. Page 24, "Levels of Analysis" box, The text should clarify and provide a basis for the ordering of "levels of analysis" (e.g., why is expert elicitation listed last or sensitivity analysis [which can take on many forms] listed first?). The text should note that sensitivity analyses can be either deterministic or probabilistic. It is not clear why Monte Carlo analysis of variability is limited to exposure data and human health and ecological effect data. Define or provide examples of "decision uncertainty analysis" and "geospatial analysis" and provide a basis for where they fall in the ordering of analyses.

13. Page 25, third paragraph, "In such a situation, depending on the resource implications of risk management, it might be appropriate to proceed with a more refined, or higher level, analysis. If the cost of intervention is less than the cost of further analysis, then it may be appropriate to simply proceed to the risk management decision as a preventive measure that is

also expedient. In some deterministic assessments, for instance, for ecological risks, the assumptions are not well assured of conservatism and the estimated risks might be biased to appear lower than the unseen actual risk." The last sentence above introduces a separate thought and potential problem with respect to deterministic analyses that should be developed on its own (issue with deterministic analyses not clearly being conservative in the face of great uncertainty). Additionally, the thought that additional ecological modeling could be more costly then remediation (making remediation potentially a more attractive option) could be more clearly made in the example. Suggest rewording the sentence for clarity and/or providing a better example.

14. Page 31, Appendix B: Glossary, Suggest adding PRA-related terms to the glossary that are used but not well-defined in the text of the White Paper: (i) dose response, (ii) target population, (iii) hazard identification, (iv) reference dose, (v) hazard index, (vi) decision uncertainty, (vii) geospatial analysis.

15. Page 34, "Sensitivity Analysis" definition, Suggest listing the different types of sensitivity analysis and sensitivity analysis techniques.

16. Page 53, Case Study 1, Explains how sensitivity analysis was used to determine key variables for population exposure variability to arsenic in chromated copper arsenate pressure-treated wood. The study found that data needed to be collected on the amount of dislodgeable residue that is transferred from the wood surface to a child's hand upon contact and the amount of dislodgeable residue that exists on the wood surface. It would seem that these parameters would change over time as the integrity of the pressure treated wood diminished. This would be an example of a scenario or structural model uncertainty that might be the most risk significant aspect of the exposure modeling, but if not considered, would not be evaluated as part of the PRA (see general comment 3 above).

17. Page 62, Figure 1, It is not clear why this plot which shows the uncertainty in risk estimates to discrete population percentiles (representing inter-individual variability) is not an example of a 2D Monte Carlo analysis (appears to be [albeit a more discrete] version of a 2D Monte Carlo analysis result similar to what is presented in Figure 2 on page 26). On the other hand, the figure on page 73 does not seem to clearly present results of a 2D Monte Carlo analysis related to ozone exposure with only uncertainty in model inputs apparently being presented. Suggest including more illustrative examples of the characteristics of 2D Monte Carlo analysis in the Case Study examples.

<u>Editorial</u>

1. Check consistency of the use of the hyphen in decision-maker, decision-making throughout the document. The Chicago Manual of Style indicates that decision making is the noun and decision-making is the adjective. While there are probably multiple correct uses, the document should be consistent in its application.

2. The reference Cullen and Frey 1999, is used more heavily than any of the other references throughout the document although there are many other references. It might be appropriate but the heavy reliance on this particular reference was noticeable.

3. Page 5, 1st paragraph, "One can use probability (chance) to quantify the frequency of occurrence or the degree of belief in information." This statement can be clarified to avoid an incorrect interpretation. Probability is not equivalent to frequency. Probability is a value between 0 and 1. Frequency can be greater than 1.

4. Page 6, last paragraph, A statement is made that "Increased uncertainty can make it more difficult to ..." perform a cost-benefit analysis. Suggest re-writing for clarity (i.e., what is the increase in uncertainty in relation to?).

5. Page 14, Suggest deleting the dates that introduce the listed items as the dates are already included in the references at the end of the bullets.

6. Page 14, Section 2.4, second paragraph, Introduce the acronym SAB used on the following page.

7. Page 15, Section 2.7, Check bullets to ensure parallelism in punctuation and sentence structure.

8. Page 27, Section A.3, This paragraph is redundant with the second paragraph on page 25.

9. Page 32, first paragraph, last sentence, Suggest providing a better example then "logistic models" which has not been defined and may not be obvious to a reader.

10. Page 32, "Resolution" definition, Check sentence stating "If the grid size selected is too small...," should this be "large" not "small" or should reference to the scale be small (as opposed to grid size).

11. Page 34, "Uncertainty" definition, Delete last sentence which appears to be a reviewer comment.

12. Page 53, first paragraph, first sentence, Should the reference to Group 2 assessment be Group 3 Assessment (for Case Study 9—see Page 51).

13. Page 57, Should Case Study 9 actually be Case Study 5 (see Page 51).

14. Page 63, title caption, Should be "report" not "reprot."

References

ANL, 2000. "Development of Probabilistic RESRAD 6.0 and RESRAD BUILD 3.0 Computer Codes," NUREG/CR-6697, Argonne National Laboratory, November, 2000.

EPA, 1997. "Policy for Use of Probabilistic Analysis in Risk Assessment," US Environmental Protection Agency, May 15, 1997.

NRC, 2000. "A Performance Assessment Methodology for Low-Level Radioactive Waste Disposal Facilities," NUREG-1573, October, 2000.

NRC, 2006. "Consolidated Decommissioning Guidance," Volume 1, Revision 2, Volume 2, Revision, 1, NUREG-1757, September, 2006.

NRC, 2007. "NRC Staff Guidance for Activities Related to US DOE Waste Determinations," Draft Final Report for Interim Use, NUREG-1854, August 2007.

Meyer, P.D., et. al., 2004. "Combined Estimation of Hydrogeologic Conceptual Model and Parameter Uncertainty," NUREG/CR-6843, Pacific Northwest National Laboratory and University of Arizona, March, 2004.

SNL, 1999. "Residual Radioactive Contamination from Decommissioning: Parameter Analysis, Draft Report for Comment," NUREG/CR-5512, Volume 3, Sandia National Laboratory, August, 1999.

WPSC
Wood Preservative Science Council

DATE: October 15, 2009

TO: Docket EPA-HQ-ORD-2009-0645

Dear sirs:

The Wood Preservative Science Council (WPSC)[1] provides the following comments on the Agency's draft white paper titled "Using Probabilistic Methods to Enhance the Role of Risk Analysis in Decision-Making With Case Study Examples" as announced at 74 FR 41695 (August 18, 2009).

The WPSC is supportive of the Agency's efforts to develop improved methodologies to assess risks, including the use of probabilistic methods where appropriate. Such methods, when properly developed with appropriate assumptions and underlying scientific justifications, can be helpful in the Agency's decision making process. The use of empirical data to replace assumptions in such models should be incorporated into the Agency's process for continued improvement of the models[2,3].

The WPSC believes that the white paper mischaracterizes the use of the SHEDS-Wood probabilistic model in relation to risk assessment and decision making for Chromated Copper Arsenate (CCA) wood preservatives and is incomplete in its description of the uses of that model. The WPSC believes that minor modifications to certain statements will better characterize the model's application and use in risk management decisions without diminishing the importance of the development, improvement, and use of probabilistic models. Our specific comments follow.

Under "Results of Risk Analysis" (Case Study 9, page 70-71 of the White Paper), the Agency states that the Office of Pesticide Programs used the SHEDS-Wood model for a risk assessment

[1] The WPSC is an association of manufacturers of water borne wood preservatives. It supports and participates in objective scientific analysis of water borne wood preservatives with a focus on CCA. We are supported by our members, Arch Wood Protection, Inc. and Osmose Inc. The WPSC consults with the nation's leading experts in the fields of environmental science, epidemiology, risk assessment, and toxicology.

[2] Barraj, L.M., and J.S. Tsuji. 2007. Letter to the editor regarding Zartarian et al. 2006 and Xue et al. 2006 in Risk Analysis Vol 26. Risk Analysis 27(1): 1-3

[3] Barraj, L.M., J.S. Tsuji, and C.G. Scrafford. 2007. The SHEDS-Wood model: incorporation of observational data to estimate exposure to arsenic for children playing on CCA-treated wood structures. Environ Health Perspect 115: 781-786

of existing stuctures and that "This included recommendations for risk reduction (use of sealants and careful attention to children's hand-washing) to homeowners with existing CCA wood structures." However, this does not state accurately what was done in regards to offering advice concerning CCA-treated wood structures. In its April 2008 general advice to consumers[4], EPA states there is no reason to remove either existing structures or surrounding soil, identifies that there is limited evidence that under some circumstances some coatings may reduce dislodgeable residues but does not recommend their use, and offers the generally applicable good hygiene practice to wash hands after handling any outdoor structures.

Also in this section, the White Paper identifies that EPA and the Consumer Product Safety Commission conducted two-year studies to evaluate the impact of commercially-available sealants on residue availability but fails to identify that the FIFRA Scientific Advisory Panel review of those studies at its November 2006[5] meeting concluded that while those studies provided some evidence that some coatings under some conditions might reduce absolute levels of dislodgeable residues, the studies themselves would not be sufficient to provide advice to the public. The SAP recommended steps to consider if further research in that area is conducted, and the Office of Pesticide Programs concurred with the SAP recommendations[6] that more definitive studies are needed.

Specifically under Case Study 9 on pages 70-71 of the White Paper, the Agency states the following under "Management Considerations":

> "The modeling product was pivotal in the risk management and re-registration eligibility decisions for CCA, and in advising the public how to minimize health risks from existing treated wood structures."

In fact, this model and its estimated exposures and risks were not relevant to and were not used by the Agency in any risk management or reregistration eligibility decisions. This was because there have been no registered uses of CCA for treatment of wood used in the scenarios addressed

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[4] EPA 2008. Consumer advice related to CCA-treated wood (available at http://www.epa.gov/oppad001/reregistration/cca/cca_consumer_doc.htm, accessed on 9/9/2009)

[5] FIFRA SAP. 2007. Transmittal of Meeting Minutes of the FIFRA Scientific Advisory Panel Meeting Held November 15 - 16, 2006 on Studies Evaluating the Impact of Surface Coatings on the Level of Dislodgeable Arsenic, Chromium and Copper from Chromated Copper Arsenate (CCA)-Treated Wood. Memorandum dated January 25, 2007. (available at http://www.epa.gov/scipoly/sap/meetings/2006/november/november2006finalmeetingminutes.pdf, accessed on 9/9/2009.

[6] See at http://www.epa.gov/oppad001/reregistration/cca/ accessed on 9/9/2009.

by SHEDS-Wood since 2003.[7] Therefore, these uses were not part of the decision to reregister CCA.[8].

For these reasons, the WPSC recommends that the Agency make revisions to its statements regarding the use of the SHEDS-Wood model in relation to CCA to better reflect the actual use and interpretation of the model estimates in risk assessment and decision-making by the Office of Pesticide Programs. Such revisions will not detract from the work done to develop the model, the overall importance of the development and use of probabilitistic models, or the potential use of the model for assessment of other types of pesticide products used to preserve wood.

Please contact me at 202-419-5166 if there are any questions regarding these comments.

Sincerely,

Elizabeth Anne Brown

Elizabeth Anne Brown, Ph.D.
Steptoe & Johnson LLP
On behalf of the Wood Preservative Science Council

[7] See 68 FR 17366, April 9, 2003

[8] EPA. 2008. Human health risk assessment and ecological effects assessment for the reregistration eligibility decision (RED) document of inorganic arsenicals and/or chromium-based wood preservatives. September 18, 2008. (available at EPA-HQ-OPP-2003-0250-0081).

CropLife
AMERICA

October 15, 2009

Submitted Via EPA Docket Number EPA-HQ-ORD-2009-0645

ORD Docket
Environmental Protection Agency
Mail code: 28221T, 1200
Pennsylvania Ave., NW
Washington, DC 20460

Re: **Using Probabilistic Methods to Enhance the Role of Risk Analysis in Decision-Making with Case Study Examples:**

Hello,

CropLife America (CLA) is a not-for-profit trade organization representing the nation's developers, manufacturers, formulators and distributors of plant science solutions for agriculture and pest management in the U.S. Our member companies produce, sell and distribute virtually all the crop protection technology products used by American farmers. CLA comments on issues that can have broad regulatory implications, which sometimes occur in the context of chemical-specific or product-specific regulatory reviews, decisions, and actions.

I am hereby respectfully submitting the comments presented below on the document cited above. Please contact me if you have any questions or require additional information.

Sincerely,

Dee Ann Staats

Dee Ann Staats, Ph.D.
Environmental Policy Director

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Croplife America Comments on
"Using Probabilistic Methods to Enhance the Role of Risk Analysis in Decision-Making with Case Study Examples"

General Comments:

As stated in the Foreword, the goals of the Probabilistic Risk Assessment white paper (PRA) is to describe the uses of probabilistic methods in the risk decision process and to encourage their further implementation in human, ecological and related decision making by the EPA. In general, the report accomplishes these goals. CropLife America fully supports the use of probabilistic methods in human and ecological risk assessments conducted for pesticides in support of registration or re-registration under FIFRA. To date, EPA has seldom used probabilistic methods to refine screening-level assessments of pesticides. This situation is disappointing, given that nearly every screening-level assessment of a pesticide identifies use scenarios that pose potential risks. With the standard tiered approach to risk assessment, these use scenarios would undergo more refined analyses, including the use of probabilistic methods. The Environmental Fate and Effects Division (EFED) of the Office of Pesticide Programs, which is responsible for conducting ecological risk assessments of pesticides under FIFRA has had formal training with probabilistic methods. Further, the Scientific Advisory Panel has endorsed the use of probabilistic methods in pesticide risk assessments on several occasions dating back to 1998. Thus, CropLife America believes that EFED should commit to the use of probabilistic methods in pesticide risk assessments conducted under FIFRA whenever calculated risk quotients exceed levels of concern.

Although the PRA white paper was intended to address the use of probabilistic methods in both human and ecological risk assessment, the paper is clearly written from the human health perspective. There are numerous instances where the text would have benefited from the inclusion of ecological examples or considerations. Only five of the 16 case studies in Appendix D involve the use of probabilistic methods in ecological risk assessments, and only studies 13 and 16 used probabilistic methods to assess risks to aquatic life or wildlife. The others could just have easily been classified as human health or "environmental" case studies (e.g., probability of sea level rise, design of a national environmental monitoring plan, the contribution of atmospheric deposition to watershed contamination). In reality, EPA has used probabilistic methods extensively in ecological risk assessments for contaminated sites in the United States. EPA Region 1 used probabilistic methods to assess risks of PCBs and dioxins and furans to selected bird and mammal species in the Housatonic River in Massachusetts. (See http://www.epa.gov/region1/ge/thesite/restofriver-reports.html#ERA.) EPA Region 6 conducted a similar probabilistic risk assessment for wildlife exposed to mercury, PCBs, dioxins and furans and lead in the Calcasieu Estuary area of Louisiana (see http://www.epa.gov/region6/6sf/sfsites/datarep.htm). EPA should consider adding these or other ecological case studies to Appendix D.

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Given the heavy reliance on the opinion (as expressed through the cited past publications) of the National Academy of Sciences/National Research Council (NAS/NRC) with regard to the use of PRA, it is surprising that this white paper has not been updated to include the more recent opinions/thoughts expressed in the "Silver Book" (Science and Decisions: Advancing Risk Assessment, 2008) as well as the 2008 report entitled Phthalates and Cumulative Risk Assessment: The Tasks Ahead. Since these two volumes represent the latest opinion of the NAS, this white paper should be updated in several areas to incorporate these latest risk assessment reports. Sections that could be improved from consideration of these two reports include, but are not limited to: 1.5; 1.6; 2.4; 2.6

Review of Section 1.8 (page 10) raised many concerns regarding an overall lack of comprehensiveness when considering past EPA efforts in PRA. It is stated that both OPP and OSWER have developed specific guidance in the use of PRA – how have these guidance directly contributed to this white paper? How has the information gained from conducting PRA techniques and methods been synthesized into "lessons learned" to help improve these guidance? For example, from past Agency experience in using these tools, where has PRA been shown NOT to improve upon information generated and decisions made from using deterministic techniques?

The argument put forth mainly in Section 2.9 (but also referenced elsewhere in the document) would benefit from stating the fact that major resources will be required "up front" to develop "Standard Evaluation Procedures" for PRA-based Agency assessments. This is in addition to the upfront resources needed merely to conduct a PRA, relative to a deterministic assessment. Furthermore, this section would be much improved by citing other sovereign government experiences and proof that "ongoing resource cost may be offset by a more informed decision." See Specific Comment on Page 17, Section 2.10.

The recommendations listed toward the end of Section 3.2 are somewhat redundant. All of these suggestions to "improve implementation" fall into one of three general categories: inform, train, and promote. Collapse all suggestions into one of these three major headings to reduce redundancy here.

The case studies could be restructured to provide more information to the reader. It is recommended that the case study information presented flow as follows:

- What is the problem?
- What is the best PRA tool/technique to solve the problem?
- What was the tool used to solve the problem, assuming the "best" tool was not used for some reason (and explain why it was not used)
- Describe the approach used.
- Describe the management considerations.
- Comment on "lessons learned" and how these can drive improvements in future applications of the approach used.

In many places in the text (particularly in Section 3), the text just asserts the many benefits of probabilistic methods. It would be much more convincing if specific examples (e.g., using text boxes) illustrated the benefits of probabilistic risk methods

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(e.g., their use led to a more effective decision than did deterministic methods for a particular contaminated site).

Appendix A provides only a superficial overview of probabilistic methods. The text should identify and discuss each of the techniques currently in common use (e.g., first- and second-order Monte Carlo analysis, Bayesian methods, etc) and others that may become useful in the future (e.g., probability bounds analysis, fuzzy arithmetic, etc).

Case study 13 is the only example provided that shows how probabilistic methods have been used to assess pesticide risks to wildlife. The case study describes a probabilistic model (the Terrestrial Investigation Model [TIM], version 2.0) that was developed to estimate the risks of a hypothetical flow of pesticide to birds that forage on treated fields. This case study, however, has several problems, including:

- The material provided is outdated and has been superseded by TIM, version 2.1, which was presented to the FIFRA Scientific Advisory Panel in February 2008. Similarly, the "Chem X" case study, conducted nearly a decade, ago has been updated and expanded upon in an actual pesticide risk assessment (carbofuran), which was presented to the same Panel in February 2008. Although the carbofuran example has a number of flaws, it does represent an improvement over the Chem X case study.
- The last paragraph on page 79 is completely out of place. It has nothing to do with the text describing the terrestrial or aquatic level II models that are described in the surrounding text.
- Little information is provided on the terrestrial and aquatic level II models and their current status of development and use in the Agency.
- The Results section does not mention birds.
- The Management Considerations section is not a balanced presentation of the opinions of the Scientific Advisory Panels that have reviewed the level II models. The Panels have suggested numerous refinements to further improve the level II models, many of which will require significant time and resources to incorporate.

Specific Comments:

Page 4, Paragraph 1. The statement that stakeholders have requested the use of probabilistic methods to ensure a "fuller characterization of risks, including uncertainties, in protecting more sensitive or vulnerable populations and life stages," does not make sense. Better characterization of risks through the use of probabilistic methods will not "protect" sensitive populations and life stages. Only effective risk management actions can accomplish that goal. Probabilistic methods do, however, contribute to a fuller characterization of risks and thus provide useful information that can contribute to effective decision making regarding protection of sensitive populations and life stages.

Page 6, Section 1.3 There is reference to "traditional methods of risk analysis" towards the end of the first paragraph of this section. It should be specified here that traditional

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methods are synonymous with the deterministic approach to risk analysis. NOTE: This comment should apply to most additional occurrences of the phrase "traditional methods."

Page 15, Section 2.6. The final bullet of this section is somewhat confusing: "By adopting PRA, EPA send the appropriate signal to the intellectual marketplace …" The white paper clearly lays out the fact that EPA has already adopted PRA techniques in many instances, so the meaning of "by adopting PRA" is unclear. Do the authors refer to some more formal EPA document/proclamation/etc. that needs to occur to "show the intellectual marketplace" that EPA has "officially" embraced PRA? Please clarify. In addition, the terms "appropriate signal" and "intellectual marketplace" are equally nebulous. Finally, "encouraging analysts to gather data" is also confusing … the implication here is that "analysts" (an undefined term – EPA analysts?) will only "gather data" subsequent to some more formal adoption process.

Page 16, Section 2.8. The title of this section is inaccurate. "Why" is explained in this section, not "How." Title should be changed to reflect this.

Page 17, Section 2.10. The final sentence states that PRA "can provide additional interpretations that compensate for additional efforts." Is this demonstrated in the case studies provided along with the white paper? This section/argument would be much improved by citing real-world examples of such compensatory interpretations. See General Comment 3.

Page 17, Section 2.11. Provide a reference that supports the statement that PRA "fits directly into a graduated hierarchical approach to risk analysis."

Page 17, Section 2.10. The text should note that there are probabilistic methods (e.g., 2nd-order Monte Carlo analysis, probability bounds analysis, interval analysis) that can and should be used in data-poor situations.

Page 18, Section 2.13. This section gives very little useful guidance on communicating the results of a probabilistic risk analysis to scientists, risk managers, stakeholders and the public. There is a rich literature on this topic that can help assessors determine what methods will work for different audiences.

Page 21, Section 3.2. Contrary to what is written in the first sentence here, there is little discussion in the paper regarding the methods and tools that are available for conducting a probabilistic risk assessment. Appendix A is insufficient in this regard.

Page 23, Section 3.4. This section briefly describes some of the challenges that must be met for there to be further use of probabilistic methods by EPA. The last three challenges listed in this section, however, are specific to a very narrow topic – addressing model uncertainty. Many broader challenges (e.g., lack of available expertise in the Agency, lack of resources for training, lack of guidance for many programs, etc) are not mentioned but are much more important than the challenges listed in this section.

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October 16, 2009

Dr. Kathryn Gallagher
Risk Assessment Forum
Mail Code 8105R
Environmental Protection Agency
1200 Pennsylvania Avenue, NW.
Washington, DC 20460

Re: EPA–HQ–ORD–2009–0645; External Peer Review Draft of Using Probabilistic Methods To Enhance the Role of Risk Analysis in Decision-Making With Case Study Examples, Federal Register / Vol. 74, No. 179 / Thursday, September 17, 2009 / Page 47794

Dear Dr. Gallagher:

The American Chemistry Council (ACC) appreciates the opportunity to comment on EPA's *External Peer Review Draft of Using Probabilistic Methods To Enhance the Role of Risk Analysis fin Decision-Making With Case Study Examples (18 August 2009)* (hereinafter referred to as *EPA's Daft Probabilistic RA Guidance Document*). EPA's guidance for conducting risk assessments is both nationally and globally significant. ACC commends EPA for its efforts to push forward with the development, peer review and eventual adoption of advanced risk assessment methods and improved science-based policies, such as probabilistic approaches in lieu of deterministic methods. ACC represents the leading companies engaged in the business of chemistry in the United States. ACC members apply the science of chemistry to make innovative products and services that make people's lives better, healthier and safer, more energy efficient and more convenient.[1] ACC is committed to improved environmental, health and safety performance through Responsible Care®, common sense advocacy designed to address major public policy issues, and health and environmental research and product testing. Chemistry is a science-based industry. Virtually all of our products are the result of extensive research by skilled chemists who strive continuously to develop new molecules that perform needed functions, and sophisticated work by chemical engineers who design processes that make these products more safely and efficiently.

ACC and its member companies have played an active role in screening and testing chemical substances, developing risk assessments and implementing science-based risk management policies. Americans need and deserve a regulatory system that reflects the scientific knowledge and technological innovations achieved over the last 30 years – a system that is built upon a firm scientific foundation – a foundation that supports risk-based product stewardship and regulatory

[1] The business of chemistry is a $635 billion enterprise and a key element of the nation's economy. It is one of the nation's largest exporters, accounting for ten cents out of every dollar in U.S. exports. Chemistry companies are among the largest investors in research and development. Safety and security have always been primary concerns of ACC members, and they have intensified their efforts, working closely with government agencies to improve security and to defend against any threat to the nation's critical infrastructure. ACC's member companies share the objective of meeting consumer, scientific and industrial demands for products and processes that protect human health and the environment. Our industry's technological innovation and progress help protect children from illness and injury, through products such as life-saving vaccines, child safety seats, and bicycle helmets, to name but a few.

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decisions which enhance protection of human health and the environment. Unfortunately, in many ways the Agency's risk assessment practices have lagged behind developments of risk assessment science. EPA has been: slow to move to biologically based risk assessment methods where mode of action data trump defaults; reluctant to adopt probabilistic methods; and has yet to put into routine practice quantitative uncertainty analyses. EPA practices still reflect a reliance on overly conservative default approaches that in many ways are now outdated due to advances in scientific knowledge of toxicology and risk assessment. Despite well intentioned efforts by many within the Agency, considerable improvement is still necessary for EPA to put modern risk assessment methods into practice.

Probabilistic risk assessment (PRA) is an advanced risk assessment tool which provides the means to obtain a broader and more accurate view of potential risks to individuals and populations than is afforded by older, deterministic, methods. As such, the development, adoption and use of probabilistic risk assessment (PRA) methods represent an important and critical step forward in assuring the best available scientific methods are used by EPA and stakeholders to evaluate potential threats to health and the environment.

General Comments:
Overall ACC is very supportive of *EPA's Daft Probabilistic RA Guidance Document* and the use of probabilistic methods to improve the Agency's ability to examine and appropriately address uncertainty and variability in their risk assessments. ACC agrees with and supports the goals of this document as stated on page 5.

Specific Comments
ACC agrees with the limitations of deterministic risk assessments as indicated on page 9 of the *EPA's Daft Probabilistic RA Guidance Document*, but also acknowledges there are situations when this approach is valuable and appropriate. ACC believes the Agency has done a good job in articulating the limitations in a clear and concise manner.

On pages 10 and 11of *EPA's Daft Probabilistic RA Guidance Document*, the Agency lists some of the case studies which they have reviewed and which have been conducted by the EPA. It may be useful also to incorporate case studies from outside the EPA that demonstrate additional applications of the PRA approach. In Attachment A, ACC's Chlorine Chemistry Division provides a case in point: *Example Application of PRA to Toxicity Values: A Case Study with Dioxin-Like Compounds.*

The Agency provides a good overview of PRA in Chapter 2 of *EPA's Daft Probabilistic RA Guidance Document*, and in particular a good summary in bulleted format, on when PRA does and does not make sense on page 13.

ACC agrees and echoes the comments from the National Research Council and the EPA Science Advisory Board that better characterization of uncertainty and variability would be helpful in many cases. ACC also acknowledges, however, that this should not create work unnecessarily, e.g. in situations where there is clearly no significant risk to human health or the environment even based on simple deterministic models.

ACC strongly endorses the use of probabilistic methods for toxicity benchmarks as implied on page 15 of *EPA's Daft Probabilistic RA Guidance Document* with RfDs. This is an area that could help to advance the entire field of risk assessment as PRA is almost exclusively focused only on exposure parameters to this point. The use of distributions and probabilistic approaches applied to toxicity endpoints would help decision makers understand that toxicity is not a cut and dry response. This approach should be brought forward and used within the Agency's Integrated Risk Information System program.

In section 2.7 of *EPA's Daft Probabilistic RA Guidance Document,* EPA touches on several limitations in implementing PRA including a lack of resources. ACC acknowledges this as a reality, but it should also be pointed out that if the Agency moves forward with a greater use of this approach there will be incentive to develop methods and tools that may reduce some of the resource demands.

ACC agrees that PRA can help to make sure assessments are conducted with the best available science (section 2.8 of *EPA's Daft Probabilistic RA Guidance Document*). With the current deterministic approach, too much of what we know about substances is often ignored or lost before it reaches a decision maker. The use of PRA helps to propagate that information forward allowing for better, more informed decisions.

ACC agrees with the assessment of resource needs described in sections 2.9 through 2.11 of *EPA's Daft Probabilistic RA Guidance Document*. The more these methods are used, the easier and less resource intensive they will become as new tools and "standard" distributions are developed.

ACC supports the use of PRA methods in all aspects of risk evaluation including the assessment of health effects and dose response. Under the current default-driven paradigm, these are too often viewed as well defined point estimates and the uncertainty is not adequately communicated to the decision makers. PRA would be a way in which this could be addressed.

* * * * * * * * *

Again, ACC commends EPA for its efforts to improve and modernize its risk assessment policies and practices within the Agency. Probabilistic risk assessment tools better reflect modern scientific understanding of exposures and risks than do default-driven deterministic methods. By providing the means to evaluate and more fully portray variability and uncertainty, probabilistic methods can substantially contribute to the accuracy of risk assessment and provide an improved scientific foundation for risk-based decision making. If you or your staff should have any questions regarding these comments, please don't hesitate to contact me at Rick_Becker@americanchemistry.com or by phone at 703-741-5000.

Sincerely,

Original Signed By

Richard A. Becker, Ph.D., DABT
Senior Toxicologist
Regulatory and Technical Affairs Department

Attachment A.

Example Application of PRA to Toxicity Values: A Case Study with Dioxin-Like Compounds (September 21, 2009)[2]

Probabilistic risk assessment (PRA) methods, including Monte Carlo analysis and other probability based techniques, are well developed and widely applied within the risk assessment science community. Although the U.S. Environmental Protection Agency (USEPA) has been slower to adopt and put into practice PRA across all of the Agency's key program offices engaged in chemical risk assessment, the release of the *External Peer Review Draft of Using Probabilistic Methods To Enhance the Role of Risk Analysis in Decision-Making With Case Study Examples (18 August 2009)* (USEPA) represents an important step in expanding PRA techniques across the Agency. Commonly, PRA techniques use distributions to represent uncertainty and variability in the concentration term and exposure parameters (e.g., exposure duration, body weight, ingestion rate). The result of a probabilistic risk assessment is therefore an estimate of the range and likelihood of risk or hazard rather than a single point estimate of risk (USEPA, 2009). These techniques provide important information such as: 1) the degree of conservatism inherent in point estimates of risk, 2) the proportion of a population that is below a risk or hazard benchmark, and 3) how resources can best be directed to reduce uncertainty and quantify variability.

Historically, probabilistic treatment of toxicity values has been limited (USEPA, 2009). However, several instances of probabilistic treatment of the dose-response relationship have recently been documented by the Agency (USEPA, 2009). For example, USEPA recently examined the concentration-response relationship between annual average ambient PM 2.5 exposures and annual mortality. This assessment was conducted in response to a recommendation from the National Research Council that probability distributions for key sources of uncertainty be developed through formal elicitation of expert judgment in a Bayesian context (USEPA, 2009). In another example, a two-dimensional Monte Carlo analysis was conducted to examine the risk associated with *Cryptosporidium* in raw water supplies. In this study, the variability of occurrence, treatment efficiency, and the dose-response relationship for *Cryptosporidium* infection were treated in a probabilistic context (USEPA, 2009). In addition, probabilistic treatment of the Reference Dose (RfD) has been suggested to help reduce the implication of zero risk below the RfD.

In instances where sufficient data are available for the probabilistic evaluation of a chemical's toxicity, there is clearly a benefit to probabilistic treatment of the toxicity values. Quantifying uncertainties associated with the dose-response relationship such as: extrapolation uncertainties (e.g., inter-species extrapolation, low-dose extrapolation), study design uncertainties (e.g., exposure regimens, endpoint selection), calculation techniques (e.g., ED50/LD50, NOEL/LOEL, NOEC/LOEC, benchmark dose), and other factors (purity of reagents, measurement errors) is essential to evaluating how alternative decision choices impact a target population and the consequences of making a decision under a given level of certainty (USEPA, 2008; USEPA, 2009). This is especially true for more complex assessments, such as those of aggregate and cumulative exposures. If there is sufficient data to allow evaluation of the toxicity value in a

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[2] Example provided by ACC's Chlorine Chemistry Division based on analysis conducted by ToxStrategies, 3420 Executive Center Drive, Suite 114 Austin, TX 78731

probabilistic framework, inclusion of these data in PRA are essential to a comprehensive evaluation of uncertainty.

The dioxin-like compounds (DLCs)[3] represent a class of compounds that is well suited for the probabilistic treatment of toxicity. DLCs are evaluated using the toxicity equivalence methodology, in which each DLC is assigned a toxicity equivalency factor (TEF) that reflects its toxicity relative to 2,3,7,8-TCDD. The concentration for each DLC is multiplied by the appropriate TEF, and the resulting products are added to calculate a 2,3,7,8-TCDD toxic equivalent (TEQ). The current TEFs represent consensus-based values recommended by the World Health Organization (WHO) (van den Berg et al., 2006). In assigning TEFs to each congener, the WHO expert panel employed scientific judgment and a qualitative weighting scheme to identify a single point estimate based on all relative potency estimates (REPs) for a given congener. Because REPs for a specific congener may be based on a host of different endpoints, test conditions, and derivation methods, they represent a heterogeneous data set and, as a result, the REP values themselves range across multiple orders of magnitude (Haws et al., 2006 and 2009; USEPA, 2008). The range of REP values for different congeners (both vivo and vitro combined) is illustrated in Figure 1 (reproduced from Figure A-2, Haws et al., 2006).

USEPA has recognized the potential for probabilistic treatment of DLC TEF values in risk assessment. In their *Framework for Application of the Toxicity Equivalence Methodology for Polychlorinated Dioxins, Furans, and Biphenyls in Ecological Risk Assessment* USEPA identified sources of variability among REP values (e.g., precision of dose and effects measurements, calculation techniques, natural variability among organisms of the same species in their response to DLCs) and flags many sources of uncertainty (e.g., purity of chemicals, study design, measurement errors). The document proposes, “more sophisticated models may be used to combine the exposure and toxicity information into distributions that may allow for the development of probability density functions, if data are adequate” (USEPA, 2008). USEPA clearly anticipates the use of probabilistic methods for the quantification of variability and uncertainty regarding DLC toxicity.

Additionally, the 2000 USEPA Science Advisory Board (SAB) charged with reviewing the draft Dioxin Reassessment “questioned whether the uncertainty in the TEFs and the application of this approach to predicting risks due to current levels of exposure was adequately presented” (USEPA SAB 2001, p. 29). The SAB concluded that the Reassessment should acknowledge the need for better uncertainty analysis of the TEF values, and although no current method for doing so has been endorsed by the scientific community, several approaches were suggested, such as the use of probabilistic distributions of TEF values in TEQ evaluation (Finley et al., 2003). Further, the SAB concluded that available information indicates a considerable amount of variability in the REP value data that were used to derive the WHO TEF values. In addition, they concluded that although the WHO TEFs were derived based on a scientific consensus evaluation of the available REP values using defined weighted criteria for individual studies, details of the quantitative basis of this weighting scheme were not clearly presented in the description publication (van den Berg et al. 1998). These issues clearly contribute to variability and uncertainty in the application of the WHO TEF values to health risk assessment. Application of a mathematical value or percentage of the overall range of REP values, such as those described by Finley et al., (2003), would be one way to make the process of determining the specific TEFs more transparent and to provide a standard method to develop TEFs for other DLCs that may be added at a later date.

[3] It should be noted that these comments are not directed towards technical issues with respect to PCBs and TEFs. It is the opinion of the Chlorine Chemistry Division of ACC that the use of TEF values in estimating PCB mixture exposure and risk is inappropriate due to the unique mixture issues of PCBs and the PCB mixture-specific toxicity values that are more relevant.

Subsequent to the review by the 2000 USEPA SAB, the USEPA Dioxin Reassessment was updated and then reviewed by a National Academy of Sciences (NAS) panel. The NAS panel also recognized the need for characterization of variability and uncertainty with regard to the toxicity of DLCs (NAS, 2006). Specifically, the NAS panel concluded that there was a significant degree of uncertainty in the current consensus-based TEFs, and the quantitative weighting considerations that went into their establishment were not clear. As such, the NAS panel strongly recommended that the USEPA consider inclusion of uncertainty analysis of the TEF values and endorsed the recommendation of the 2000 USEPA SAB panel "that, as a followup to the Reassessment, the EPA should establish a task force to build 'consensus probability density functions' for the thirty chemicals for which TEFs have been established, or to examine related approaches such as those based on fuzzy logic" (EPA SAB 2001, p. 29).

The TEF methodology was also recently reviewed by a World Health Organization (WHO) expert panel (van den Berg et al., 2006). Although this panel once again relied upon qualitative scientific judgment as the basis for establishing the TEFs, the panel acknowledged that distributions could be used in the future once a consensus-based weighting framework had been developed (van den Berg et al., 2006). Further, the panel stated that recent papers advocating the use of a probabilistic approach for determine TEFs (Finley et al., 2003; Haws et al., 2006) provided a clear advantage because such approaches allow for better description of the level of uncertainty present in a TEF value.

Several studies have demonstrated the use of distributions for TEF values. Finley et al., (2003) suggested that the WHO TEFs are likely to be a significant source of uncertainty and variability in health risk assessments involving complex mixtures of PCDD/Fs and PCBs. To examine this issue more closely, Finley and coworkers obtained the original 1997 WHO REP database that the 1998 WHO panel relied upon to establish the 1998 TEFs (van den Berg et al., 1998). This database contained 936 REP values, of which 759 were determined to be useable. The number of REPs ranged from 117 (PCB 126) to 1 (1,2,3,7,8,9-HxCDF). Distributions were fit for congeners (where possible), and a simple weighting scheme was developed which gave higher weights based on endpoint (tumor production > P-450 induction > other), and cell lines tested (human > non-human > unknown). Weighted and un-weighted distributions were tested using concentrations of striped bass filet and blue crab muscle in a Monte Carlo PRA. It was found that upper bound PCDD/F risk was consistent with point estimates, while upper bound PCB risk increased by approximately ten-fold (weighted and un-weighted results were similar). It was hypothesized that this result reflected the location of the WHO TEF in the distribution of REP values: for PCDD/F the WHO TEF reflects an upper percentile of the REP distribution (75^{th}-99^{th} percentile) while for PCBs the WHO TEF generally reflects a central percentile (40^{th}-57^{th} percentile).

Haws et al., (2006) briefly reviewed the evolution of the TEF methodology and development of the 1997 REP database, and presented definitive criteria for evaluating REPs from different studies. The result of this evaluation was the development of a refined REP database, as well as summary statistics for congeners having more than 10 REPs (min, max, and percentiles: 25^{th}, 50^{th}, 75^{th}, 90^{th}) and congeners having less than 10 REPs (min, max, and 50^{th} percentile). Summary statistics are presented in Tables 1 and 2 (reproduced from Tables 6 and 7 of Haws et al., 2006). As a note, this refined REP database was relied upon by the 2005 WHO expert panel during their most recent review of the TEF methodology (van den Berg et al., 2006). This refined database provides the structure to assess variability in the underlying data, as well as the uncertainty inherent in the TEF values assigned to individual congeners. Building upon this work, Haws et al., (2009) have proposed a consensus-based weighting framework that incorporates consideration of multiple criteria: study type (*in vivo, in vitro*), pharmacokinetics, REP derivation quality (maximum response achieved, sufficient number of replicates, at least 3 doses plus control), REP derivation method, and endpoint (toxic, biochemical). This framework is illustrated in the flowchart in Figure 1 (reproduced from Figure 2 of Haws et al., 2009).

Utilizing this weighting framework, Haws et al., (2008) evaluated the impact of using weighted distributions of REPs by estimating the intake associated with consumption of catfish containing DLCs. This study estimated intake of DLCs using three methods: WHO TEFs, point estimate TEFs based on a series of selected percentiles from the weighted and un-weighted REP distributions, and the full weighted and un-weighted REP distributions in a Monte Carlo PRA. In addition, the intake estimates calculated with the WHO TEFs were consistent with the estimates based on the 50th percentile of the weighted and un-weighted distributions. Intake estimates based on un-weighted distributions were generally higher than those based on weighted distributions, particularly when the upper percentiles were selected. Weighting had a greater impact when percentiles > 75^{th} were selected. The ratio of PCB risk to PCDD/F risk increased when PRA was applied with TEF distributions, consistent with the results of Finley et al., (2002). The use of distributions had a greater impact on intake calculations than did the weighting process alone. These results are shown in Table 3 (Reproduced from Table 1 of Haws et al., 2008).

Urban et al., (2009) performed a risk assessment using fish tissue data for the Lower Passaic River. In Phase 1 of this assessment, multiple estimates of risk were generated: 1) a deterministic point estimate, 2) PRA using distributions for exposure parameters and the WHO 2006 TEFs, 3) a PRA using distributions for exposure parameters and DLC TEFs (including the weighting framework proposed in Haws et al., 2009). This data is illustrated in Figure 5 (reproduced from Figure 2, Urban et al., 2009). From this figure, it is clear for this example that while the use of weighted REP distributions has little impact on the PCDD/F risk, there is a substantial impact on the PCB risks; this is consistent with the findings of Finley et al., (2003) and Haws et al., (2008). Phase 2 included a more refined probabilistic analysis that incorporated distributions for the concentration associated with each congener in fish tissue. These results (also shown in Figure 2) are similar to Phase 1 results with regard to proportion of PCDD/F and PCB contribution to risk. However, it should be noted that total risk is now below the upper bound acceptable risk benchmark of 1E-4.

The impact of using distributions for toxicity criteria in PRA is an essential element of quantifying variability and uncertainty in a PRA. In particular, the use of distribution values for REP values used to derive TEFs for the evaluation of DLCs is established in the literature. There are three criteria specified by USEPA that indicate when a PRA is typically not necessary: when a screening level deterministic PRA indicates that risks are negligible, when the cost of averting the exposure are small, and when there is little uncertainty or variability in the analysis. These three criteria are infrequently met for DLCs. First, the slope factor for 2,3,7,8-TCDD is sufficiently large that the evaluation of DLCs using the TEF methodology can often lead to estimates of unacceptable risk. Second, given that a number of DLC contamination scenarios involve the ingestion of fish associated with a particular waterway, the potential cost of averting exposure is rarely small. Third, the establishment of TEF values is certainly a process in which there is documented uncertainty and variability. For these reasons, the incorporation of variability and uncertainty estimates in risk assessment involving exposure to DLCs is essential.

Tables and Figures

Table 1: Summary statistics for in vivo + in vitro REPs in the REP2004 database

Congener	N	Min	25th Percentile	50th Percentile	75th Percentile	90th Percentile	Max	WHO_{98} TEF	Rank[a]
PCDDs									
12378PeCDD	45	0.044	0.18	0.4	0.6	0.8	1.5	1	94%ile
123478HxCDD	21	0.0076	0.049	0.075	0.12	0.35	0.61	0.1	72%ile
1234678HpCDD	18	0.001	0.0073	0.01	0.028	0.042	0.1	0.01	35%ile
PCDFs									
TCDF	30	0.006	0.026	0.08	0.19	0.3	0.63	0.1	62%ile
12378PeCDF	28	0.0027	0.015	0.048	0.12	0.15	0.95	0.05	52%ile
23478PeCDF	99	0.0065	0.12	0.22	0.46	1.1	3.7	0.5	78%ile
123478HxCDF	13	0.014	0.043	0.07	0.3	0.49	4	0.1	54%ile
123678HxCDF	18	0.0031	0.03	0.072	0.098	0.15	0.16	0.1	76%ile
234678HxCDF	10	0.0085	0.034	0.21	0.31	0.32	0.32	0.1	33%ile
Non-ortho PCBs									
PCB77	49	2.0E−06	0.0001	0.00079	0.018	0.1	0.48	0.0001	25%ile
PCB81	12	0.000042	0.0041	0.0065	0.01	0.023	0.05	0.0001	2%ile
PCB126	115	0.000067	0.05	0.1	0.18	0.42	0.86	0.1	47%ile
PCB169	30	1.8E−06	0.0016	0.0055	0.063	0.53	0.77	0.01	58%ile
Mono-ortho PCBs									
PCB105	26	4.7E−07	1.1E−05	0.000081	0.0003	0.0019	0.074	0.0001	60%ile
PCB118	25	4.2E−07	7.1E−06	0.00002	0.00045	0.0021	0.075	0.0001	68%ile
PCB156	30	2.1E−06	0.000036	0.000095	0.00052	0.19	0.51	0.0005	72%ile

Note. Summary statistics reflect retained *in vivo* + *in vitro* REPs combined. Those REP values meeting the following criteria were excluded when developing the summary statistics based on the REP_{2004} Database: (1) non-numeric REPs; (2) REPs based on repetitive endpoints; (3) REPs based on repetitive studies; *(4) REPs based on a single dose level of the test or reference compound; and (5) REPs identified as invalid for other reasons (e.g., no response; REP based on* mixtures study; data omitted from final peer-reviewed publication).
[a]Percentile rank of WHO_{98} TEF relative to the distribution of REP values in the REP_{2004} Database.

Table 2: Summary statistics for congeners in the REP 2004 database having less than 10 in vivo+ in vitro REPs

Congener	N	Min	Max	50th Percentile	WHO_{98} TEF
PCDDs					
123789HxCDD	6	0.0054	0.07	0.052	0.1
123678HxCDD	5	0.031	0.2	0.043	0.1
OCDD	6	0.00025	0.0032	0.00035	0.0001
PCDFs					
123789HxCDF	2	0.11	0.2	0.15	0.1
1234678HpCDF	2	0.024	0.32	0.17	0.01
1234789HpCDF	2	0.018	0.044	0.031	0.01
OCDF	9	4.0E−06	0.0028	0.0007	0.0001
Mono-ortho PCBs					
PCB114	8	0.000072	0.0024	0.00054	0.0005
PCB123	6	3.0E−06	0.00071	0.000044	0.0001
PCB157	9	0.00004	0.002	0.00042	0.0005
PCB167	5	2.0E−06	0.00063	0.00001	0.00001
PCB189	5	2.0E−06	0.00018	0.000037	0.0001

Note. Summary statistics reflect retained *in vivo* + *in vitro* REPs combined. Those REP values meeting the following criteria were excluded when developing the summary statistics for the REP_{2004} Database: (1) non-numeric REPs; (2) REPs based on repetitive endpoints; (3) REPs based on repetitive studies; (4) REPs based on a single dose level of the test or reference compound; and (5) REPs identified as invalid for other reasons (e.g., no response; REP based on mixtures study; data omitted from final peer-reviewed publication).

Table 3: Apportionment of intake (TEQ pg/kg-day) by chemical group

Approach	PCB Intake	PCDD/F Intake	Ratio of PCB Intake to PCDD/F Intake
Deterministic			
1998 TEFs	2.56E-04	1.28E-03	0.2
2006 TEFs	3.21E-04	1.28E-03	0.3
Probabilistic			
50th Percentile			
Unweighted Probabilistic	3.85E-02	1.92E-03	20
Weighted Probabilistic	6.41E-03	1.28E-03	5
75th Percentile			
Unweighted Probabilistic	6.41E-03	1.28E-03	5
Weighted Probabilistic	1.28E-03	1.28E-03	1
95th Percentile			
Unweighted Probabilistic	3.85E-03	1.28E-03	3
Weighted Probabilistic	5.13E-04	6.41E-04	0.8

Figure 1: Weighting Framework

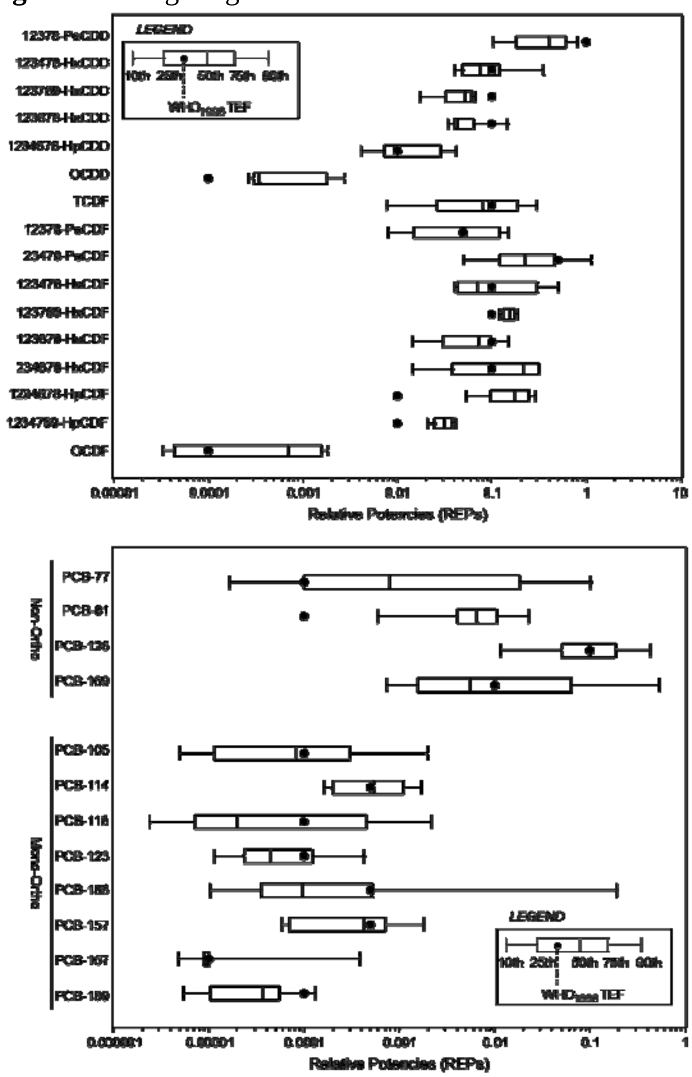
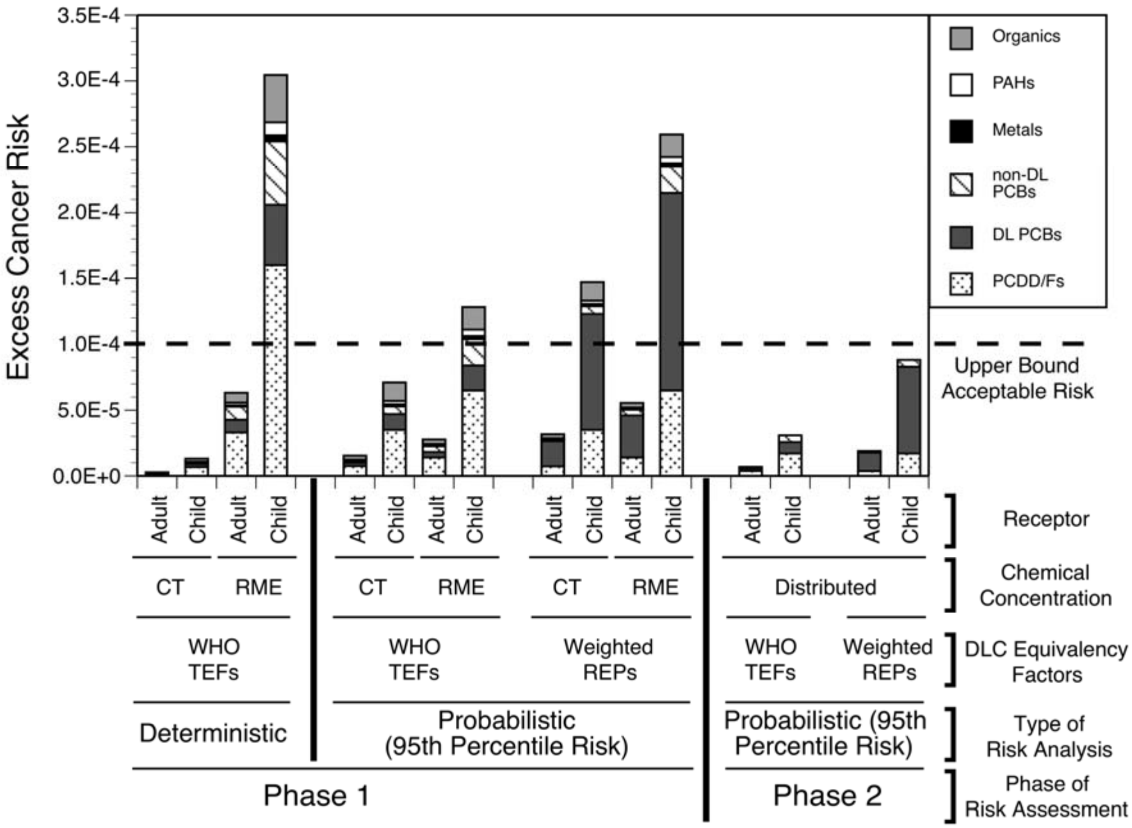


Figure 2: Excess cancer risk summary for ingestion of fish from the Lower Passaic River. The central tendency (CT) and reasonable maximum exposure (RME) estimates represent the 50th and 95th percentiles, respectively, of the sampling distribution of the composite concentration means.



References

Finley, B.L., Connor, K.T., Scott, P.K. (2003). *The use of Toxic Equivalency Factor Distributions in Probabilistic Risk Assessments for Dioxins, Furans, and PCBs. J Toxicol Environ Health, Part A.* 66: 533-550

Haws, L.C., Su, S.H., Marris, M., DeVito, M.J., Walker, N.J., Farland, W.H., Finley, B., Birnbaum, L.S. (2006).*Development of a Refined Database of Mammalian Relative Potency Estimates for Dioxin-like Compounds*. *Toxicol Sci*: 89 (1), 4-30.

Haws, L.C., Unice, K.M., Tachovsky, J.A., Harris, M.A., DeVito, J.J., Walker, N.J., Birnbaum, L.S., Farland W.H., Nguyen, L., Staskal, D.F. (2008). *Assessment of the impact of using weighted distributions of REPs to develop exposure estimates for Dioxin-Like compounds. Organohalogen Comp*.

Haws, L.C., DeVito, M.J., Walker, N.J., Birnbaum, L.S., Farland, W.H., Harris, M.A., Tachovsky, J.A., Unice, K.M., Scott, P.K., Staskal-Wikoff, D.F.(2009). Development of distributions of relative potency estimates to quantitatively assess uncertainty inherent in the TEFS for dioxin-like compounds: A proposed consensus-based weighting framework. *Organohalogen Comp*.

NAS (National Academy of Sciences).(2006). Health risks from dioxin and related compounds: evaluation of the EPA reassessment. Committee on EPA's Exposure and Human Health Reassessment of TCDD and Related Compounds. Washington, DC: National Research Council.

Urban, J.D., Tachovsky, J.A., Haws, L.C., Staskal, D.W., Harris, M.A. (2009*). Assessment of human health risks posed by consumption of fish from the Lower Passaic River, New Jersey*. *Sci Total Environ*. Doi: 10.1016/j.scitotenv.2009.03.004

USEPA SAB (U.S. Environmental Protection Agency Science Advisory Board). 2001. Dioxin Reassessment-An SAB Review of the Office of Research and Development's Reassessment of Dioxin. Review of the Revised Sections (Dose Response Modeling, Integrated Summary, Risk Characterization, and Toxicity Equivalency Factors) of the EPA's Reassessment of Dioxin by the Dioxin Reassessment Review Subcommittee of the EPA Science Advisory Board (SAB). EPA-SAB-EC-01-006. Science Advisory Board, Washington, DC. May 2001 [online]. Available: http://www.epa.gov/ttn/atw/ec01006.pdf.

USEPA (U. S. Environmental Protection Agency) (2008).*Framework for Application of the Toxicity Equivalence Methodology for Polychlorinated Dioxins, Furans, and Biphenyls in Ecological Risk Assessment.* Office of the Science Advisor. EPA 100/R-08/004.

USEPA (U. S. Environmental Protection Agency) (2009).*Using Probabilistic Methods to Enhance the Role of Risk Analysis in Decision-Making With Case Study Examples*. Risk Assessment Forum, PRA Technical Panel Working Group. EPA/100/R-09/001.

van den Berg, M., L. Birnbaum, A.T. Bosveld, B. Brunstrom, P. Cook, M. Feeley, J.P. Giesy, A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X. van Leeuwen, A.K. Liem, C. Nolt, R.E. Peterson, L. Poellinger, S. Safe, D. Schrenk, D. Tillitt, M. Tysklind, M. Younes, F. Waern, and T. Zacharewski. (1998).Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. *Environ Health Perspect*. 106(12):775-792.

van den Berg M, Birnbaum LS, Denison M, De Vito M, Farland W, Feeley M, et al. (2006). The2005 World Health Organization reevaluation of human and mammalian toxic equivalency factors for dioxins and dioxin-like compounds. *Toxicol Sci*. 2006;93(2):223–41.

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Re: EPA–HQ–ORD–2009–0645; External Peer Review Draft — *Using Probabilistic Methods To Enhance the Role of Risk Analysis in Decision-Making With Case Study Examples*, 74 Fed. Reg. 41695 (Aug. 18, 2009)

Dear Dr. Gallagher:

Enclosed please find the comments of The General Electric Company (GE) on the *External Peer Review Draft – Using Probabilistic Methods To Enhance the Role of Risk Analysis in Decision-Making with Case Study Examples,* 74 Fed. Reg. 41695 (Aug. 18, 2009). As explained more fully in our comments, GE supports and encourages EPA's use of probabilistic methods to perform risk assessments.

We appreciate your consideration of these comments.

Sincerely,

Patricia Kablach Casano
Counsel, Government Affairs
Corporate Environmental Programs

Enc.

Comments of the General Electric Company
on the
External Review Draft (18 August 2009)
Using Probabilistic Methods to Enhance the Role of Risk Analysis in Decision-Making With Case Study Examples

INTRODUCTION

The General Electric Company (GE) appreciates the opportunity to comment on EPA's External Review Draft (18 August 2009): *Using Probabilistic Methods To Enhance the Role of Risk Analysis in Decision-Making With Case Study Examples* (*Draft White Paper*). Probabilistic risk assessment encompasses a variety of advanced techniques that evaluate and characterize the stochastic variability and uncertainty inherent in the risk assessment process. Probabilistic methods provide the means to obtain a broader and more accurate view of potential risks to individuals and populations than are afforded by more limited deterministic (point-estimate) methods. As stated in the *Draft White Paper (Manager's Summary,* p. 2*)*, numerous advisory groups, including the EPA Science Advisory Board and the National Academy of Sciences, have requested EPA to enhance its use of probabilistic risk assessment (PRA) in its decision-making. Such use would lead to a more comprehensive characterization of uncertainty and variability in each step of a risk assessment, and improve the transparency and quality of the assessment. The *Draft White Paper* will serve as a useful introduction to PRA and its application as a means to examine and address uncertainty and variability in risk assessment. Moreover, as EPA recognizes, use of PRA enhances risk management and improves confidence in decision-making. It is, therefore, essential that PRAs be based on the best available science, and that the results of PRAs be given adequate consideration, peer review and acceptance. They should not be ignored or used only to justify deterministic risk estimates.

COMMENTS

Good Intentions to Apply PRA Methods Should Be Bolstered In Practice

Overall, the *Draft White Paper* represents a positive advancement in risk assessment, and has the potential to improve risk management by promoting the use of PRA in decision-making. PRA techniques allow the consideration of a range of estimates in each step of the risk assessment process, instead of relying on a single point estimate to evaluate exposure. Likewise, PRA methods are not limited to reliance upon a single risk value as the basis for making a risk management decision. The Draft White Paper (Section 3.1, pg 21) states:

> Using PRA, one can obtain insight regarding whether one risk management strategy is more likely to reduce risks compared to another, and by how much. The methodology facilitates the investigation of potential changes in decisions that may result from the collection of additional information that could better characterize variability and potentially reduce uncertainty and helps determine how expenses incurred by activities to reduce uncertainty are offset by improved decision-making capabilities gained from the acquisition of that knowledge. PRA can facilitate the construction and simultaneous consideration of multiple model alternatives. Probabilistic methods offer a number of tools designed to promote robust management and increased confidence in decision making through the incorporation of input variability and uncertainty characterization and prioritization in risk analyses. For example, sensitivity analyses can be used to identify influential knowledge gaps involved in the estimation of risk, allowing for improved transparency and the ability to more clearly communicate or articulate the most relevant information to decision makers and stakeholders.

GE supports the recommendations of the *Draft White Paper* (Section 2.2, p. 13) concerning the application and use of PRA in the following situations:

- When a screening level deterministic risk assessment indicates that risks are possibly higher than a level of concern and, therefore, a more refined assessment is needed;
- When the consequences of using potentially biased point estimates of risk are unacceptably high;
- To estimate the value of collecting additional information to reduce uncertainty;
- When significant equity issues are raised by inter-individual variability;
- To identify promising critical control points and critical levels when evaluating risk management alternatives; and
- To rank exposure pathways, sites, contaminants, and so on for purposes of prioritizing model development or further research.

Without question, probabilistic tools reduce uncertainty and provide a more detailed comparison of protectiveness and costs for risk managers to use when making risk management decisions.

EPA Must Fully Embrace the Use of PRA for Toxicity Values

Although the *Draft White Paper* supports and encourages the use of PRA, it lists a number of challenges, including the application of PRA techniques in toxicity assessment:

> Although highly sophisticated human exposure assessment and ecological risk applications have been developed, use of PRA models to evaluate toxicity data has been very limited. Scientific, technical, and science policy discussions are needed in this area. [Draft White Paper, Section 3.2, p. 23.]

GE supports the use of PRA methods to address uncertainty associated with the use of toxicity criteria and benchmarks, including cancer slope factors (CSFs) and reference doses (RfDs) listed on IRIS. One advantage of using PRA methods to evaluate toxicity values is alluded to on page 15 of the *Draft White Paper,* which refers to use of a probabilistic RfD. This discussion should be expanded, and EPA should endorse and promote the use of PRA methods to address toxicity values, because, as explained below, EPA and GE have demonstrated that such use is feasible.

More than a decade ago, GE provided financial and in-kind support for the first private sector Cooperative Research and Development Agreement (CRADA) under the *Federal Technology Transfer Act* with EPA in the field of regulatory toxicology and risk assessment. The CRADA provided the framework for cooperative research between EPA researchers and scientists supported by GE to develop PRA methods for characterizing the uncertainty in reference dose estimates. Six papers were published in the peer-reviewed literature, and more than a dozen short papers, conference presentations, and symposia at national scientific meetings were produced as work products.

In spite of these accomplishments, EPA has been reluctant to embrace these techniques in toxicity assessment. EPA. 2001. For example, in a previous EPA white paper, the Agency dismissed the application of PRA techniques for addressing toxicological uncertainty (EPA. 2004, p. 40). The Agency also has inappropriately labeled at least one of the important published papers that emerged from the CRADA project -- Swartout et al. 1998 -- as a "preliminary work." EPA. 2004. In fact, that paper developed a method for expressing an RfD in probabilistic terms for practically any chemical. The paper won an award from the Society of Toxicology as the best published paper in the field of risk assessment for that year, and was far from a "preliminary work." The method described Swartout et al. 1998 uses the typical RfD equations, but replaces the uncertainty factors with distributions. It can be used to determine toxicological uncertainty for use in non-carcinogenic risk assessments, and the results should be presented as an integral part of the toxicity information for chemicals published on IRIS.

The Agency's reluctance to embrace PRA gives rise to the perception that EPA has used its "policies" to avoid the use of the best available science when it appeared that the use of that science would lead to outcomes that run contrary to the Agency's preconceived outcome. One pertinent example is the unwillingness of EPA Region 1, in the context of the Housatonic River Human Health Risk Assessment, to use PRA to characterize the uncertainties associated with toxicity values as recommended by an EPA-convened peer review panel. In reviewing EPA's draft Housatonic River Human Health Risk Assessment, the peer review panel generally agreed that the uncertainties associated with the toxicity values were substantial, and should be included in the evaluation of uncertainties in the risk estimates. EPA Region 1 ignored these recommendations, and did not include a quantitative evaluation of those uncertainties in the final Housatonic River Human Health Risk Assessment, even though GE had commissioned and submitted to the administrative record a PRA that included toxicity distributions (AMEC. 2003). Nevertheless, GE is encouraged by EPA's articulated conviction in the *Draft White Paper* that PRA can be an effective and powerful tool throughout the entire risk assessment process.

PRA Models Must Be Scientifically Defensible

Uncertainty is inherent in all risk assessments; therefore, it is crucial that the risk assessment process treat uncertainties in a manner that is transparent and scientifically defensible. It also is imperative that probabilistic models be transparent and scientifically defensible. *Case Study 5: One-Dimensional Probabilistic Risk Analysis of Exposure to Polychlorinated Biphenyls (PCBs) via Consumption of Fish from a Contaminated Sediment Site* actually is an example of a probabilistic model that lacked transparency, was poorly described, inconsistent with EPA guidance, and inadequate in its characterization of the uncertainties in the exposure estimates. As described in more detail in Section 4.0 of the attached Comments of General Electric Company on Hudson River PCBs Superfund Site Reassessment RI/FS Phase 2 Human Health Risk Assessment (Sept. 7, 1999)(*Hudson Comments*)[1], the key limitations of this probabilistic model are:

- The model failed to satisfy the criteria for acceptance of probabilistic analyses established in EPA's guidance for use of Monte Carlo analyses (EPA. 1997b). The probabilistic analysis failed to meet many of the criteria, including criteria for model design and documentation of the assessment.
- Although acknowledging the importance of modeling angler exposures as a series of separate annual events, EPA's model failed to incorporate this approach, and instead modeled angler doses as single events that often lasted more than 40 years. As a result, the model assumed that anglers consumed unrealistic amounts of fish harvested from the same locations, cooked in the same fashion, and composed of the same mixture of species every year for periods longer than 40 years. This approach is not remotely realistic, because an individual's behavior does, in fact, vary over time. An assessment cannot be truly probabilistic if it ignores the range of variation of the behaviors being assessed.

Distributions Must Be Based on the Best Science

A principal advantage of a probabilistic assessment is that it produces a distribution and range of likely exposures and risks. This is preferable to a deterministic assessment, which yields a point estimate of exposure at some unknown point in the range of possible risks. PRA, through statistical techniques, can be used to analyze sources of variability and uncertainty in the exposure and risk assessments. As is the case with any model, PRA models are only as good as the data that are used. If PRAs rely on conservative default assumptions instead of real data, the quality of the results will be in question. The same is true when data distributions are censored inappropriately to exclude values that are not to the liking of the analyst. With

[1] The attached copy of the *Hudson Comments* does not include Attachment A to that document, as it is not relevant to these comments.

enhanced use of PRA, EPA hopes to reduce criticism that their assessments are overly conservative and unrealistic. To achieve that goal, the best science must be applied transparently in developing probability distributions and other data necessary for PRAs.

In *Case Study 5: One-Dimensional Probabilistic Risk Analysis of Exposure to Polychlorinated Biphenyls (PCBs) via Consumption of Fish from a Contaminated Sediment Site*, more robust exposure data were available, but EPA chose to use more conservative data in its PRA. *Hudson Comments*, Att. B. For describing a surrogate distribution of fish consumption rates among anglers and their families fishing the upper Hudson River, this PRA relied on Connelly et al. (1992), which showed some fisherman eating up to 1,000 fish meals a year. EPA, however, has recognized the significant limitations of that study, and did not designate it as a "key study" to evaluate sport-caught freshwater fish consumption by recreational anglers. EPA. 1997a. Accordingly, EPA should have used other surveys (e.g., Connelly et al. 1996 and Ebert et al. 1993) that were specifically designed to measure fish consumption by recreational anglers. Those surveys would have provided a stronger basis for the consumption rate distribution than the Connelly et al. 1992 survey data. Results of EPA's 1-dimensional Monte Carlo analysis of exposure to Hudson River sediments via consumption of contaminated fish would have been more realistic if more robust data had been used for fish consumption.

Because of these deficiencies, EPA's PRA for the upper Hudson River, as recounted in *Case Study 5*, should not be included in the *Draft White Paper* as a positive example.

PRAs Can Be Used for Sensitivity Analysis If Properly Structured and Conducted

Appendix A to the *Draft White Paper* (p. 29) states:

> Sensitivity analysis is complementary to probabilistic methods. There are many types of sensitivity analysis methods, including, for example, simple techniques that involve changing the value of one input at a time and assessing the effect on an output and statistical methods that evaluate which of many simultaneously varying inputs contributes the most to the variance of the model output.

Sensitivity analysis is an important tool for evaluating the most sensitive inputs. *Case Study 3: Probabilistic Assessment of Angling Duration Used in Assessment of Exposure to Hudson River Sediments via Consumption of Contaminated Fish*, however, is an example of a sensitivity analysis where EPA focused on factors that would only have a minimal impact on the final estimates of risk, and disregarded factors that would have a significant impact.

As described in the attached *Hudson Comments* (Att. C, p. 8), in Case Study 3 EPA deemed exposure duration to be a sensitive input, even though the Agency's risk assessment stated that there was little difference between the distributions of exposure duration that were based upon residential mobility and those that were based jointly upon residential mobility and cessation of angling. Exposure duration therefore would likely have only a minimal impact on the final estimates of risk. On the other hand, EPA excluded factors that have a major impact on the risk estimates, including uncertainty in the cancer slope factor and the reference dose, angler recall bias, inter-year variation in fish consumption rates, and use of consumption data from multiple water bodies. For a sensitivity analysis to be meaningful, it is essential to avoid an arbitrary selection of inputs. Failure to consider important sources of uncertainty defeats the purpose of an analysis.

EPA's PRA for the upper Hudson River, as depicted in *Case Study 3*, is a poor example that should not be included in the final version of this otherwise fine introduction to probabilistic techniques and practices for examining and addressing uncertainty, variability, and realism in risk assessment.

REFERENCES

AMEC. 2003. *Attachment H: Evaluation of EPA's Monte Carlo and Microexposure Event Analyses of the Fish and Waterfowl Consumption Scenarios,* in: *Comments of the General Electric Co. on the U.S. Environmental Protection Agency's Human Health Risk Assessment for the Housatonic River Site – Rest of River*. AMEC Earth & Environmental, Portland, ME. July 28. 61 p.

Connelly, N.A., B.A.Knuth & C.A.Bisogni. 1992. *Effects of the Health Advisory Changes on Fishing Habits and Fish Consumption in New York Sport Fisheries*. Human Dimension Research Unit. Cornell University (Report for the New York Sea Grant Institute Project No. R/FHD-2-PD).

Connelly, N.A., B.A. Knuth, and T.L. Brown. 1996. Sportfish consumption patterns of Lake Ontario anglers and the relationship to health advisories. North American Journal of Fisheries Management 16:90-101.

Ebert, E.S., N.W. Harrington, K.J. Boyle, J.W. Knight, and R.E. Keenan. 1993. Estimating consumption of freshwater fish among Maine anglers. N. Am. J. Fish. Mgt. 13:737-745.

EPA. 1997a. *Exposure Factors Handbook*. Office of Research and Development National Center for Environmental Assessment. EPA/600/P-95/002Fa.

EPA. 1997b. *Policy for Use of Probabilistic Analysis in Risk Assessments at the U.S. Environmental Protection Agency*, Office of Research and Development, Washington, DC. May 15.

EPA. 2001. Risk Assessment Guidance for Superfund: Volume 3 - (Part A, Process for Conducting Probabilistic Risk Assessment). Final. United States Environmental Protection Agency, Solid Waste and Emergency Response. EPA 540-R-02-002. December.

EPA. 2004. *An Examination of EPA Risk Assessment Principles and Practices*. Staff Paper Prepared for the U.S. Environmental Protection Agency by members of the Risk Assessment Task Force. Office of the Science Advisor, Washington, DC.

Swartout, J.C., P.S. Price, M.L. Dourson, H. Carlson-Lynch, and R.E. Keenan. 1998. A probabilistic framework for the reference dose. *Risk Anal.* 18(3): 271-282.

Comments of General Electric Company on Hudson River PCBs Superfund Site Reassessment RI/FS Phase 2 Human Health Risk Assessment

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1.0 Introduction and Executive Summary

General Electric Company (GE) submits these comments on EPA's Phase 2 Report – Review Copy; Further Site Characterization and Analysis, Volume 2F - Human Health Risk Assessment, Hudson River PCBs Reassessment RI/FS (HHRA).

There are three major aspects of the HHRA that require emphasis:

1. **No Unacceptable Present Risk.**

The crucial central conclusion of EPA's assessment of risk to human health is that there is no unacceptable risk today from the PCBs in the sediments of the Upper Hudson River.[1] There is no such risk to those who swim, wade or boat on the River or to those who drink the River water. There is no such risk from breathing the air near the River. Under the present catch and release fishery, EPA did not find any such risk to anglers or fishermen on the Upper Hudson. The Agency did not contend that the catch and release fishing regulations were being violated in any material manner. These are important findings: the present conditions on the Upper Hudson River do not present any unacceptable risk to human health.

2. **Hypothetical Risk Relies On Highly Implausible Assumptions**.

EPA makes a number of highly implausible assumptions in order to develop the scenario in the assessment that claims possible risks on the Hudson which may be used to justify a substantial and intrusive "remedy" in the River:

- catch and release fishing is abandoned
- anglers, or at least a few anglers, kill and eat extraordinarily large amounts of fish for extraordinarily long periods of time
- these anglers only eat fish from the Upper Hudson River

[1] The Upper Hudson River is the 40 mile stretch between Hudson Falls and the Federal Dam at Troy. For reasons explained previously to the Agency, GE maintains its position that the Hudson River PCBs Superfund Site encompasses only these 40 miles and does not extend to the Lower Hudson River.

- the future PCB concentrations in fish are calculated from a base which sets the PCB content higher than it is today

EPA makes two calculations of risk to anglers. In one:

- anglers fish in the River every year for forty-one years
- anglers eat fish from the River at the rate of half a pound of fish every week

In the second:

- some anglers fish in the River every year for up to sixty years
- some anglers eat up to 600 meals of half a pound of fish every year

These scenarios are beyond credibility.

3. **Recent Major Study Shows No Adverse Health Effects From PCBs**.

The Agency adopts a view on the toxicity of PCBs that discounts the latest and most thorough study of the workers in GE's capacitor plants which shows that more than 7000 workers who were highly exposed to PCBs are now as healthy as the general public. Among these 7000, there were fewer cancer deaths than expected from national or local rates. The mortality rates did not exceed the national and local rates for any other disease. These are the facts that count: people actually exposed – at high concentrations – to the PCBs now found in the Upper Hudson River are healthy. More than twenty years after the use of PCBs stopped at the two GE plants there is no evidence of adverse health effects in the exposed population.

In a disservice to public understanding, EPA chose not to underscore these three facts about the Upper Hudson:

- There is no unacceptable risk today from PCBs in the Upper Hudson River. One can drink, swim, wade and boat on the River without fear. Catch and release fishing is protective of human health.

- There is no evidence that people in the Hudson River plants who were actually exposed to the PCBs at high concentrations show any adverse health effects attributable to PCBs. They are as healthy as the general population.

- The calculation of possible risk, based on animal studies rather than human epidemiology, is built on a series of highly implausible assumptions of how fishermen would behave if catch and release fishing were abandoned.

Moreover, in making its calculations of risk, EPA's assessment was poorly and inadequately designed, contains calculation errors, and relies on inaccurate or inappropriate assumptions to such an extent that the risk calculations are vastly overstated and unreliable. As a result, the assessment is so seriously flawed that it should not serve as the scientific basis for decision-making for the Hudson River.

EPA Downplays Important Findings Of No Risk From PCBs While Emphasizing A Hypothetical PCB Risk Scenario.

EPA acknowledges that it found that PCBs in the Hudson River present no material risk to those who use the river for swimming, wading, boating and other recreational uses. These findings are downplayed in the assessment and in EPA's public statements. EPA chose to emphasize the sole hypothetical risk it identified – a flawed conclusion that someone who eats large amounts of fish from the Upper Hudson River for many years may face an elevated health risk. This faulty conclusion was the heart of EPA's public presentations -- which did not fairly and directly state that this risk does not exist today because for twenty years it has been illegal to keep fish from the Upper Hudson River.

In fact, EPA does not contend that anyone is presently eating fish, or has eaten fish, from the Upper Hudson River in the amounts and for the number of years assumed in its risk calculations. This is supported by data from the conservation officer patrolling the Upper Hudson; from mid-1995 to mid-1998, he checked more than 1400 anglers and issued only nine tickets and three warnings. EPA's risk result is based on implausible and incorrect assumptions, some of which do not pass simple common sense tests. EPA's risk result relies on the highly improbable scenario that someone will eat one-half pound or more of fish he caught in the River every week of every year for forty years.

EPA's Assessment Overstates the Toxicity of PCBs.

EPA's assessment uses excessively high toxicity values based on animal studies and improperly rejects persuasive evidence from more than 20 human epidemiological studies. EPA's preference for animal studies and default assumptions in the face of the actual human data is arbitrary and capricious. There have been studies of the worker populations in GE's Hudson River plants over the past 20 years that demonstrate that the cancer and non-cancer toxicity of PCBs is significantly lower than EPA estimates. These studies focused not on laboratory animals but on the very workers who were exposed to PCBs daily – the PCBs that were discharged to the Hudson River and are that are now in river fish. Studies by a broad array of experts – from Dr. Renata Kimbrough to scientists from the New York State Department of Health and NIOSH - have demonstrated that these workers are just as healthy as the rest of the general population. A weight-of-evidence assessment of the epidemiological and clinical studies shows that there is no credible evidence that PCBs cause cancer in humans.

Analyzing the possible non-cancer human health effects of PCBs by the weight-of-evidence approach leads to the conclusion that there is little, if any, evidence that PCBs cause any adverse effects in humans at environmental levels. EPA is on unsure scientific footing in this area; the assessment admits that the safe level for non-cancer effects may be significantly higher than the level used in the assessment. Indeed the non-cancer human health effects are plainly speculative since particular adverse human health effects are not identified.

EPA's Numerous Flawed Assumptions Result in an Overstatement of Hypothetical Exposure of Anglers to PCBs in the Hudson River.

EPA materially overstates the hypothetical future exposure of anglers to PCBs in Hudson River fish because of a series of scientific errors.

- EPA improperly relies on preliminary and flawed models to project PCB levels into the future despite EPA's acknowledgement that these models are undergoing significant revisions and have not been peer reviewed.

- EPA's assessment improperly relies on a study to derive fish consumption rates that was not designed for that purpose. Indeed, EPA's own guidance does not categorize this as a "key" study. The appropriate studies, designed to measure how much fish anglers eat, show much lower rates of fish consumption than the study used by EPA which shows some fishermen eating up to 1000 fish meals a year. It is not plausible to assume that only fish caught in the Upper Hudson River will be eaten for breakfast, lunch, and dinner.

- Finally, the assessment improperly defines the angler population, miscalculates and underestimates the annual mobility rates of anglers and does not take full account of the literature on cooking losses of PCBs. These errors collectively lead to an overestimate of potential exposure to PCBs.

EPA Incorrectly and Improperly Dismisses the Findings of the Largest Epidemiological Study of PCB-exposed Workers Ever Conducted.

The study of workers in GE's capacitor plants on the Hudson River found that, despite the high PCB levels to which these workers were exposed and that were reflected in high blood levels, death rates from cancer or other diseases were no higher than national or local rates. This is the latest in a series of studies that consistently reached similar results. EPA produces no evidence that these workers actually exhibited any unusual adverse health effects. Nevertheless, EPA erroneously dismisses the findings of the study because of alleged limitations:

- EPA claims that more than 75% of the workers studied never worked with PCBs. In fact, all workers at the plants inhaled and touched PCBs each day at concentrations significantly greater than found in the environment.

- EPA incorrectly claims that the actual level of PCB exposure to workers could not be confirmed. Data are available confirming the extremely high air levels of PCBs to which these workers were exposed: air levels were measured and independent research examined plant conditions.

- EPA claims that "less than 25% of the workers" were employed for less than one year and that such exposure is not comparable to long-term environmental exposures. It is unclear how EPA derived this estimate. The 90-day cut-off for inclusion in the study is consistent with and longer than cut-offs used in other epidemiological studies referenced with approval by EPA.

- EPA claims that the average age of the workers at the end of the study period is too young to draw conclusions. In fact, many older workers were included within the study. Further, the study included an age-adjusted examination of the workers' health and concluded that PCBs were not associated with higher incidence of death.

- EPA claims that the study did not examine "vulnerable populations," including children and the elderly. The study did include elderly and people with existing health problems. Given the fact that it was an occupational study, it was not designed to examine children.

The heart of EPA's attack on the study of capacitor workers is that the workers were not exposed to much PCB. This defies common sense and the evidence. The workers had levels of PCBs in their blood well above background, far higher than is found in any segment of the population today or was generally the case in the 1970s. The PCBs came from exposure in the plants. High levels of PCBs were measured in the air throughout the plant and an independent study of capacitor plants showed that high level air exposures were typical. In fact, the air circulation system in the plants combined with working with PCBs in large quantities in open spaces inevitably led to high air levels. EPA's criticisms are plainly disingenuous; the Agency has made no similar critique of earlier studies of this same cohort of workers. EPA's erroneous assertions about the Kimbrough study are a sloppy attempt to dismiss a study that was prepared and reviewed by some of the world's most respected and experienced experts in this field.

The Probabilistic Model Used in the HHRA is Flawed, Overestimates Risk to Anglers, and Fails to Confirm to EPA's Guidance

EPA's probabilistic modeling of angler PCB exposure lacks transparency, is poorly described and inconsistent with EPA guidance, and is inadequate in its characterization of the uncertainties in the exposure estimates:

- Although acknowledging the importance of modeling angler exposures as a series of separate annual events, the model used in the assessment fails to incorporate this approach, instead modeling angler doses as single events that often last more than 40 years. As a result, the model assumes that anglers consume unrealistic amounts of fish harvested from the same locations, cooked in the same fashion, and composed of the same mixture of species every year for periods longer than 40 years.

- The model inappropriately evaluates non-cancer risks to anglers exposed for only one or two years as if those exposures occurred over seven or more years. This leads to a significant overestimate of non-cancer risks to these anglers.

- The assessment does not adequately describe the Agency's probabilistic model. The failure to document the model properly, including presenting the model code, information on the random number generator used in the model, information on post-analysis manipulation of model output, and information on key model inputs, effectively impeded GE's ability to review and comment on the model. GE has recently received additional information from EPA and will submit supplemental comments following the company's review.

- The model fails to meet the standards established by EPA guidance for Monte Carlo models, including deficiencies in model design and documentation.

- The Agency fails to separate uncertainty and variability in its risk estimate and does not provide a quantitative analysis of uncertainty although methods are available for doing so. The Agency's "sensitivity analyses" are useful for identifying factors that contribute to the uncertainty in risk estimates, but are no substitute for a quantitative characterization of the uncertainty associated with the Agency's estimate of risk.

- The Agency's selection of factors to consider in its sensitivity analyses is arbitrary. The Agency failed to consider important sources of uncertainty in these analyses (e.g., uncertainty in toxicity, angler recall bias, inter-year variation in fish consumption rates and use of consumption data from multiple water bodies), while evaluating sources of uncertainty that were not appropriate (e.g., location) or of minor importance.

Although EPA's analysis is flawed, it is nevertheless apparent that the future risks of eating fish from the Upper Hudson River are clearly limited. It is important to retain focus on the central issue of whether a remedy will materially accelerate the time at which people can eat fish from the Upper Hudson. Nothing in this risk assessment alters the basic facts that natural recovery will lead to edible fish in the not too distant future and a remedy such as dredging will not materially accelerate that date.

2.0 The HHRA Overstates the Toxicity of PCBs

The HHRA overstates the toxicity of PCBs in the Upper Hudson River by relying on extremely conservative estimates of PCB toxicity that are based solely on the results of laboratory studies of animals. For estimating cancer risk, the HHRA uses a "cancer slope factor" (CSF) derived from studies in which particular strains of laboratory rats have been fed massive doses of PCBs. For estimating noncancer risk, the HHRA uses "reference doses" (RfDs) derived from laboratory studies of Rhesus monkeys. Thus, the assessment of the health risks of PCBs gives inadequate consideration to the human epidemiological data as well as data that would assist in assessing the relevance of the animal studies to the potential effects of PCBs on people.

Relying primarily on animal data to assess the risks posed by a chemical may be appropriate in cases where little data exists on the effects of the chemical in humans. This approach, however, is wholly inappropriate in the case of PCBs because extensive information exists on the actual health effects of PCBs in humans and the relative sensitivity of humans and animals to PCBs. Moreover, the risk assessment approach EPA has taken with respect to PCBs is contrary to EPA guidance. As set out in detail in Attachment A, the human epidemiological data, as well as information on the mechanisms by which PCBs are metabolized in humans and animals, is invaluable in assessing both the potential cancer and noncancer effects of PCBs. Accordingly, EPA should use <u>all</u> of the available data and a weight-of-evidence approach to reassess the health risks posed by PCBs and to derive a new CSF and new RfDs that are consistent with this data.

In addition, the HHRA mistakenly dismisses the findings of Kimbrough et al. (1999) by alleging several limitations in that study. As we show below, EPA's contentions are unfounded. EPA should also incorporate the uncertainty factors used to derive the PCB RfD directly into its probabilistic model to provide a more realistic assessment of non-cancer PCB risks to the hypothesized Hudson River angler. Finally, EPA properly rejected the use of "Toxic Equivalency Factors" in the HHRA, as this would have added unreasonable uncertainty to its risk estimates.

2.1 EPA Should Have Used the Weight-of-Evidence Approach to Assess the Potential for PCBs to Cause Adverse Effects

Attachment A provides a detailed review of the relevant toxicological data and demonstrates how EPA can use the available epidemiological evidence in a weight-of-evidence approach to assess the potential for PCBs to cause adverse effects in humans. The major items discussed in Attachment A are summarized below.

The weight-of-evidence approach to human health risk assessment, which has been endorsed by EPA, is justified by several important scientific findings, including that chemicals often have different effects in human and animals, that the sensitivities of humans and animals to the same health effect can vary widely, and that studies of health effects in both humans and animals can vary greatly in quality, relevance and statistical power. Given the large human epidemiological database for PCBs, as well as the extensive knowledge that has accumulated regarding metabolism of PCBs, failure to use the weight-of-evidence approach in PCB risk assessment leads to systematic exclusion of highly relevant and probative data.

Although laboratory studies indicate that PCBs promote tumors in certain strains of rats, the weight of the evidence from the human epidemiological studies demonstrates that there is no credible evidence that PCBs cause cancer in humans. This view is shared by numerous respected scientists and has recently been confirmed by the results of the largest PCB epidemiological study yet performed (Kimbrough et al. 1999). This study found no association between high dose human exposure to PCBs and deaths from cancer or any other disease.

Although the weight of the evidence shows that PCBs are not human carcinogens, it is nevertheless possible to calculate an "upper bound" CSF from one or more of the studies. The CSFs that can be derived from the human epidemiological studies are 100 to 3,000 fold lower than the CSF EPA has derived from rat studies (TERRA 1993). EPA should proceed to derive a CSF for PCBs from the epidemiological data, relying primarily on the findings of Kimbrough et al. (1999).

Application of the weight-of-evidence approach to studies of the noncancer human health effects of PCBs also leads to the conclusion that there is little, if any, evidence that PCBs cause any adverse effect in humans at environmental exposure levels. This conclusion is shared by many experts in the field and is supported by the Agency for Toxic Substances and Disease Registry in its draft update to the Toxicological Profile for PCBs. Although recent studies of cohorts in Michigan, North Carolina and the Netherlands have been cited by some as evidence that PCBs can have minor and temporary health effects at environmental doses, these studies have come to disparate conclusions, which suggests that factors other than PCB exposure are causing the reported effects. Moreover, the studies reporting the most potentially significant effects are flawed in many respects, including serious problems with the definition of the "high" and "low" exposure groups within the cohort, analytical problems in quantifying and interpreting PCB concentrations in fish and blood samples from the cohort, failure to quantify other potential neurotoxicants, lack of internal consistency, and methodological problems. Thus, these studies do not, in fact, provide credible evidence of the claimed health effects.

The noncancer human health data, along with scientific findings on the mechanisms by which PCBs cause adverse effects in certain animal species, should be used by EPA to reevaluate its current RfD for PCBs. EPA's RfD for Aroclor 1254, which was used to assess Hudson River PCB risks through the fish ingestion pathway, is based on a study of Rhesus monkeys that has little relevance to assessing human noncancer risks. The immunological findings of the study clearly do not demonstrate clinically significant effects. Moreover, the minor dermal and ocular effects reported in Rhesus monkeys are of little or no relevance to humans because such effects are not observed in humans at similar exposures and the reasons for this are apparent from an understanding of the differences in metabolic pathways in Rhesus monkeys and humans. In fact, the data indicate that humans are 15 times less sensitive to PCBs than Rhesus monkeys. Accordingly, EPA should reassess its current RfD for Aroclor 1254 to take into account the extensive human health data which demonstrate that the RfD is based on a gross exaggeration of the potential human health risks of PCBs.

Finally, EPA's application of the IRIS-derived value in the HHRA is contrary to Agency guidance on the use of IRIS values in Superfund Risk Assessments (EPA, 1993), which explains that using IRIS values in Superfund risk assessments is not mandatory and that the Agency must consider other available credible and relevant toxicological information. The epidemiological information not considered in the development of the IRIS PCB toxicological values falls squarely within the type of information that the guidance requires EPA to consider.

2.2 EPA Incorrectly Dismissed the Findings of the Kimbrough Study (Kimbrough et al., 1999)

Kimbrough et al. (1999) recently completed a follow-up study of the same cohort examined in four previous studies: Taylor (1988), Nicholson (1987), Brown (1987), and Brown and Jones (1981). The cohort consisted of workers and managers at GE's Hudson Falls and Fort Edward capacitor manufacturing facilities. This study, the largest study of PCB-exposed workers ever conducted, found no association between actual human exposure and deaths from cancer or any other disease and confirmed the findings of the previous studies of the GE cohort. The cohort consisted of 4,062 men and 3,013 women who worked between 1946 and 1977. The average follow-up time for the workers was 31 years, providing a sufficiently long latency period in which to determine whether there was a statistically significant increase in mortality due to cancer or other causes. The cohort was followed through 1993, providing 120,811 person years of observation for men, and 92,032 person years of observation for women. There were 763 (19 percent) deceased males and 432 (14 percent) deceased females. Death certificates were available for 98.5 percent of the decedents and only 1.3 percent of the cohort was lost to follow-up. Standardized mortality rates (SMRs) were calculated using both U.S. and local county mortality tables. The major findings of the Kimbrough study are as follows:

- The workers' exposure to PCBs resulted in significantly higher blood concentrations of PCBs than those found in the general population in the 1970s and 80s and much higher than current levels.

- Among all of the workers, including those classified as having the highest PCB exposure, no statistically significant increase in deaths due to cancer or any other disease was found. There was also no statistically significant increase or decrease in mortality associated with the length of employment or latency.

- The death rate due to all types of cancer combined was at or significantly below the expected level. Based on national cancer death rates, 699 and 420 deaths were expected among the hourly male and female workers, respectively. Based on regional cancer death rates, 713 and 449 deaths would have been expected among hourly male and female workers, respectively. Only 586 and 380 cancer deaths were observed for the men and women, respectively.

The HHRA sets forth several alleged “limitations” of Kimbrough et al. (1999) and states that the study is undergoing peer review by the Agency. Prejudging the outcome of the peer review, the HHRA then states that the Kimbrough et al. (1999) study will likely not lead EPA to reassess its views regarding the cancer potency of PCBs. Each of the “limitations” cited by EPA is based on a misunderstanding of either the extent of the workers’ exposure to PCBs or to the length and latency of that exposure. Responses to EPA’s perceived limitations of this landmark study are provided below.

“More than 75% of the workers in the study never worked with PCBs.” [HHRA, page C-3]

Both GE plants exclusively manufactured capacitors, all of which were filled with PCBs during the relevant time period. In their study, Kimbrough et al. (1999) included employees who had worked for at least three months in one or both of the GE plants between January 1946, when PCB use was first introduced, until June 1977, when the use of PCBs was discontinued.

All occupants of the plants were exposed to PCBs to varying degrees well above environmental background levels. The method by which PCBs were handled at the plants resulted in very high PCB concentrations in the workplace air. PCBs were heated to better impregnate the thin paper between the aluminum foil in the capacitors. After the capacitors were filled by immersion in open tanks containing PCBs, the uncovered canisters were put into vacuum ovens, thus increasing the rate of volatilization of the PCBs. When the ovens were opened, PCBs were released into the air, both in vapor and as aerosols, and were circulated by the air handling system. As pointed out by Kimbrough et al. (1999), the same air ventilating system served the entire building in which capacitor filling was performed, including the shipping and winding areas, the offices, and the break rooms.

In addition, all workers at the plant had dermal exposure to PCBs. Dermal exposure was obviously highest for workers employed in filling capacitors. However, due to the presence of PCBs in the workplace air as aerosols, virtually all surfaces within the plant buildings became contaminated with PCBs (Nicholson, 1987).

Furthermore, as pointed out by Kimbrough et al. (1999), workers did not always hold the same jobs. Consequently, the number of workers with the highest exposure is much larger than the number of workers involved with filling capacitors. Workers rotated through jobs with high exposure, with undefinable exposure (where the precise workplace location within the plant could not be determined and may have involved high or low exposure or both), and with low exposure. The four groups of workers in the study -- male hourly workers, female hourly workers, male salaried workers, and female salaried workers -- were always analyzed separately.

Finally, it is ludicrous for EPA to suggest that the GE plants provide a poor cohort for an epidemiological study of the health effects of PCBs. The same cohort was studied in Brown (1987), a study cited with approval by EPA in the HHRA as well as in IRIS (1999).

"The actual level of PCB exposure in the remaining workers could not be confirmed." [HHRA, page C-3]

This statement is untrue. In occupational exposure assessments, air concentrations of chemicals are frequently used to assess worker exposure and PCB air concentration data are available for the GE plants. GE and others (NIOSH) made these measurements in 1975 and 1976. This information is summarized in Kimbrough et al. (1999). These air levels were obtained at the end of the period during which capacitors containing PCBs were manufactured and after changes in the plants' ventilation systems reduced PCB air levels. No information is available on the earlier PCB air concentrations, but they were likely much higher. Based on these data, there can be no doubt that the GE workers were exposed to air concentrations of PCBs that were orders of magnitude above the level of exposure in the general population.

Nicholson (1987) investigated PCB concentrations in workplace air at several capacitor plants that used PCBs, including plants studied by Bertazzi et al. (1987) and Brown (1987). The GE plants were also included in this evaluation. Nicholson (1987) arrived at the following conclusion:

> While the industrial hygiene data that are available are extremely limited, they suggest that the time weighted average work place air exposures of electrical

> capacitor manufacturing workers ranged from concentrations in excess of 1 mg/m^3 in the high exposure areas to general plant-wide concentrations of 0.05 - 0.1 mg/m^3. There is no evidence for substantially different airborne concentrations in the different plants here reviewed.

The PCB air concentrations reported by Nicholson (1987) are consistent with the concentrations cited in Kimbrough et al. (1999), which were measured in the winding area and shipping area where workers did not have the highest exposure to PCBs.

"Less than 25% of the workers who were exposed to PCBs at the General Electric facility were employed in these jobs for less than a year. Such short-term occupational exposure is generally not comparable to the long-term exposure that may occur in the environment." [HHRA, page C-3]

As written, the first sentence is difficult to parse; perhaps the first word should be "more" rather than "less." Regardless, GE does not understand the basis for EPA's estimate of workers employed for less than one year. It is clear that even workers who were employed for relatively short periods of time carried body burdens of PCBs much higher than those carried by members of the general population.

Further, each member of the Kimbrough et al. (1999) cohort was employed at the plants for at least 90 days. The HHRA cites with approval the studies of Brown (1987), Bertazzi et al. (1987), and Sinks et al. (1992). The Brown (1987) cohort, like the Kimbrough et al. (1999) cohort, used an employment cut-off of 90 days. The Bertazzi et al. (1987) cohort included workers employed for as little as one week. The Sinks et al. (1992) cohort included workers employed for as little as one day. EPA has no basis to suggest that the employment cut-off used by Kimbrough et al. (1999) was unusual or inappropriate.

"At the end of the study period in December 1993, most of the workers were still quite young (average age 57). Because cancer deaths usually occur in older individuals, the workers in the General Electric company study may have been too young to die from cancer." [HHRA, page C-3]

First, the average follow-up time for the workers in Kimbrough et al. (1999) was 31 years, providing a very long latency period in which to determine whether there was a statistically significant increase in mortality due to cancer or other causes. Kimbrough et al. (1999) has by far the longest latency period and by far the largest number of deaths of any of the PCB epidemiological studies. It is incomprehensible that EPA would criticize Kimbrough et al. (1999) on this ground when all other studies, including studies cited with approval by EPA, had much shorter latency periods and evaluated much smaller numbers of deaths.

Second, although EPA is correct about the average age of the cohort, it neglects to point out that the cohort contains a significant number of retired workers who are over 90 years old and who are still alive and active. The National Center for Health Statistics publishes mortality rates for all causes of deaths and for specific causes by five-year intervals. Examination of these data shows that quite a number of younger people also die of cancer and other chronic diseases. The analysis set forth in Kimbrough et al. (1999) was, of course, age-adjusted.

> *"The study did not investigate vulnerable populations such as children, the elderly, or people with existing health problems."* [HHRA, page C-3]

This comment is highly misleading. Kimbrough et al. (1999) was a mortality study of capacitor workers, those people most highly exposed to PCBs, so it did not investigate children. Kimbrough et al. (1999) did include the elderly and "people with existing health problems." There were 7,075 people in the cohort, and this size population can be expected to include persons of various ages and individuals with "health problems."

2.3 EPA Should Use a Distribution of RfD Values in the Monte Carlo Assessment

EPA has traditionally evaluated non-carcinogenic risks based on a simple finding of whether an estimated dose rate was above or below the RfD. Under this approach, the measure of risk is the ratio of the predicted dose rate to the RfD. If the ratio (called the hazard quotient) is less than one, then the dose is less than the RfD and no risk is predicted.

The RfD has been defined as the "lower confidence limit of a NOAEL in sensitive humans" (Swartout et al., 1998). This definition implies that the RfD is the lower bound value of a range of doses that could be protective and that the actual level that is protective is likely to be higher than the RfD. As Swartout et al. (1998) explain, this range of RfDs is a function of the uncertainty in the actual size of the “safety” (or uncertainty) factors used in the derivation of the RfD. The magnitude of the current uncertainty factors are believed to be greater than is necessary for most chemicals (Lewis et al., 1990). Thus, most if not all RfDs are lower than is necessary to be protective of human health.

Recently, a number of authors have investigated how to characterize this uncertainty in the derivation of the RfD (Baird et al., 1997; Slob and Pieters, 1997; Swartout et al., 1998). There is general agreement that the uncertainty can be characterized by using distributions that reflect the range of values required by different compounds. The total uncertainty of the protective dose can then be calculated using probabilistic techniques. This approach has been applied to Aroclor 1254 (Widner et al., 1999). This study reported that the range of protective dose estimates had a median value of 240 ng/kg-day with a 90 percent confidence limit of 60 to 730 ng/kg-day. These findings demonstrate that the PCB RfD used in the HHRA will likely overestimate risk by factors of 3 to 36.

Techniques to incorporate the uncertainty of the RfD into the current framework have been established (Carlson-Lynch et al., 1999). Under this approach, a two-dimensional Monte Carlo model of the uncertainty and variation in the hazard quotient is developed. The uncertainty in the RfD is considered, along with the uncertainty in the estimates of exposure, to characterize the uncertainty in the estimates of specific percentiles of a cumulative distribution of the interindividual variation in the hazard quotient (Carlson-Lynch et al., 1999).

This technique has been applied to the evaluation of PCB exposures from the consumption of fish in the Clinch and Tennessee Rivers (Widner et al., 1999). In this assessment, a two-dimensional Monte Carlo model was created of the uncertainty and variability of the hazard quotient for anglers consuming such fish. The findings of the study demonstrated that the fraction of the population that was potentially at risk from PCBs was far smaller than the fraction that received a dose that was greater than the RfD. This report established an uncertainty distribution for PCBs based on the best available data. The report found that similar distributions could be established using either a default distribution proposed by Swartout et al. (1998) or evaluating available toxicity information on PCBs.

The Agency thus can incorporate the uncertainty in the protective dose directly into its Monte Carlo model instead of simply plugging in the current (and uncertain) RfD (Carlson Lynch et al., 1999; Widner et al., 1999). While the RfD may be appropriate for screening assessments, the uncertainty in the estimate of the protective dose should be used instead of the RfD when conducting a probabilistic assessment of exposure. Failure to do this will unnecessarily bias the risk estimate upward. The use of a distribution eliminates this bias and allows the decision maker to consider properly the uncertainty in the dose response portion of the non-carcinogenic risk assessment process.

2.4 EPA Improperly Excluded Uncertainty in Measures of Chemical Toxicity

EPA should have considered the variability and/or uncertainty associated with chemical toxicity in the Monte Carlo analysis. As justification for not evaluating these, the HHRA states that,

> as a matter of USEPA policy, the variability and/or uncertainty associated with chemical toxicity is not included quantitatively in a Monte Carlo risk analysis. USEPA recognizes the uncertainty inherent in the determination of cancer and non-cancer toxicity factors, and the uncertainty is factored into the determination of the toxicity factors when they are published in USEPA's Integrated Risk Information System (IRIS). . . . For the Monte Carlo analysis of cancer risk via fish ingestion, only the upper bound CSF of 2.0 (mg/kg-day)$^{-1}$ is used. Consistent with USEPA policy (EPA, 1997a), variability and uncertainty in chemical toxicity is not quantitatively evaluated in the Monte Carlo analysis. HHRA at 35.

EPA's decision not to consider uncertainty in toxicity is unreasonable and arbitrary. Current Agency policies do not prevent the consideration of this source of uncertainty. Indeed, excluding a known source of uncertainty and bias is contrary to the Agency's commitment to make decisions that are open, transparent, and based on the best science available (EPA, 1995). The risk assessment appears to refer to *Use of Probabilistic Techniques (Including Monte Carlo Analysis) in Risk Assessment* (EPA, 1997b), which focuses on issues relating to the characterization of exposure rather than dose response:

> [C]onditions for exceptions and associated guiding principles are not intended to apply to dose response evaluation to human health risk assessment until this application of probabilistic analysis has been studied further. (EPA, 1997b, page 2)

EPA (1997a) also makes it clear that the guiding principles are not intended to restrict the valid application of techniques to new and innovative areas:

> EPA recognizes that quantitative risk assessment methods in quantitative variability and uncertainty analysis are undergoing rapid development. These guiding principles are intended to serve as a minimum set of principles that are not intended to constrain or prevent the use of new or innovative improvements where scientifically defensible (Guiding Principles for Monte Carlo Analysis at 3).

There is considerable information available on the uncertainty of toxicity criteria. The Agency's own guidance for the evaluation of carcinogenic risks describes the estimate of central tendency and 95 percent upper confidence limits to carcinogenic potency. This information is used in the HHRA for the evaluation of carcinogenic risks from the consumption of fish (p. 64). While these estimates of uncertainty in the cancer slope factor only reflect the uncertainty associated with the limited number of animals included in the assays, they demonstrate that the Agency has valid technical information on the uncertainty of the cancer slope factor. As explained above, techniques have also been developed to evaluate the uncertainty and bias in the RfD.

The HHRA's failure to consider uncertainty in toxicity information is inconsistent with recommendations of EPA's Science Advisory Panel (SAP) under FIFRA. In February 1999, the SAP reviewed EPA's proposed approach for assessing non-carcinogenic risks from aggregate exposure to pesticides (EPA, 1999b). The Science Advisory Board (SAB) report for that meeting (EPA, 1999c) includes several sections calling for the use of quantitative techniques for the evaluation of uncertainty in non-carcinogenic and carcinogenic risks as a means of improving EPA decision making:

> Eventually, the majority of the Panel would like to see the whole NOAEL/uncertainty factor framework replaced by a more quantitative risk assessment approach in which all of the safety factors are replaced by distributions based on the best available data from well studied cases. The results of this would ideally be fully quantitative analyses for non-cancer effects as well as cancer risks with an understanding of both uncertainty and variability. Standards would then need to be set for safety goals. (EPA, 1999b; page 37)
>
> The dilemma above arises because the 10-fold factors are hard to interpret as adjustments for the means of distributed extrapolation factors or as allowances for the worst-case tail of these distributions. A distributional approach to noncancer risk analysis would resolve the dilemma by specifying the whole distribution of

> the factors in question. If different components of an aggregation have different uncertainties, the distributional approach easily accommodates calculation of the uncertainty of their sum, with the mean of the output distribution making the necessary extrapolation adjustments without conservatism and its spread providing a measure of the uncertainty, providing a basis for risk managers to apply allowances for uncertainty as they see fit. (EPA, 1999b; page 45)

The SAB rightly observes that the use of RfDs with fixed values of safety factors prevents decision-makers from understanding the uncertainty in these values and the conservative assumptions that already have been used to account for this uncertainty.

2.5 EPA Correctly Rejected Separate Consideration of Dioxin-like Risks of PCBs

Considerable and unnecessary uncertainty is added to the risk assessment when Toxic Equivalency Factors (TEFs) are assigned to PCB congeners to convert them to 2,3,7,8-TCDD equivalents and a CSF for 2,3,7,8-TCDD is applied. EPA acted appropriately by not using this flawed approach to estimate risk. To use TEFs, on total PCBs, one needs to assume incorrectly that: (1) the studies used to derive the toxicological, epidemiological, and analytical databases for total PCBs are less reliable and complete than those for the individual PCB congeners, which are, in reality, based on TCDD as a surrogate for PCB congeners; (2) the effects of PCBs are mediated through the Ah receptor; (3) the toxicity of individual PCBs is additive when combined in mixtures; (4) no variability occurs in sensitivities between endpoints and within broad groups of species; and (5) the dose-response curve for TCDD is parallel to that for individual PCB congeners. Exceptions to all of these assumptions have been reported in the literature (Safe, 1994; Pohjanvirta et al., 1995; Putzrath, 1997; Starr et al., 1997; WHO, 1997).

In addition, the use of congener-specific data to estimate separate risks for dioxin-like congeners and non-dioxin-like congeners, using the PCB CSF of 2 $(mg/kg\text{-}day)^{-1}$, has numerous scientific deficiencies. It results in a substantial overestimation of carcinogenic risks due to PCBs because it double-counts their carcinogenic potential. This is because the cancer slope factor for PCBs characterizes the carcinogenic potential of the entire PCB mixture, which includes both dioxin-like and non-dioxin-like congeners. Thus, if one evaluates the dioxin-like congeners using dioxin Toxic Equivalent Quotients (TEQs) and then evaluates the non-dioxin-like components using the PCB CSF, it is counting the carcinogenic potential of the dioxin-like congeners twice because their carcinogenic potential is already inherent in the CSF for PCBs.

Even if the analysis subtracts out the concentrations of the dioxin-like congeners in making the risk calculations for the remaining PCBs, the double-counting still occurs because the calculated CSF for PCBs is based on toxicological studies of Aroclor mixtures that contained both dioxin-like and non-dioxin-like congeners. Indeed, EPA has attributed much of the so-called carcinogenic potency of PCB mixtures to the dioxin-like congeners (IRIS, 1999). Thus, the CSF of 2 $(mg/kg\text{-}day)^{-1}$ incorporates of the carcinogenic activity of both types of congeners and is much too high to represent the carcinogenic potential of only the non-dioxin-like congeners. Without a CSF for non-dioxin-like PCBs, there is no defensible way to use both the TCDD CSF and the PCB CSF in the same assessment. There also is substantial uncertainty about the appropriate TCDD CSF, with estimates varying by more than an order of magnitude.

Given the current state of scientific information, any effort to use congener-specific PCB data in this human health risk assessment is unnecessary and scientifically unjustified.

3.0 EPA's Selection of Conservative Exposure Assumptions Overestimates Risks

EPA made a number of assumptions that materially overstate the likely exposure of anglers to Hudson River PCBs. Use of appropriate and more realistic exposure scenarios results in a materially decreased risk.

3.1 EPA Did Not Select the Most Appropriate Study for Estimating Rates of Fish Consumption

EPA misused the results of Connelly et al. (1992) study on which it based the fish consumption rates used in the HHRA. This study has significant limitations, causing the Agency to overestimate fish consumption rates and adding considerable uncertainty to these estimates.

The Connelly et al. (1992) survey of New York's recreational anglers was intended "to (1) assess New York licensed angler awareness and knowledge about advisories and contaminants in fish, and fishing and fish-consuming behavior, and (2) identify changes in these factors that have occurred since the explanatory information in the advisory was expanded" (Connelly et al., 1992; page viii). While the study did collect some information on the fish consumption habits of the surveyed anglers, it was not designed to provide a reliable basis for estimating the long-term fish consumption rates of the surveyed anglers and the data from the study are not adequate to do so. The key limitations of the Connelly et al. (1992) are summarized below and explained in detail in Attachment B.

- The fish consumption rates calculated by EPA from the Connelly et al. (1992) data are not supported by fish consumption rates calculated from other surveys of northeastern anglers, which show consistently lower rates of consumption (Table 1).

Table 1. Comparison of Fish Ingestion Rates from Studies of Northeastern Recreational Anglers

Consumption Rate Percentile	Connelly et al. 1992 New York Multiple Rivers[a]	Ebert et al. 1993 Maine Multiple Rivers	ChemRisk 1991 Maine Single River[b]	Connelly et al. 1996 New York All Waters[c]	Ebert et al. 1996 Connecticut Single River[d]
50th	4.0	0.99	0.49	2.2	0.17
90th	31.9	6.1	5.3	13.2	5.8
95th	63.4	12.4	10.7	17.9	12
Arith. Mean	17.3	3.7	3.0	4.9	2.6

a. EPA (1999a) analysis
b. West Branch Penobscot River
c. EPA (1997a) analysis
d. Housatonic River

- The survey response rate reported by Connelly et al. (1992) was 52.3 percent, which is on the low-end of accepted standards for mail surveys.

- EPA has not correctly weighted the non-respondents to the survey to determine their impact on the fish ingestion distribution. Correct weighting of these responses would result in substantially lower estimates of fish consumption for the total angler population.

- The Connelly et al. (1992) survey overestimates consumption rates as a result of the long-term recall bias (Westat Inc., 1989; West et al., 1989; Connelly et al., 1995).

- Connelly et al. (1992) did not request information on meal sizes of individual fish. EPA's assumptions concerning meal sizes add considerable uncertainty to the fish ingestion estimates.

- The instructions for completing the fish consumption matrix of the Connelly et al. (1992) survey instructed anglers to place a "?" in the appropriate box if they knew that they had eaten some fish but could not remember how many. A total of 179 of the individuals who completed the matrix marked a "?" on at least one occasion, and some individuals reported a "?" for all fish meals. It is not possible to reliably assign a fish consumption rate to the "?" responses, and EPA eliminated all cases where a "?" was marked. EPA's approach added considerable uncertainty to the analysis.

Out of 17,788 meals reported by the anglers who completed the consumption matrix, 5,816 (33 percent of total meals) had no source waterbody identified (GE analysis of raw data) and thus could not be apportioned by waterbody type. EPA attempted to offset this limitation by making assumptions about the relative rates of ingestion from standing vs. flowing waterbodies (see equation on page 42 of the HHRA). EPA's inability to validate these assumptions contributes substantial uncertainty to the resulting fish ingestion rates.

The fish ingestion rate distribution for the HHRA should use a survey designed to collect detailed information on long-term fish consumption habits, should target the population, region, and waterbody type being evaluated, and should minimize recall bias. Both the Connelly et al. (1996) survey of New York's Lake Ontario anglers and the Ebert et al. (1993) survey of Maine's freshwater anglers meet these criteria better than the Connelly et al. (1993) data:

- The data from both studies are regionally appropriate. Connelly et al. (1996) focused on a subset of New York anglers and Ebert et al. (1993) focused on all Maine anglers. While neither of these is the exact population targeted by the HHRA, the consumption behaviors of these two groups of anglers should not vary considerably from Hudson River anglers.

- Both the Connelly et al. (1996) and Ebert et al. (1993) surveys focus on sport-caught fish consumption by freshwater recreational anglers in the northeastern U.S. who have substantial access to high quality fisheries with similar geography and a similar fishing season.

- The demographics of surveyed Maine anglers are similar to New York anglers.

- While all three surveys collected information on long-term consumption rates, the Connelly et al. (1996) survey minimized recall bias by using food diaries, making consumption rates from this study more accurate than the Connelly et al. (1992) survey data.

- The response rates for both the Ebert et al. (1993) and Connelly et al. (1996) surveys are considerably higher than the response rate for Connelly et al. (1992) and can, therefore, be considered more representative of the targeted angler population.

- Because of the way in which the data were collected by both Connelly et al. (1996) and Ebert et al. (1993), one need not make assumptions about meal sizes in deriving consumption estimates. EPA's approach of assuming 0.5 pound for each meal recorded in the Connelly et al. (1992) survey adds considerable uncertainty to the analysis.

- The Ebert et al. (1993) fish consumption distribution is similar to the data collected in the Connelly et al. (1996) one-year diary survey of New York Lake Ontario anglers and lower than rates from Connelly et al. (1992). (Table 1); (Figure B-1).

- The similarities between the Ebert et al. (1993) and Connelly et al. (1996) data confirm that there are no substantial differences in behavior between New York and Maine anglers and that EPA's analysis of Connelly et al. (1992) overestimates consumption by this population.

- Fish consumption advisories did not substantially affect the Maine angler results. At the time that the survey was conducted, such advisories applied to only 200 miles of Maine's 37,000 miles of river and stream fisheries.

As a result, both the Connelly et al. (1996) and the Ebert et al. (1993) surveys provide a stronger basis for the consumption rate distribution than the Connelly et al. (1992) survey data. EPA (1997a) recognized the limitations of the Connelly et al. (1992) survey in its review of the fish consumption literature and consequently did not select that survey as a "Key" study to evaluate sport-caught freshwater fish consumption by recreational anglers. EPA should recalculate exposures for "Upper Hudson River" anglers using data from either the Ebert et al. (1993) or Connelly et al. (1996) studies.

3.2 EPA Failed to Consider Year-to-Year Variation In Fish Consumption

EPA implausibly assumed that an individual eats the same amount of fish every year for more than 30 years. The Agency acknowledged that this assumption was not supported by the available data:

> Actual year-to-year ingestion rates are probably correlated to a high degree, but not perfectly (100 percent). This assumption is supported by the finding that when classified as either low or high avidity (in relationship to the median fishing effort), two-thirds of Lake Ontario anglers were classified the same in 1991 and 1992 (Connelly and Brown, 1995). Assuming there is no correlation between yearly ingestion rates would effectively average high-end consumers out of the analysis, and would be clearly inappropriate. Thus, although there are no data available to quantify the correlation between yearly ingestion rates, the approach taken in the risk assessment is reasonable and protective of human health. (EPA, 1999a, page 74).

The Agency has created a false dilemma by implying that there are only two options for the evaluation of year-to-year variation in intake rates: 1) the no-change or fixed option, and 2) an option that varies the intake rates randomly.

There is a third and better option. One can use the available information on inter-year variation to model fish consumption rates. The data include Boyle et al. (1990), who found that 30 percent of anglers do not fish every year, Connelly et al. (1999), who reported that only 25 percent of surveyed anglers fished in each of the previous six years, and the data cited by the Agency (Connelly and Brown, 1995) that one-third of all anglers move from high avidity to low avidity each year. This information can be used to model year-to-year variation. For example, the model could assign a given angler a 25 percent chance of being a consistent angler and a 75 percent chance of fishing occasionally. In addition, the model could change an angler's consumption rate percentile for each year. For example, if the angler's consumption rate percentile were above 50 percent on a given year, the following year there would be a 30 percent chance that it would move to a percentile below 50 percent. This process could be repeated for each year that an angler fishes the "Upper Hudson River". In this way, the angler consumption rates would not be fixed but also would not vary in a totally random fashion.

Studies of long-term exposure rates to contaminants in fish have demonstrated that the distribution of chronic exposure rates in a population of anglers is greatly affected by inter-year variation in consumption rates (Price et al., 1996). Therefore, the Agency's failure to model inter-year variation significantly overestimates the upper percentiles of exposure and risk.

3.3 EPA Inconsistently Defined the Angler Population

The HHRA defined the exposed angler population in a number of conflicting ways. On page 5, the exposed population is defined as anglers who <u>may</u> fish, indicating that the population of concern should include anglers who potentially could consume fish from the "Upper Hudson River". Later (page 72), the population is defined as those anglers who consume a minimum of one fish meal per year in the absence of a fishing ban or health advisory.

EPA's first definition would include those anglers who might fish the "Upper Hudson River" but might do so with less regularity than one meal per year. As documented by Boyle et al. (1990), Connelly et al. (1992), Phillips et al. (1990), Ebert et al. (1993), and Connelly et al. (1999), a substantial portion of anglers do not fish every year. This fraction may be as high as seventy-five percent of all anglers (Connelly et al., 1999). Excluding those anglers who do not fish every year results in overestimates of fish consumption per capita and therefore, the distribution of doses is biased towards overestimation of risk. This is not appropriate. EPA should include all individuals who might consume fish from the "Upper Hudson River", including those who eat less than one meal per year.

3.4 EPA Incorrectly Calculated Exposure Duration

In characterizing annual mobility rates, the HHRA incorrectly asserts that the number of individuals moving out of an area in a single year is equal to the number who move out over a five-year time period divided by 5. Simple division does not determine the relationship between the probability of moving in one year and the probability of moving in five years. The reason for this is that once some fraction of a population has moved in the first year, they are not available to move in subsequent years. Because of this effect, the relationship between a five-year mobility rate and the one-year mobility rate is given by the following equation:

$$M_1 = 1- (1-M_5)^{1/5}$$

Where, M_1 is the probability of moving one year and M_5 is the probability of moving in five years.

3.5 EPA Improperly Accounted for Cooking Loss

The HHRA states that "[b]ased on the available data, it is not possible to quantify the importance of specific factors influencing the extent of PCB cooking losses." (HHRA at 49). The Agency also concludes "[i]t is not possible to develop a probability distribution representing the variability of cooking loss expected either among different consumers, or due to different preparation methods."

Percent loss of PCBs can be related to cooking methods, and the method used to prepare the fish can be linked to fish species. EPA acknowledges that "[o]verall, studies support the conclusion that some PCBs are lost during cooking….but quantitative estimates of cooking losses remain uncertain." HHRA at 48. At issue is the inconsistency in the way the authors of the available studies have reported their results. Authors have reported reductions as the amount of PCBs lost per gram of fat, per gram of fish wet weight, per gram of fish dry weight, or the total mass of PCBs lost. This inconsistency can hamper comparisons and compilations of results and increases the uncertainty associated with the determination of a single cooking loss value or a percentage loss of PCBs resulting from each of the different cooking methods. Sherer and Price

(1993) developed a methodology to convert the results of cooking loss studies to a percent loss of PCBs on a total mass basis. Conversion of the results to the same units allows one to determine an average PCB loss for different cooking methods.

In addition to quantitative estimates of PCB loss by various cooking methods, it is possible to link those cooking methods to fish species. Survey data collected by Connelly et al. (1996) for New York anglers can be used to identify the cooking methods used for each species and the relative probabilities of their usage for those species that are known to be present in the "Upper Hudson River". Cooking preference, in combination with the reduction of PCBs by cooking method, adequately characterizes PCB loss during cooking so that it is possible to develop a probability distribution representing the variability of cooking loss expected among the anglers.

3.6 EPA Improperly Relied on the Connelly et al. (1992) Data to Establish Species Preferences

The Connelly et al. (1992) survey data on species preference do not provide an appropriate basis for estimating species preferences of "Upper Hudson River" anglers. As explained above, the Connelly et al. (1992) survey was designed to measure anglers' understanding and compliance with the existing fish consumption advisories. Consequently, the species list provided in their fish consumption matrix is limited to those species and length classes of fish that correlated with concurrent advisory recommendations. As a result, the species list included many species not found in the "Upper Hudson River" and excluded species that would be expected to be caught and consumed from the Upper Hudson River. Accordingly, the data from Connelly at al. (1992) are too limited to characterize the species consumption preference for the Upper Hudson River.

Because many relevant species were omitted from the species list, a large number of responses to the survey listed meals of "Other" species. According to EPA (1999a Table 3-3), 25 percent of all fish consumed from flowing waterbodies were reported in the "Other" category. When attempting to calculate species preferences based on these data, EPA inappropriately ignored those species that were reported as "Other," instead using data from only six species (bass, walleye, bullhead, carp, eel, and perch) and placing them into three groupings with a single surrogate species to represent each group. Not only did the Agency not provide a rationale for grouping the fish in this manner, there are a number of problems associated with this approach.

First, ignoring the "Other" category places too much emphasis on only six fish species. In fact, the six species reported in the Connelly et al. (1992) data, only accounted for 38 percent of all of the fish that were consumed from flowing waterbodies statewide. Thus, while bullhead only represented fourteen percent of the fish eaten from flowing waterbodies, EPA's approach results in an assumed preference of 36 percent. EPA's approach inappropriately biases the estimates of species preference and artificially inflates actual levels of exposure to "Upper Hudson River" anglers.

Second, the species appear to have been grouped by habitat rather than by trophic level or lipid content. Bullhead, carp, and eel are all bottom feeders and have been grouped together, while bass and walleye are both surface feeders, and white perch are mid-column feeders. This grouping ignores the important species-specific variations in food sources and lipid contents, which drastically impact the concentration of PCBs in their tissues. Because of bioaccumulation potential, higher trophic level fish will be exposed to higher levels of PCBs than fish feeding at a lower trophic level. In addition, even fish that feed at the same trophic level will have substantially different PCB body burdens if their lipid contents vary. EPA fails to take these important issues into consideration in its grouping for species preference and oversimplifies and unnecessarily biases this important parameter.

Finally, EPA ignores more relevant data that provide better information in species preference (Connelly, 1996). For bullhead (including bullhead, carp, and American eel), EPA assumes a combined preference of 44 percent for this group, while the Connelly et al. (1996) data indicate that these species represented only 8.9 percent of the fish consumed from rivers and streams. EPA's estimate of angler preference for bass (including bass and walleye) (47 percent) contrasts with the information in the Connelly et al. (1996) survey, in which bass represented 58 percent of species preference.

EPA's estimate of preference for perch (white and yellow combined) (9 percent) also appears to be underestimated. Connelly et al. (1996) reported that perch accounted for 12.5 percent of the fish consumed. This underestimation in species preference is probably due to the fact that the Connelly et al. (1992) questionnaire asked only for information about white perch. Thus, any meals that were yellow perch would have been included in the "Other" category and would have been excluded from the EPA's analysis.

EPA cannot ignore angler preferences for other species of fish simply because the database upon which these preferences are based is inadequate. Instead, EPA should have selected an alternative database that provides more insight into consumption preferences. The best source of information on the species preference for in the absence of the fish consumption bans would be Connelly et al. (1996).

3.7 EPA Improperly Relied on the Output of Fate, Transport and Bioaccumulation Models That Have Not Been Peer Reviewed

EPA relied on the output of fate, transport, and bioaccumulation models that have not yet been subjected to peer review and may not be reliable. In addition, there are substantial problems with the way in which future fish concentrations have been estimated.

A critical component of the HHRA is estimating future risks to human health. To perform this task, EPA needs to incorporate valid and reliable estimates of future PCB concentrations in fish. The only reliable tools to provide such estimates are properly calibrated and validated fate, transport, and bioaccumulation models. The Agency used the output of the fate, transport, and bioaccumulation models presented in the 1999 Baseline Monitoring Report (BMR) for the HHRA. While GE concurs with this conceptual approach, the specific models used by EPA are flawed, and have not yet undergone peer review and should not be used until the flaws are corrected and peer review completed.

EPA issued the BMR on May 18, 1999 and, in public meetings, described it as a "work in progress." GE submitted extensive comment on the BMR on June 23, 1999. EPA has not responded to these comments, which are incorporated by reference into these comments.

EPA released the HHRA in August 1999. Thus, for one of the most important parameters in the HHRA – future PCB concentrations in fish – EPA is using the output of models that do not reflect changes that might result from public comments and peer review. The HHRA should incorporate data based on final and complete models, not ones that are very likely to be changed. To use models which are works in progress results in a misleading and incorrect assessment of risks to human health. Such an HHRA has little utility for a risk manager.

For example, the modeled PCB levels of fish at Stillwater presented in the BMR exceed the actual data in the 1990s, indicating that the model is not a reliable predictor of fish PCB levels and will overpredict PCB exposure. Moreover, the projected PCB concentrations in fish presented in the HHRA differ from projected concentrations presented in the BMR. Concentrations in largemouth bass from Stillwater in 1998, presented in HHRA Figure 2-5 (approximately 7 ppm wet weight), differ from those in the BMR (Figure 7-14; approximately 5 ppm wet weight). Second, the drop seen in PCB concentrations in largemouth bass from Stillwater in 1999 (BMR Figure 7-14) is not observed in the projections in the HHRA (Figure 2-5). The HHRA references the BMR as the source for the results, which is obviously wrong. The reason for this discrepancy needs to be explained.

4.0 EPA Failed to Produce a Meaningful Probabilistic Model of Potential Exposure to Anglers on the Upper Hudson River

EPA's Monte Carlo analysis of the inter-angler variation of PCB exposure is overly simplistic, poorly documented, inconsistent with EPA guidance, and inadequate in its characterization of the uncertainties in the exposure estimates. Therefore, the findings do not provide a reasonable basis for assessing the risks to anglers or for confirming the point estimates of risk. The key limitations of the probabilistic model are summarized below and explained in detail in Attachment C.

- EPA's model fails to satisfy the criteria established in its guidance for use of Monte Carlo analyses (EPA, 1997b). This guidance sets out a number of criteria for the acceptance of probabilistic analyses. The probabilistic analysis in the HHRA fails to meet many of the criteria, including deficiencies in the model design and in documentation of the assessment.

- Although the modeling approach outlined in the HHRA is generally sound, the actual model used by EPA is fundamentally different from and substantially more limited than the HHRA's general description of the model. On page 36 of the HHRA, EPA acknowledges that modeling PCB exposures to anglers must be performed as a series of separate annual exposure events. Unfortunately, EPA does not model anglers' doses as separate events but instead models them as single blocks of time that last for periods ranging from one year to longer than 30 years. This approach greatly limits the Agency's ability to model temporal changes in inputs and prevents the correct determination of chronic and lifetime doses.

- EPA failed to provide in a timely manner, an adequate description of the probabilistic model used to evaluate angler exposures, impairing the public's opportunity to analyze and comment on matters highly germane to this entire risk assessment. (As requested by GE, EPA provided additional information on September 3, 1999).

- The design of the model forces the Agency to assume that anglers consume unrealistic amounts of fish harvested from the same locations, cooked in the same fashion, and composed of the same mixture of species every year for more than 30 years. People's behavior does vary over time.

- The method used to characterize chronic non-cancer endpoints incorrectly identifies certain anglers with short-term exposures as having very high chronic doses. These anglers only fish for one or two years but are assumed to have the highest chronic doses. This assumption biases the estimates of the hazard quotient for the higher percentiles of the distribution of chronic risks.

- EPA's failure to separate uncertainty and variability weakens its analysis of risk.

- To address uncertainty in model inputs, the Agency performed a sensitivity analysis but presented the results as if it had performed a more sophisticated discrete probability analysis

(DPA) (Morgan and Henrion, 1990). Although DPA can be used to evaluate the range and distribution of uncertainty, a sensitivity analysis cannot. Sensitivity analysis can only identify the most significant sources of uncertainty but cannot quantify the significance of that uncertainty. As a result, the Agency's uncertainty assessment does not support the HHRA's conclusions that 1) the findings of significant cancer and noncancer risks occur no matter what assumptions are made for model inputs, and 2) the findings of the probabilistic assessment support the point estimates.

- The Agency made a number of inappropriate choices in the sensitivity analysis. EPA includes sources of uncertainty (e.g., location) that are not appropriate or are of minor importance (e.g., cooking loss and mobility rates). EPA excludes factors that have a major impact on the risk estimates, including uncertainty in the cancer slope factor and the reference dose, angler recall bias, inter-year variation in fish consumption rates, and use of consumption data from multiple waterbodies. Finally, the Agency considers a fish consumption study (West et al., 1989a,b) that is irrelevant to the evaluation of risks at this site. The Agency provides no information on how it selected the sources of uncertainty considered in the sensitivity assessment. As a result, the sensitivity analysis has little or no meaning.

- EPA asserts that the data were insufficient to characterize uncertainty and variability jointly using a two-dimensional Monte Carlo analysis but never justifies this decision. The Agency states that it views uncertainty in distributions in terms of parametric uncertainty but does not attempt to actually define the uncertainty in the parameters of the distributions of variability. In addition, the Agency does not identify what factors or data gaps prevent it from defining the uncertainty in parameters.

5.0 Conclusions

The purpose of the HHRA is to inform the risk manager of what risks are present and to understand the uncertainty in the risk calculations. On this basis, the risk manager can evaluate potential remedial options in terms of risk reduction.

In some regards the Agency has performed well, and in others it has not. The HHRA concludes that the only material human health risk is the potential consumption of fish from the Upper Hudson River. Of course, fishing has been restricted for over 20 years in the Upper Hudson River; catch-and-release fishing does not present such a risk. Drinking the water, contact with PCBs in the sediment, or breathing PCBs in the air during recreational activities, such as wading, boating or swimming, do not present an unacceptable health risk.

EPA, however, has poorly characterized and communicated the potential risks from fish consumption. The major problems include:

- EPA did a poor job of communicating the fact that the risks from fish consumption calculated by EPA are hypothetical. This leads to mischaracterization of the risk to citizens using the Upper Hudson River.

- EPA's critique of Kimbrough et al. (1999) is superficial and the claim of limitations is unfounded. EPA needs to complete an objective and scientific evaluation of this groundbreaking study.

- EPA grossly overestimates the toxicity of PCBs and as a result overstates potential risks. Based on a weight-of-evidence appraisal, there is no credible information that PCBs cause cancer in humans. Additionally, there is little, if any, evidence that PCBs cause adverse effects in humans at environmental exposure levels.

- The exposure assumptions made to estimate risks to the hypothetical angler materially overstate potential exposures. Key problems include:

    - Use of the results of a flawed PCB food chain model for estimating fish PCB levels.

    - Implausibly high estimates of fish consumption rates and the duration of high fish consumption.

- Miscalculation of angler mobility, improperly defining the angler population, and not properly accounting for cooking losses.

As a result, it is apparent that EPA needs to redo the calculations of potential risk to the hypothetical angler in the Upper Hudson River to correct these errors and to remove the unnecessary uncertainties in the calculations that result in gross overestimates of risk. The data and methods to do this are available, and making such changes is consistent with EPA policy. EPA policy on this point was articulated by Administrator Browner in her cover letter on EPA's Guidance for Risk Characterization: "while I believe that the American public expects us to err on the side of protection in the face of scientific uncertainty, I do not want our assessments to be unrealistically conservative. We cannot lead the fight for environmental protection into the next century unless we use common sense in all we do."

After the modifications are made, EPA will need to reissue this report not only to communicate more accurately the risks to the citizens who use the Upper Hudson River for recreation but also to provide more realistic information to the risk manager who needs to evaluate the need for additional remedial actions.

6.0 References

Arnold, D.L., F. Bryce, R. Stapley, P.F. McGuire, D. Burns, J.R. Tanner, and K. Karpinski. 1993a. *Toxicological Consequences of Aroclor 1254 Ingestion by Female Rhesus (Macaca mulatta) Monkeys, Part 1A: Prebreeding Phase - Clinical Health Findings.* Health and Welfare Canada, Health Protection Branch, Toxicology Research Division.

Arnold, D.L., F. Bryce, K. Karpinski, J. Mes, S. Fernie, H. Tryphonas, J. Truelove, P.F. McGuire, D. Burns, J.R. Tanner, R. Stapley, Z.Z. Zawidzka, and D. Basford. 1993b. *Toxicological Consequences of Aroclor 1254 Ingestion by Female Rhesus (Macaca mulatta) Monkeys, Part 1A: Prebreeding Phase - Clinical Health Findings.* Health and Welfare Canada, Health Protection Branch, Toxicology Research Division.

ATSDR. 1998. *Toxicological Profile for PCBs (Update).* Agency for Toxic Substances and Disease Registry, Atlanta, GA and U.S. Department of Health and Human Services, Public Health Service. December.

Baird, S.J.S., J. T. Cohen, J. D. Graham, A. I. Shlyakhter and J. S. Evans, 1996. Noncancer Risk Assessment: Probabilistic Characterization of Population Threshold Doses, HERA. 2(1), 78-99.

Barclay, B. 1993. *Hudson River Angler Survey: A Report on the Adherence to Fish Consumption Health Advisories Among Hudson River Anglers.* Hudson River Sloop Clearwater, Inc., Poughkeepsie, New York. March.

Bertazzi, P.A., L. Riboldi, A. Pesatori, L. Radice, and C. Zocchetti. 1987. Cancer mortality of capacitor manufacturing workers. *Am. J. Ind. Med.* 11:165-176.

Boyle, K.J., R.K. Roper, S.D. Reiling. 1990. Highlights from the 1988 Survey of Open Water Fishing in Maine. Department of Agricultural and Resource Economics. Staff Paper Series in Resource Economics. ARE 416. July.

Brown, D.P. 1987. Mortality of workers exposed to polychlorinated biphenyls: An update. *Arch. Environ. Health.* 42(6):333-339.

Carlson-Lynch, H., P.S. Price, J.C. Swartout, M.L. Dourson, and R.E. Keenan. 1999. Application of quantitative information on the uncertainty in the RfD to non-carcinogenic risk assessments. HERA 5(3) p. 527-547.

ChemRisk. 1991. *Penobscot River Creel Survey*. McLaren/Hart-ChemRisk, Portland, ME. November.

Coddington, K. 1986. A review of arsenicals in biology. *Toxicol. And Environ. Chem.* 11:281-290.

Connelly, N.A., T.L. Brown, and B.A. Knuth. 1990. *New York Statewide Angler Survey 1988.* New York State Department of Environmental Conservation, Division of Fish and Wildlife, Albany, NY. April.

Connelly, N.A., B.A. Knuth, and C.A. Bisogni. 1992. *Effects of the Health Advisory Changes on Fishing Habits and Fish Consumption in New York Sport Fisheries.* Human Dimension Research Unit, Department of Natural Resources, New York State College of Agriculture and Life Sciences, Fernow Hall, Cornell University, Ithaca, NY. Report for the New York Sea Grant Institute Project No. R/FHD-2-PD. September.

Connelly, N.A., and T.L. Brown. 1995. Use of Angler Diaries to Examine Biases Associated with 12-Month Recall on Mail Questionnaires. *Transactions of the American Fisheries Society* 124:413-422.

Connelly, N.A., B.A. Knuth, and T.L Brown. 1996. Sportfish Consumption Patterns of Lake Ontario Anglers and the Relationship to Health Advisories. *North American Journal of Fisheries Management* 16:90-101.

Connelly, N.A., D. Kuehn, T.L. Brown and B.A. Knuth. 1999. *New York's Great Lakes Angler Markets.* Coastal Recreation FACT SHEET, Sea Grant, New York. April.

Cook, R.R. 1982. The role of epidemiology in risk assessment. *Drug. Metab. Review* 12(5):913-923.

Dietrich, D.R. and J.A. Swenberg. 1991. The presence of 2µ-globulin is necessary for d-limonene promotion of male rat kidney tumors. *Cancer Res.* 51:3512-3521.

Dinman, B.D. and N.B. Sussman. 1983. Uncertainty, risk, and the role of epidemiology in public policy development. *Reg. Toxicol. Pharmacol.* 7:206-216.

Ebert, E.S., N.W. Harrington, K.J. Boyle, J.W. Knight, and R.E. Keenan. 1993. Estimating consumption of freshwater fish among Maine anglers. *N. Am. J. Fish. Mgt.* 13:737-745.

Ebert, E.S., S.H. Su, T.J. Barry, M.N. Gray, and N.W. Harrington. 1996. Estimated rates of fish consumption by anglers participating in the Connecticut Housatonic River Creel Survey. *North American Journal of Fisheries Management* 16:81-89.

EPA. 1985. Chemical Carcinogens: A Review of the Science and its Associated Principles. (February). U.S. Environmental Protection Agency, Office of Science and Technology Policy, Washington, DC. 50 *Fed. Reg*. 10380 (March 14).

EPA. 1992a. *Final Guidelines for Exposure Assessment*. U.S. Environmental Protection Agency, Washington, D.C. 57 FR 104. (May 29).

EPA. 1992b. *Dermal Exposure Assessment: Principles and Application*. U.S. Environmental Protection Agency, Office of Research and Development. Washington, DC EPA/600/8-91/011B. January.

EPA. 1993. *Use of IRIS Values in Superfund Risk Assessment*. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response. Washington, DC OSWER Directive #9285.7-16.

EPA. 1995. *Guidance for Risk Characterization.* U.S. Environmental Protection Agency, Science Policy Council, Washington, DC. February.

EPA, 1996. *Proposed Guidelines for Carcinogen Risk Assessment*. U.S. Environmental Protection Agency, Office of Research and Development. EPA/600/P-96/003C. April.

EPA. 1997a. *Exposure Factors Handbook*. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. EPA/600/P-95/002Fa. Washington, D.C.

EPA. 1997b. "Policy for Use of Probabilistic Analysis in Risk Assessment at the U.S. Environmental Protection Agency." Office of Research and Development, Washington, DC, USEPA/630/R-97/001.

EPA. 1999a. *Phase 2 Report – Review Copy, Further Site Characterization and Analysis. Volume 2F–Human Health Risk Assessment, Hudson River PCBs Reassessment RI/FS.* Prepared by TAMS Consultants, Inc. Gradient Corporation for the U.S. Environmental Protection Agency Region II and U.S. Army Corps of Engineers. August.

EPA. 1999b. Guidance for Performing Aggregate Exposure and Risk Assessments Office of Pesticide Programs -DRAFT-February 1, 1999

EPA, 1999c. Report FIFRA Scientific Advisory Panel Meeting February 23-24, 1999, held at the Holiday Inn, Arlington, Virginia, Sets of Scientific Issues Being Considered by the Environmental Protection Agency Regarding: Session I - Sediment Toxicity and Fate of Synthetic Pyrethroids, Session II- Time Sensitive Reversibility of Aldicarb Induced Cholinesterase Inhibition as a Factor in Acute Dietary Risk Assessment, Session III-Consultation on Development of Draft Aggregate Exposure Assessment Guidance Document for Combining Exposure from Multiple Sources and Routes. SAP Report No. 99-02 March 25, 1999.

Gold, L.S., B.R. Stern, T.H. Slone et al. 1997. Pesticide residues in food: investigation of disparities in cancer risk estimates. *Cancer Let.* 117:195-207.

Gold, L.S., T.H. Slone, and B.N. Ames. 1998. What do animal cancer tests tell us about human cancer risk? Overview of analyses of the carcinogenic potency database. *Drug Metab. Rev.* 30:359-404.

Goodman, G. and R. Wilson. 1991. Predicting the carcinogenicity of chemicals in humans from rodent bioassay data. *Environ. Health Perspect.* 94:195-218.

Gustavsson, P., C. Hogstedt, C. Rappi. 1986. Short term mortality and cancer incidence in capacitor manufacturing workers exposed to polychlorinated biphenyls (PCBs). *Am. J. Ind. Med* 10:341-344.

Gustavsson, P., and C. Hogstedt. 1997. A cohort study of Swedish capacitor manufacturing workers exposed to polychlorinated biphenyls (PCBs). *Am J. Ind. Med* 32:234-239.

Hard, G.C. and J. Whysner. 1994. Risk assessment of d-limonene: An example of male rat-specific renal tumorigens. *Crit. Rev. Toxicol.* 24:231-254.

Hay, A. and J. Tarrel. 1997. Mortality of power workers exposed to phenoxy herbicides and polychlorinated biphenyls in waste transformer oil. *Annals. N.Y. Acad. Sci.* 837:138-156.

Hernberg, S. 1992. *Introduction to Occupational Epidemiology*. Lewis Publishers, Chelsea, Michigan.

Hill, A.B. 1965. The environment and disease: association or causation? Proceedings of the Royal Society of Medicine, Section of Occupational Medicine.

Houk, V.N. 1990. Testimony of Vernon N. Houk, M.D. before the Subcommittee on Human Resources and Intergovernmental Relations Committee on Government Operations.

International Agency for Research on Cancer (IARC). 1987. Monographs on the evaluation of the carcinogenic risk of chemicals to humans. Supplement 7.

IRIS. 1999. *Chemical Search for PCBs.* Integrated Risk Information Service, U.S. Environmental Protection Agency, Cincinnati, OH and National Research Library of Medicine, Bethesda, MD.

Kimbrough, R.D. 1990. *The Human Health Effects of Polychlorinated Biphenyls.* U.S. Environmental Protection Agency, Washington, D.C.

Kimbrough, R.D., M.L. Doemland, and M.E. LeVois. 1999. Mortality in male and female capacitor workers exposed to polychlorinated biphenyls. *Journal of Occupational and Environmental Medicine* 41(3):161-171.

Layard, M.W. and A. Silvers. 1989. Epidemiology in environmental risk assessment, p. 157-173, In, D.J. Paustenbach (ed.), *The Risk Assessment of Environmental and Human Health Hazards.* John Wiley & Sons, Inc., New York.

Lewis, A.C., J.R. Lynch, and A.I. Nikiforov. 1990. A new approach to deriving community exposure guidelines from” no-observed-adverse-effect levels”. *Regulatory Toxicology and Pharmacology*11, 314-330.

Mausner, J.S. and S. Kramer. 1985. *Epidemiology - An Introductory Text*. Philadelphia: W. B. Saunders Co.

Mayes, B. A., E. E. McConnell, B. H. Neal, M. J. Brunner, S. B. Hamilton, T. M. Sullivan, A. C. Peters, M. J. Ryan, J. D. Toft, A. W. Singer, J. F. Brown, Jr., R. G. Menton, and J. A. Moore. 1998. "Comparative carcinogenicity in Sprague-Dawley rats of the polychlorinated biphenyl mixtures Aroclors 1016, 1242, 1254, and 1260," *Toxicol. Sciences* 41(1), 62-76.

Monson, R. 1988. *Occupational Epidemiology*. CRC Press, Boca Raton, Florida.

Moore, J.A., J.F. Hardisty, D.A. Banas et al. 1994. A comparison of liver tumor diagnoses from seven PCB studies in rats. *Reg. Toxicol. Pharmacol.* 20:362-370.

Morgan, M.G., M. Henrion. 1990. *Uncertainty: A Guide to Dealing with Uncertainty in Quantitative Risk and Policy Analysis,* Cambridge University Press, New York.

Nicholson, J.W. 1987. Report of the Special Panel on Occupational PCB Exposure and Various Cancers: Human Health Effects and Carcinogenic Risk Potential of PCB. Mount Sinai School of Medicine. (August). Reprinted in the Ontario Gazette in the Matter of Section 86p of the Workers Compensation Act. December.

NYSDOH. 1999. Health Consultation: 1996 survey of Hudson River anglers, Hudson Falls to Tappan Zee Bridge at Tarrytown, New York. New York State Department of Health. February.

Peterson, R.E., H.M. Theobald, and G.L. Kimmel. 1993. Developmental and reproductive toxicity of dioxins and related compounds: Cross-species comparisons. *Crit. Rev. Toxicol.* 23:283-335.

Phillips, M.L., K.J. Boyle, S.D. Reiling. 1990. Highlights from the Survey of Anglers Holding a 1988 Maine Fishing License. Department of Agricultural and Resource Economics. Staff Paper Series in Resource Economics. ARE 415. June.

Pitot, H.C. and Y.P Dragan. 1987. Facts and theories concerning the mechanisms of carcinogenesis. *FASEB Journal* 5:2280-2286.

Pohjanvirta, R., M. Unkila, J. Linden, J.T. Tuomisto, and J. Tuomisto. 1995. Toxic equivalency factors do not predict the acute toxicities of dioxins in rats. *European J. Pharmacol., Environ. Toxicol. Pharmacol*. 293:341–353.

Price, P.S., S.H. Su, J.R. Harrington, and R.E. Keenan. 1996. Uncertainty and variation in indirect exposure assessments: An analysis of exposure to tetrachlorodibenzo-*p*-dioxin from a beef consumption pathway. *Risk Analysis* 16(2):263-277.

Putzrath, R.M. 1997. Estimating relative potency for receptor-mediated toxicity: Reevaluating the toxicity equivalence factor (TEF) model. *Reg. Toxicol. Pharmacol*. 25:68–78.

Rothman, K.J. 1988. *Causal Inference*. Epidemiology Resources, Inc. Chestnut Hill, Massachusetts.

Rylander, L. and L. Hagman. 1995. Mortality and cancer incidence among women with a high consumption of fatty fish contaminated with persistent organochlorine compounds. *Scand. Jour. Work* 21:419-426.

Safe, S.H. 1994. Polychlorinated biphenyls (PCBs): Environmental impact, biochemical and toxic responses, and implications for risk assessment. *Critical Reviews in Toxicology* 24(2):87-149.

Sherer, R.A. and P.S. Price. 1993. The effect of cooking processes on PCB levels in edible fish tissue. *Quality Assurance Good Practices, Regulation, and Law* 2(4):396-407. December.

Silbergeld, E.K., J. Schwartz, and K. Mahaffey. 1988. Lead and osteoporosis: Mobilization of lead from bone in postmenopausal women. *Environ. Res*. 47:79-94.

Sinks, T., G. Steele, A.B. Smith, K. Watkins and R.A. Shults. 1992. Mortality among workers exposed to polychlorinated biphenyls. *Amer. J. Epidem*. 136(6):389-398.

Slob, W. and M.N. Pieters. 1997. A probabilistic approach for deriving acceptable human intake limits and human health risks from toxicological studies: general framework. National Institute of Public Health and The Environment. Report 620110- 005.

Starr, T.B., T.R. Zacharewski, T.R. Sutter, S.H. Safe, W.F. Greenlee, and R.B. Connolly. 1997. Concerns with the use of a toxicity equivalence factor (TEF) approach for risk assessment of “dioxin-like” compounds. *Organohal. Comp*. 34:91–94.

Swartout, J.C., P.S. Price, M.L. Dourson, H. Carlson-Lynch, and R.E. Keenan. 1998. A probabilistic framework for the reference dose, *Risk Anal*. (18)3, 271-282

Taylor, P.R. 1988. *The Health Effects of Polychlorinated Biphenyls*. Harvard School of Public Health, Boston, MA.

TERRA, Inc. 1993. Comments on the Water Quality Guidance for the Great Lakes System.

Tryphonas, H., S. Hayward, L. O'Grady, J.C.K. Loo, D.L. Arnold, F. Bryce and Z.Z. Zawidzka. 1989. Immunotoxicity studies of PCB (Aroclor 1254) in the adult rhesus *(Macaca mulatta)* monkey - Preliminary report. *Int. J. Immunoph.* 11(2):199-206.

Tryphonas, H., M.I. Luster, G. Schiffman, L.L. Dawson, M. Hodgen, D. Germolec, S. Hayward, F. Bryce, J.C.K. Loo, F. Mandy and D.L. Arnold. 1991a. Effects of chronic exposure of PCB (Aroclor 1254) on specific and nonspecific immune parameters in the rhesus *(Macaca mulatta)* monkey. *Fund. Appl. Toxicol.* 16:773-786.

Tryphonas, H., M.I. Luster, K.L. White, P.H. Naylor, M.R. Erdos, G.R. Burleson, D. Germolec, M. Hodgen, S. Hayward, and D.L. Arnold. 1991b. Effects of PCB (Aroclor® 1254) on non-specific immune parameters in rhesus (*Macaca mulatta*) monkeys. *Int. J. Immunoph.* 13(6):639-648.

Unger, M. and J. Olsen. 1980. Organochlorine compounds in the adipose tissue of deceased people with and without cancer. *Environ. Res* 23:257-263.

Wasserman, M., D.P. Nogueira, L. Tomatis, et al. 1976. Organochlorine compounds in neoplastic and adjacent apparently normal breast tissue. *Bull. Environ. Contam. Toxicol.* 15:478-484.

West, P., J.M. Fly, R. Marans and F. Larkin. 1989a. Michigan sport anglers fish consumption survey. A report to the Michigan Toxic Substance Control Commission. Natural Resource Sociology Research Lab Technical Report #1. May.

West, P., J.M. Fly, R. Marans, and F. Larkin. 1989b. Michigan sport anglers fish consumption survey. Supplement I – Non-response bias and consumption suppression effect adjustments. A Report to the Michigan Toxic Substance Control Commission. Natural Resource Sociology Research Lab Technical Report #2. September.

Westat, Inc. 1989. *Investigation of possible recall/preference period bias in national surveys of fishing, hunting and wildlife-associated recreation*. Final Report. Contract No. 14-16-009-87-008. Prepared for the U.S. Department of the Interior, Fish and Wildlife service, Washington, D.C. by Westat, Inc., Rockville, MD. December.

Wester, R.C., H.I. Maibach, L. Sedik, J. Melendres, and M. Wade. 1993. Percutaneous absorption of PCBs from soil: *In vivo* Rhesus Monkey, *in vitro* human skin, and binding to powdered human stratum corneum. *J. of Toxicology and Environ. Health* 39:375-382.

WHO. 1997. Draft Report on the meeting on the derivation of toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs, and other dioxin-like compounds for humans and wildlife. World Health Organization. June 15-18. Stockholm. 55 p.

Widner, T. 1999. Oak Ridge Dose Reconstruction Project Summary Report. September.

ATTACHMENT B
Selection of an Appropriate Fish Consumption Rate Distribution for Use in Evaluating Risks in the Upper Hudson River.

After considering the available data on fish consumption by recreational anglers in the northeastern U.S., EPA selected the Connelly et al. (1992) study as the basis for the fish ingestion distribution. The Connelly et al. (1992) study's objectives were "to (1) assess New York licensed angler awareness and knowledge about advisories and contaminants in fish, and fishing and fish-consuming behavior, and (2) identify changes in these factors that have occurred since the explanatory information the advisory was expanded." (Connelly et al., 1992; page viii) However, the study has significant limitations and should not be used to estimate the long-term rates at which anglers eat the fish they catch. The data do not allow estimates of fish consumption to be derived unless one makes numerous assumptions, resulting in substantial uncertainties. The derived fish ingestion distributions used in the HHRA. The biases associated with the data and EPA's analysis of them indicate that consumption estimates are overestimated using the approach outlined by EPA. A comparison of this fish ingestion distribution with other distributions of fish consumption by northeastern anglers demonstrates this overestimation.

To select Connelly et al. (1992), EPA claimed that other studies of fish consumption by recreational anglers were less appropriate. Two of the studies, Ebert et al. (1993) and Connelly et al. (1996), provide a more appropriate, less biased, and less uncertain basis for the fish ingestion distribution in the HHRA. The basis for this conclusion is discussed below.

Limitations of the Connelly et al. (1992) Dataset

Survey Design

The Connelly et al. (1992) survey was not designed or intended to collect fish consumption information but rather to determine anglers' levels of understanding and compliance with fish advisories. Connelly et al. (1992) has several limitations for estimating fish consumption rates, including improper survey design, inadequate sample size, poor response rate and high recall bias. EPA (1997) recognized these significant limitations and consequently did not select the survey as a "Key" study to be considered in evaluating sport-caught freshwater fish consumption by recreational anglers.

First, Connelly et al. (1992) did not collect information on the sizes of the fish meals consumed. In the HHRA, the Agency assumed that all meals were 0.5 pound in size (227 g) because this is the most commonly reported meal size. This assumption is unfounded. Meal size is frequently reported by anglers who are asked, and meal sizes vary considerably among anglers and are often dependent upon the species of fish consumed. For example, the Connelly et al. (1996) diary study of Lake Ontario anglers demonstrated that meal sizes varied considerably by species (GE analysis of raw data). While 65 percent of rock bass meals consumed by those anglers were ½ pound in size, 60 percent of calico bass meals were less than 0.5 pound (assumed by Connelly et al. to be 5 ounce portions). Over all sport-caught fish meal sizes reported in the Connelly et al. (1996) diary study, only 55 percent of them were 0.5 pound in size. Thus, by assuming a single portion size of 8 ounces, the Agency may have substantially over- or underestimated intakes by individual anglers and did not consider the variability associated with this parameter.

Second, the goal of the fish consumption portion of the Connelly et al. (1992) survey was to determine whether anglers were eating types, sizes, or amounts of fish that were specifically limited by applicable advisories. Consequently, the species list in the survey was focused on those species and sizes that were listed in the advisory. It excluded many of the species that are known to be present in the Upper Hudson River, and included many species and sizes that would not be found in the Upper Hudson River. Out of 17 species provided in the survey matrix, only seven included species of fish likely to be found in the Upper Hudson River (EPA used only six of the species in its analysis). There was no provision for many of the pan fish species that are commonly caught and consumed by recreational anglers. The only way in which these other types of fish could be captured in the survey was through inclusion of an "Other" category. The omission of commonly consumed species other than the species listed may have impacted the ability of anglers to recall their meals of those other species. Thus, this aspect of the survey contributes additional uncertainty to EPA's fish ingestion estimates.

Third, instructions for completing the fish consumption matrix of the Connelly et al. (1992) survey, instructed anglers to place a "?" in the appropriate box if they knew that they had eaten some fish but could not remember how many. 179 respondents completed the matrix and indicated a "?" on at least one occasion, and some individuals reported a "?" for all fish meals. Because it was not possible to assign a value to the "?" responses, EPA eliminated all cases where a "?" was indicated. The level of uncertainty associated with these fish consumption rates cannot be quantified.

Finally, EPA's method for segregating the Connelly et al. (1992) data by waterbody type was problematic. A large number of anglers did not identify all of the waterbodies from which they obtained the fish they ate. Out of 17,788 meals reported by the anglers who completed the consumption matrix, 5,816 of the reported meals (33 percent) had no source waterbody identified (GE analyses of raw data). As a result, one cannot determine whether those meals were obtained from flowing or standing waterbodies.

EPA attempted to offset this problem by making assumptions about the relative rates of ingestion from standing vs. flowing waterbodies (see equation on page 42 of the HHRA). The validity of this assumption cannot be demonstrated and contributes substantial uncertainty to the resulting fish ingestion rates. As shown in Table B-1, the degree of uncertainty associated with this extrapolation can vary considerably depending upon the assumptions used in making it, particularly at the upper end of the distribution. Depending upon the assumption used, the 95th percentile can vary by more than a factor of two.

Table B-1. Comparison of Connelly et al. (1992) Fish Consumption Rates (g/day) When Differing Assumptions Are Made About the Sources of Fish Meals with No Identified Waterbody

Percentile of Consumption	Flowing Waters; Assuming All Uncoded Waterbodies are Non-Flowing[a]	Flowing Waters; Scaled According to EPA for Flowing vs. Non-Flowing Waterbodies[b]	Flowing Waters; Assuming All Uncoded Waterbodies are Flowing[c]
25th	1.2	1.9	1.9
50th	3.1	4.5	4.4
75th	9.2	11.8	11.2
90th	23.5	33.0	34.9
95th	37.3	77.1	70.8
Arithmetic Mean	11.3	19.3	17.5

a. Assuming that all meals from unidentified waterbodies were obtained from non-flowing waters.
b. Meals from unidentified waterbodies apportioned according to the equation provided on page 42 (EPA, 1999).
c. Assuming that all meals from unidentified waterbodies were obtained from flowing waterbodies.

Response Rate

The response rate reported by Connelly et al. (1992) was 52.3 percent, which is on the low-end of standards acceptable for mail surveys. Brown et al. (1989) reported a range of response rates from 41.7 percent to 89.8 percent for 38 recreational surveys conducted by their research unit at Cornell University, with a mean response rate overall of 71.8 percent.

A lower response rate is likely to bias fish consumption estimates toward higher level consumers, leading to an overestimate of fish consumption rates. Individuals who do not respond to surveys of this type are likely to consume considerably less fish than individuals who do respond (Connelly et al. 1992; West et al., 1989a,b).

While EPA attempted to correct for this non-response bias by incorporating the data from the follow-up interviews with non-respondents, this correction was not made correctly. According to the Agency, there were 919 non-respondents to the survey, of which 100 individuals were surveyed by telephone. Of these 100 individuals, 55 (55 percent) reported that they consumed at least one fish meal during the survey period. In attempting to correct for recall bias, the Agency simply added the 55 consumers from the follow-up survey to the 226 anglers who consumed fish from flowing waters and then recalculated the consumption rate distribution for the resulting 281 individuals.

This approach does not give adequate weight to the remainder of non-respondents. If it is assumed that the subsample of the 919 non-respondents to the survey is representative of the entire non-respondent population, this means that 55 percent of all non-respondents, or 505 individuals, were consumers of fish. According to the data provided by respondents to the survey, 37.6 percent of the respondents who ate fish consumed fish from flowing waterbodies. If this same fraction is applied to the 505 non-respondents who consumed fish, it can be assumed that 190 non-respondents consumed fish from flowing waterbodies during the survey period. These individuals should have been included in the correction for non-response bias to provide a total sample of 416 anglers (226 respondents plus 190 non-respondents). Inclusion of these additional, non-responding consumers would have resulted in substantially lower estimates of fish consumption for the total angler population.

GE has not been able to duplicate EPA's recalculation of consumption rates for respondents and non-respondents combined because adequate data have not been provided in the HHRA.

Consistency Among Studies of Similar Populations

The fish consumption rates calculated by EPA from Connelly et al. (1992) are not supported by fish consumption rates calculated from other surveys of northeastern anglers, which consistently show lower rates of consumption. (Table B-2).

Table B-2. Comparison of Fish Ingestion Rates from Studies of Northeastern Recreational Anglers

Consumption Rate Percentile	Connelly et al. 1992 New York Multiple Rivers[a]	Ebert et al. 1993 Maine Multiple Rivers	ChemRisk 1991 Maine Single River[b]	Connelly et al. 1996 New York All Waters[c]	Ebert et al. 1996 Connecticut Single River[d]
50th	4.0	0.99	0.49	2.2	0.17
90th	31.9	6.1	5.3	13.2	5.8
95th	63.4	12.4	10.7	17.9	12
Arith. Mean	17.3	3.7	3.0	4.9	2.6

a. EPA (1999) analysis
b. West Branch Penobscot River
c. EPA (1997) analysis
d. Housatonic River

As shown in Table B-2, the Connelly et al. (1992) data, as interpreted by EPA, result in fish consumption rates that are substantially higher than consumption rates reported in other studies of northeastern anglers. In fact, EPA's analysis is inconsistent with the limited findings on fish consumption reported by Connelly et al. (1992) report of their survey. In that report, Connelly et al. (1992) stated that the average number of meals consumed by responding anglers was 11 meals per year. If the meal size employed by EPA, 0.5 pound or 227 g, is applied to this consumption rate, the result is a mean estimate of consumption of 6.8 g/day instead of the 17.3 g/day calculated by EPA. This is more than 2.5 times higher than the rate reported by Connelly et al. (1992) in the analysis of their own data.

Selection of the Connelly et al. (1992) Study Instead of the Ebert et al. (1993) Study

EPA provided three reasons to justify its selection of the Connelly et al. (1992) data instead of the Ebert et al. (1993) data for the HHRA. First, the Agency stated that the climate and characteristics of other New York waterbodies reported in Connelly et al. (1992) were likely to be more similar to the Upper Hudson River than Maine waterbodies. Second, EPA stated that it was not possible to evaluate the Maine dataset for more "Hudson-like" rivers and streams. Third, EPA faulted the Ebert et al. (1993) study because there was no correction for non-response bias in the survey design. These objections are unfounded. The Ebert et al. (1993) data are more appropriate to use to determine for the HHRA fish ingestion distribution.

Both New York and Maine are northeastern states with similar climates, lengths of fishing seasons, and angler demographics (Table B-3).

Table B-3. Comparison of Demographics of Anglers surveyed by Ebert et al. (1993) and Connelly et al. (1992)

Socioeconomic Parameter		Ebert et al., 1993 Maine Anglers	Connelly et al.,1992 New York Anglers
Gender	Male	85%	86%
	Female	15%	14%
Average age		44	42
Race	White	88%	93%
	Hispanic	0.19%	0.77%
	Native American	9.2%	0.48%
	Asian/Pacific Islander	0.12%	0.38%
	African American	0.062%	1.6%
	Other	0.19%	0.58%
	Missing	2.2%	3.2%
Average Education		High School Graduate	Some College
Average Income		$31,125	$43,000

Both states have a variety of waterbodies, ranging from large warmwater lakes to small, fast-moving coldwater streams. In addition, both states have ready access to numerous, high quality freshwater fisheries. There is no demographic or geographic reason to believe that fishing pressure or consumption habits would vary substantially between the two states.

The Maine data were collected by waterbody type so that it is possible to differentiate between fish meals obtained from standing waters and those obtained from flowing waters. This is the same approach that EPA used with the HHRA from the Connelly et al. (1992) data. The fish ingestion distribution used by EPA was based on all meals consumed from flowing waters and was not limited to "Hudson-like" waterbodies. EPA does not provide a clear description of what it believes constitutes a "Hudson-like" water body. Thus, EPA's objection to the use of the Maine survey data is equally applicable to its use of the Connelly et al. (1992) data.

GE conducted an analysis of consumption from "Hudson-like" waters reported in the Connelly et al. (1992) data. To do this, fishing data from New York State were evaluated, and regional fishery personnel were contacted and asked to indicate which of the flowing waterbodies recorded in the survey could be considered similar to the Upper Hudson River. A total of 25 waterbodies were identified and are listed in Table B-4.

Table B-4. New York State Warmwater Rivers and Streams Similar to the Upper Hudson River

Name	Counties
Allegheny River	Cattaraugus
Batten Kill River	Washington
Black River	Lewis
Butternut Creek	Otsego, Onondaga
Chemung River	Chemung, Steuben, Broome, Chenango
Chittenango Creek	Madison, Onondaga
Delaware River	Delaware, Orange, Sullivan
East Branch Delaware River	Delaware
Genesee River	Livingston, Monroe, Wyoming
Hudson River	Warren
Lower Genesee River	Monroe
Mohawk River/Barge Canal	Herkimer, Montgomery, Oneida, Saratoga, Schenectady
Neversink River	Orange
Oak Orchard Creek	Genesee
Oswego River	Onondaga
Ramapo River	Orange
Raquette River	Franklin, St. Lawrence
Sandy Creek – 1	Jefferson
Schoharie Creek	Montgomery, Schenectady, Schoharie
Schroon River	Warren
Seneca River	Seneca, Cayuga, Onondaga
Susquehanna River	Delaware, Otsego, Broome, Chenango, Tioga
Tonawanda Creek	Genesee, Erie, Niagara, Wyoming
Wallkill River	Orange, Ulster
West Branch Delaware River	Delaware, Broome

When respondents to the Connelly et al. (1992) survey were sorted to exclude those anglers who had not consumed at least one fish meal from a "Hudson-like" water, only 95 respondents remained. The rates of consumption from these waterbodies were then calculated for those respondents. These rates are summarized in Table B-5.

Table B-5. Rates of Consumption from Hudson-like Waterbodies

Percentile	Consumption Rate
25^{th}	1.2
50^{th}	3.1
75^{th}	6.4
90^{th}	20.3
95^{th}	31.1
Arithmetic Mean	11

These rates are lower than the rates used in the HHRA even before corrections are made for non-response bias. It is likely that the correction for non-respondents would further reduce these estimates.

Finally, although Ebert et al. (1993) did not correct for non-response bias, this is not a sound basis for discarding those data. The available literature on non-response bias clearly indicates that individuals who do not respond to surveys of this type are less avid anglers and eat less fish than responding anglers (Brown and Wilkins, 1978; West et al. 1989a,b; Connelly et al., 1990; Connelly et al., 1992). Thus, the direction of bias in the survey is known. Because of this bias, it is likely that the Ebert et al. (1993) fish consumption overestimated actual consumption and would provide a conservative estimate for the HHRA.

Basis for Eliminating Connelly et al. (1996) From Further Consideration

EPA rejected the Connelly et al. (1996) survey of Lake Ontario anglers because this study focused on fish caught in the Great Lakes and alleged differences in the types of waterbodies and the primary species present. This study has substantial strengths that make it an important source of fish consumption information for the HHRA. The study was specifically designed to be a consumption study that targeted the total and sport-caught fish consumption of New York anglers who fished Lake Ontario. The survey used a diary approach to collect long term fish consumption data, minimize recall bias, differentiate between sport-caught and other fish, and identify portion sizes and preparation methods by meal and by species. While the survey focused on anglers who fished Lake Ontario, the data collected were not limited to Lake Ontario, and specific information was collected about consumption from individual waterbodies, including many rivers and streams. Thus, this survey provides valuable information about the consumption habits and preferences of New York anglers.

As shown in Table B-6, results of the Connelly et al. (1996) survey are similar to the Ebert et al. (1993) consumption estimates for “All Waters”.

Table B-6. Comparison of Connelly et al. (1996) Diary Survey with Ebert et al. (1993)

Consumption Rate Percentile	Connelly et al., 1996 Sport-caught Consumption	Ebert et al., 1993 All Waters Consumers
25^{th}	0.6	0.72
50^{th}	2.2	2.0
75^{th}	6.6	5.8
90^{th}	13.2	13
95^{th}	17.9	26
Arithmetic Mean	4.9	6.4

The similarities between these studies, and the support provided by other northeastern studies (ChemRisk, 1991; Ebert et al., 1996; Table B2 in the attached comments) indicate that consumption rates are fairly consistent among northeastern anglers. They also show that the Connelly et al. (1992) data are not consistent with other studies and thus may not be reliable estimates for the HHRA.

Selection of the Most Appropriate Fish Consumption Distribution

<u>Meal Sizes</u>

Using Connelly et al. (1992) required EPA to make an assumption about the size of each meal in order to derive annualized daily consumption rates. Such assumptions are not needed to use either the Connelly et al. (1996) or the Ebert et al. (1993) surveys. Connelly et al. (1996) required that each respondent record the size of each fish meal consumed as either “less than”, “equal to”, or “more than” a 0.5 pound meal pictured among the survey materials. While this approach also requires that some assumptions be made as to the actual sizes of the meals, it provides an added degree of precision not possible with the Connelly et al. (1992) data. Indeed, Connelly et al. (1996) assigned 5 ounces to represent meals that were less than 0.5 pound and 12 ounces to represent meals that were larger than 0.5 pound.

The Ebert et al. (1993) survey used a different approach for estimating the amount of fish consumed. In that survey, anglers were asked to report the length of each fish caught that was consumed. Then species-specific length weight regressions were used to calculate the mass of each fish consumed. It was then assumed that all edible mass of each fish was eaten. Thus, the consumption rates one can calculate from those survey data are based on actual edible masses of the fish consumed, rather than assumptions about meal sizes. While there is some uncertainty associated with these estimates, due to the fact that some of the edible fish may have been discarded, this uncertainty would result in the consumption rates being overestimated.

Species Lists

As discussed previously, the Connelly et al. (1992) survey instrument included a prescribed list of fish species that included only the species referenced in the current advisory. As a result, numerous species that might be consumed were not included in the species list, and 25 percent of fish meals were recorded as "Other" species. Limiting the listed species may have impeded accurate recall by participating anglers.

The Connelly et al. (1996) did not include a prescribed species list but instead asked respondents to list the species of each sport-caught meal consumed. Consequently, there were no "Other" species included in the survey data.

The Maine angler survey (Ebert et al. 1993) focused its species list on species that were most likely to be consumed. As a result, less than one percent of the fish consumed were categorized as "Other" species.

Segregation of Data by Waterbody Type

The Connelly et al. (1992) survey asked anglers to recall, by waterbody, the fish that they had caught and consumed. Waterbody-specific data allows consumption rates to be derived by waterbody type. While the approach used in the survey design was reasonable, its execution was compromised by the fact that approximately one third of the meals reported were not attributed to a specific waterbody. Consequently, EPA had to make assumptions about where those meals were obtained. As discussed previously, differing assumptions about the sources of fish yield considerably different estimates of consumption, resulting in substantial uncertainties in those estimates.

The Ebert et al. (1993) study does not suffer from this problem because respondents were required to record fish consumed in one of two categories of waterbodies: flowing or standing waters. Thus, all fish consumed can be attributed to a particular type of waterbody, thereby reducing the uncertainty in these estimates.

The Connelly et al. (1996) diary data do not permit fish meals to be segregated by waterbody type because individual meals were not attributed to a waterbody. Thus, consumption rates derived from the Connelly et al. (1996) data include total sport-caught consumption from all types of waterbodies combined, including both standing and flowing waters.

While one cannot segregate fish consumption from flowing waterbodies as opposed to lakes and ponds using the Connelly et al. (1996) data, consumption distribution can be developed using the data available from the Ebert et al. (1993) and Connelly et al. (1992) survey data. According to GE's analysis of the data provided in the Connelly et al. (1992) survey, rates of consumption from rivers and streams were $\leq$70 percent of rates from all waterbodies combined (excluding meals from waterbodies that could not be identified). When comparing consumption from flowing and standing waterbodies reported by Ebert et al. (1993), consumption from flowing waterbodies was $\leq$60 percent of consumption from all waterbodies. If the more conservative of these two ratios is applied to the Connelly et al. (1996) data, the ingestion rates in Table B-7 can be estimated for flowing water consumption by those anglers.

Table B-7. Estimation of Flowing Water Consumption Rates (g/day)[a]
Based on Total Consumption Reported by Connelly et al. (1996)

Percentile of Consumption	Estimates for Consumption from Flowing Waters Based on Connelly et al. (1996)
25th	0.42
50th	1.5
75th	4.6
90th	9.2
95th	13
Arithmetic Mean	3.4

a. Estimated by assuming that 70 percent of rates of total consumption (all waterbodies combined) could be attributed to consumption from flowing waterbodies.

Uncertainty in Fish Meal Estimates

As discussed previously, 179 respondents to the Connelly et al. (1992) survey provided a "?" in at least one section of the matrix related to the number of fish meals consumed. Because a number could not be assigned to the "?" responses, EPA dropped these fish meals from consideration in developing fish ingestion rates, increasing uncertainty and underestimating the ingestion distribution.

The Maine angler survey does not suffer from this problem because anglers were asked to recall all fish consumed from all waterbodies. Although recall and digit bias may introduce some uncertainty, it is likely that responses were more accurate than responses given as a "?". In addition, because both surveys were mail surveys with a one-year recall period, the direction and degree of recall bias is likely to be similar for both. Because long-term recall tends to result in overestimation of fishing activities (Westat Inc., 1989; West et al., 1989a,b; Connelly and Brown, 1995; Roach et al., 1999), it is likely that any inaccuracies from this type of bias result in an overestimation of fish consumed, providing an additional degree of conservatism in the Ebert et al. (1993) distribution.

The Connelly et al. (1996) survey does not suffer from this problem because survey respondents were asked to record all fish meals consumed on a daily basis. Consequently, it is likely that fish meals were not overlooked.

Sample Size

The Connelly et al. (1992) survey had an initial sample size of 2,000 licensed anglers. Of those, 1,033 individuals responded to the survey and 920 completed at least a portion of the fish consumption matrix. Of those individuals who completed the matrix, 601 (58 percent) had consumed at least one fish meal during the one-year survey period and only 226 (22 percent) had consumed a fish meal from flowing waterbodies.

The Ebert et al. (1993) data provide a more robust sample of ingestion rates. The initial sample size was 2,500 licensed Maine anglers. A total of 1,612 surveys were completed and returned. Of those, 1,053 individuals (65 percent) reported consuming at least one sport-caught fish meal during the one-year survey period and 464 individuals (29 percent) reported consuming at least one fish meal from flowing waterbodies during that period. Consequently, the Ebert et al. (1993) sample size that is more than twice as large as the sample provided by Connelly et al. (1992).

The number of individuals who consumed fish from flowing waterbodies can not be established from the Connelly et al. (1996) due to the fact that specific fish meals were not recorded on a waterbody-specific basis. However, a total of 853 individuals participated in the diary survey.

Response Rate

As discussed previously, the response rate reported by Connelly et al. (1992) was 52.3 percent.

The response rate reported by Ebert et al. (1993) was considerably higher (69 percent) and exceeded the 62 percent response rate that had been predicted for it using the Heberlein and Baumgartner (1978, 1981) model for predicting response rates to mail surveys. Thus, the survey performed above the standards for its design. A higher response rate means that a higher percentage of the actual survey population is represented and reduces non-response bias. Thus, it is likely that the calculated consumption rates are more representative of the total angler population.

The HHRA faulted the Ebert et al. (1993) study for not having completed a follow-up survey of non-respondents that would have allowed an adjustment for non-response bias in the survey results. The findings of other non-response follow-ups in studies of angler participation and consumption have shown that non-respondents tend to have lower participation and consume less fish than do respondents (Brown and Wilkins, 1978; West et al., 1989a,b; Connelly et al., 1990). This relationship was confirmed by the follow-up results reported by Connelly et al. (1992). As a result, it is likely that the Ebert et al. (1993) survey of Maine anglers overestimates consumption by the total angler population and thus represents a conservative estimate of consumption by freshwater recreational anglers in the Northeast.

The response rate for the Connelly et al. (1996) survey falls between these two surveys. Of the 1,410 anglers who were eligible for the study, 85 percent (1,210) agreed to participate in the study and, of those, 853 provided diary data. This means that of the eligible sample, only 60 percent participated in the survey. However, 70 percent of the individuals who agreed to participate actually provided diary data.

Summary

The selection of an appropriate fish ingestion rate distribution should be based on a survey of the population, region, and waterbody type being evaluated. A reliable study of fish consumption drawn from the Upper Hudson River is not possible in a catch-and-release fishery. The Connelly et al. (1996) and Ebert et al. (1993) data provide a more reliable basis for estimating consumption because:

- The data from both studies are regionally appropriate. Connelly et al. (1996) focused on a subset of New York anglers and Ebert et al. (1993) focused on all Maine anglers. The consumption behaviors of these two groups of anglers should not vary considerably from potential Hudson River anglers (in the absence of a ban or advisories). The Connelly et al. (1992) survey did focus on New York anglers but was not specific to the Hudson River.

- Both the Connelly et al. (1996) and Ebert et al. (1993) surveys focus on sport-caught fish consumption by freshwater recreational anglers in the northeastern U.S. who have substantial access to high quality fisheries with similar geography and a similar fishing season. In this respect, they are consistent with the data collected by Connelly et al. (1992) data.

- The demographics of the Maine anglers surveyed by Ebert et al. (1993) are similar to the demographics of the New York anglers surveyed by Connelly et al. (1992), indicating that there were no substantial socioeconomic differences between them (Table B-3).

- The Connelly et al. (1996) survey substantially reduced recall bias by using food diaries making the consumption rates derived from this study more accurate than the Connelly et al. (1992) survey data.

- The response rates for both the Ebert et al. (1993) and Connelly et al. (1996) surveys were higher than for Connelly et al. (1992) and are more representative of the targeted angler population.

- There is no need to make assumptions about meal sizes in deriving consumption estimates using Connelly et al. (1996) or Ebert et al. (1993) whereas EPA had to assume 0.5 pound for each meal recorded in the Connelly et al. (1992) survey, adding considerable uncertainty.

- The Ebert et al. (1993) fish consumption distribution for "All Waters, Consuming Anglers" is similar to the data collected in the Connelly et al. (1996) survey of New York's Lake Ontario anglers (Table B-7) but substantially less EPA's analysis of the Connelly et al. (1992) (Table B-8 and Figure B-1).

Table B-8. Comparison of Total Consumption by Anglers Participating in the Connelly et al. (1992), Connelly et al., (1996) and Ebert et al. (1993) Fish Consumption Studies.

Percentile of Consumption	Connelly et al. 1992 Total Consumption[a]	Ebert et al. (1993) Total Consumption[a]	Connelly et al. 1996 Total Consumption[a]
25th	2.5	0.72	0.60
50th	6.2	2.0	2.2
75th	14	5.8	6.6
90th	41	13	13
95th	81	26	18
Arithmetic Mean	18	6.4	4.9

a. Total sport-caught consumption reported by anglers participating in the surveys.

Because diary surveys are less subject to recall bias than mail surveys, that the Connelly et al. (1996) survey data are more representative of long-term consumption habits than are the Connelly et al. (1992) data. The similarities between Ebert et al. (1993) and Connelly et al. (1996) for all types of waterbodies show that there are no substantial differences in behavior between New York and Maine anglers and that EPA's analysis of Connelly et al. (1992) overestimates consumption by this population.

- The Maine angler survey was not substantially impacted by fish consumption advisories because fish consumption advisories were present on only 200 miles of the Maine's 37,000 miles of river and stream fisheries.

In sum, both the Connelly et al. (1996) and Ebert et al. (1993) surveys provide a stronger basis for the ingestion distribution for the HHRA than do the Connelly et al. (1992) survey data. EPA (1997) recognized the limitations of the Connelly et al. (1992) survey in its review of the fish consumption literature for the *Exposure Factors Handbook* and consequently did not select that survey as a "Key" study to evaluate sport-caught freshwater fish consumption by recreational anglers. EPA should recalculate exposure using data from either Ebert et al. (1993) or Connelly et al. (1996).

References

Brown, T.L, and B.T. Wilkins. 1978. Clues to reasons for non-response and its effect on variable estimates. *Journal of Leisure Research* 10(3):226-231.

Brown. T.L., J. Decker, and N.A. Connelly. 1989. Response to mail surveys ion resource-based recreation topics: A behavioral model and an empirical analysis. *Leisure Sciences* 11:99-110.

ChemRisk. 1991. *Penobscot River Creel Survey*. McLaren/Hart-ChemRisk, Portland, ME. November.

Connelly, N.A., T.L. Brown, and B.A. Knuth. 1990. *New York Statewide Angler Survey 1988.* New York State Department of Environmental Conservation, Division of Fish and Wildlife, Albany, NY. April.

Connelly, N.A., B.A. Knuth, and C.A. Bisogni. 1992. *Effects of the Health Advisory Changes on Fishing Habits and Fish Consumption in New York Sport Fisheries.* Human Dimension Research Unit, Department of Natural Resources, New York State College of Agriculture and Life Sciences, Fernow Hall, Cornell University, Ithaca, NY. Report for the New York Sea Grant Institute Project No. R/FHD-2-PD. September.

Connelly, N.A., and T.L. Brown. 1995. Use of Angler Diaries to Examine Biases Associated with 12-Month Recall on Mail Questionnaires. *Transactions of the American Fisheries Society* 124:413-422.

Connelly, N.A., B.A. Knuth, and T.L Brown. 1996. Sportfish Consumption Patterns of Lake Ontario Anglers and the Relationship to Health Advisories. *North American Journal of Fisheries Management* 16:90-101.

Ebert, E.S., N.W. Harrington, K.J. Boyle, J.W. Knight, and R.E. Keenan. 1993. Estimating consumption of freshwater fish among Maine anglers. *N. Am. J. Fish. Mgt.* 13:737-745.

Ebert, E.S., S.H. Su, T.J. Barry, M.N. Gray, and N.W. Harrington. 1996. Estimated rates of fish consumption by anglers participating in the Connecticut Housatonic River Creel Survey. *North American Journal of Fisheries Management* 16:81-89.

EPA. 1997. *Exposure Factors Handbook*. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. EPA/600/P-95/002Fa. Washington, D.C.

EPA. 1999. *Phase 2 Report – Review Copy, Further Site Characterization and Analysis. Volume 2F – Human Health Risk Assessment*, *Hudson River PCBs Reassessment RI/FS.* Prepared by TAMS Consultants, Inc. Gradient Corporation for the U.S. Environmental Protection Agency Region II and U.S. Army Corps of Engineers. August.

Heberlein, T.A. and R. Baumgartner. 1978. Factors affecting response rates to mailed questionnaires: A quantitative analysis of the published literature. *American Sociological Review* 43(4):447-462.

Heberlein, T.A. and R.M. Baumgartner. 1981. The effectiveness of the Heberlein-Baumgartner models for predicting response rates to mailed questionnaires: European and U.S. examples. *American Sociological Review* 46(3):361-363.

West, P., J.M. Fly, R. Marans and F. Larkin. 1989a. Michigan sport anglers fish consumption survey. A report to the Michigan Toxic Substance Control Commission. Natural Resource Sociology Research Lab Technical Report #1. May.

West, P., J.M. Fly, R. Marans, and F. Larkin. 1989b. Michigan sport anglers fish consumption survey. Supplement I – Non-response bias and consumption suppression effect adjustments. A Report to the Michigan Toxic Substance Control Commission. Natural Resource Sociology Research Lab Technical Report #2. September.

Westat, Inc. 1989. *Investigation of possible recall/preference period bias in national surveys of fishing, hunting and wildlife-associated recreation*. Final Report. Contract No. 14-16-009-87-008. Prepared for the U.S. Department of the Interior, Fish and Wildlife service, Washington, D.C. by Westat, Inc., Rockville, MD. December.

ATTACHMENT C
A Review of the Issues Associated with EPA's Probabilistic Risk Assessment in the Human Health Risk Assessment

The HHRA Fails to Provide an Adequate Description of the Probabilistic Assessment and Uncertainty Analysis

The Hudson River Human Health Risk Assessment (EPA, 1999) (HHRA) fails to provide an adequate description of the probabilistic model of exposure to potential PCB to anglers who might consume PCB containing fish from the Upper Hudson River. The limited description of the model impedes evaluation of comment on the model's structure. As discussed in EPA's guidance on Monte Carlo analyses (EPA, 1997), it is critical that sufficient information be provided to allow the reader to conduct an independent reproduction of the analysis. The level of detail provided in the HHRA fails to meet this and other requirements of EPA's guidance on acceptable Monte Carlo analyses.

The problem of model evaluation is greatly exacerbated by EPA's software selection. Monte Carlo assessments have typically been performed using Excel spreadsheets and commercially available software "add-ons," which allow one to provide only a limited description of the model, because the software provides standard formats for describing distributions, modeling decisions, and outputs. EPA's SAS software, however, lacks such standard formats. The code documentation for this model must clearly define all the steps in the analysis, including defining the inputs and managing the output of the analysis. More importantly, the software must perform the mechanics of the Monte Carlo analysis itself, including the following tasks:

- Generating random numbers,
- Randomly selecting values from the input distributions,
- Calculating the doses for each modeled individual based on the selected inputs, and
- Storing and tracking the doses.

The model must also select the input values in a specific order. For example, the values for body weight and fish concentration are a function of exposure duration, and the duration of exposure is, in turn, a function of each angler's age. Determining whether these functions are occurring properly is not feasible without access to the actual code. To allow review of the model, the Agency should have provided the following data:

- An electronic copy of the model itself,
- A list of instructions for running the model in SAS,
- A paper copy of the model code,
- A complete description of each step in the model in sufficient detail to allow another analyst to duplicate the step in another software program,
- Information on the nature of the random number generator used in the model,
- Information on any post-analysis manipulation of the output of the Monte Carlo model (selection of percentiles, etc.), and

- A copy of any QA/QC (debugging) assessments performed on the model.

In addition, information should have been provided on the specific model inputs:

- A paper and electronic copy of all model inputs for each of the 72 model runs,
- A copy of the raw data and description of the interim steps used in the derivation of the model inputs. (Data in the form of summary tables of select percentiles are not sufficient.), and
- A description of the process used by EPA to select the assumptions used in the uncertainty assessment.

The Probabilistic Assessment Fails to Meet Agency Guidance

EPA (1997) provides guidance to the regulated community on the preparation of probabilistic assessments and establishes the objective framework by which Agency personnel are expected to evaluate probabilistic analyses. The guidance establishes a number of criteria for probabilistic analysis. The model used in the HHRA fails to meet many criteria established by the EPA (1997b) for conducting acceptable probabilistic analyses.

1. *The methods used in the analysis must be well documented and easily located in the report, (i.e. there should be sufficient information to independently reproduce the results of the analysis.) Methods include:*
   - *All data,*
   - *All models, and*
   - *All the assumptions in the assessment that have a significant impact upon the results.*

The HHRA fails to provide an adequate description of many of the data sets used to derive inputs. Specifically, the report fails to include information on the specific data extracted from the Connelly et al. (1992) study or the specific consumption rate distributions taken from other studies.

As noted above, the HHRA fails to provide an adequate description of the model. As a result, one cannot determine if the model is operating as the Agency asserts.

2. *Documentation should include names of the models and software used to create the risk assessment analysis.*

As discussed above, merely providing the names of the software does not provide an adequate description of the model used in the assessment because the Agency used unique software.

3. *Sensitivity analysis results must be presented and discussed.*
    - *Probabilistic techniques should be applied to compounds, pathways, and factors of importance to the assessment, as determined by sensitivity analyses or other basic requirements of the assessment, and*
    - *Discuss and account for the presence or absence of moderate to strong correlations or dependencies between input variables along with the effects these have on the output distribution.*

While the HHRA includes a sensitivity analysis, this analysis was not used to refine the probabilistic analysis (e.g., by identifying those factors that are critical for inclusion in the probabilistic analysis), but is used in lieu of a true uncertainty analysis.

The HHRA does not provide any discussion of correlations between variables. Correlations that should have been considered include:

- Correlation between cooking methods (and cooking losses) and species of fish, and
- Avidity and the potential for recall bias.

4. *Information for each input and output distribution must be given in the report including:*
    - *Tables and graphs of the distributions,*
    - *An explanation for the choice of distributions, and*
    - *Differentiate variability and uncertainty for both input and output distributions.*

The HHRA fails to provide detailed descriptions of any of the inputs to the 72 model runs. Presenting the information as a graphic or in the form of a summary table is not a substitute for the actual model inputs.

While the HHRA includes extensive discussions of the differences between variability and uncertainty (p. 33 to p. 35), it does not separate uncertainty and variability in the Monte Carlo model and fails to provide any technical justification for not doing so.

5. *Exposure estimates from the probabilistic output distribution are to be aligned with the toxicity metric since fixed exposure assumptions are sometimes embedded in the toxicity metrics (e.g., Reference Doses, Reference Concentrations…)*

The estimates of exposure for chronic toxicity incorrectly include individuals who have exposure durations of only one or two years.

The Monte Carlo Model Suffers from a Number of Poor Design Decisions

Failure to Model Temporal Variation in Model Inputs Properly

The EPA probabilistic model does not fairly represent likely behavior of anglers. The fundamental structure proposed for the Monte Carlo analysis is sound, but is poorly and incompletely implemented. Although EPA acknowledges that modeling PCB exposures to anglers must be performed as a series of separate annual exposure events (HHRA at 36), the model fails to follow this framework. To the contrary, the Agency's model uses a "single" rather than a "nested" loop model of exposure as described by Price et al. (1996), which greatly limits the Agency's ability to model temporal changes in angler behavior and thus exposure. Each input of the dose equation is assigned a single value which is held constant for a block of time equal to the duration of an angler's exposure, a period ranging from one year to more than 30 years. This approach eliminates the ability to model each year's annual exposures as separate and varying events. Consequently, Equation 3-1 (describing intake as the product of the sum of the annual intakes) is not the basis of the model; rather the model is based on the simpler and more limited equation given at the top of page 36.

The HHRA attempts to address this limitation by first modeling the duration of exposure and then, based on the duration, estimating time-weighted averages of body weight and fish PCB concentrations. This approach might work if time-weighted averages can be defined based on the selection of an initial value of the input (for the first year) and the duration. In the case of body weight, this approach may be reasonable. However, this approach cannot model factors that may change randomly over time. For example, changes in fishing frequency, fishing success, and species consumed are likely to change over time in a random fashion rather than as a simple progression. The model also cannot incorporate time-dependent information on the uncertainties in estimates of inputs. The Agency's model is incapable of capturing most of the important temporal changes in angler behavior.

As a result, the model requires anglers to consume identical amounts of fish, to fish in exactly the same locations, consume exactly the same species, and prepare the fish in exactly the same manner for every year of their exposure.

Failure to Characterize Variation and Uncertainty Jointly

A second problem in model design is its failure to model uncertainty separately from variability. The Agency defends this decision (HHRA at 34-35) by stating that "an explicit 2-D analysis was not performed due to insufficient information available to define quantitative uncertainty distributions for several important exposure factors. The analysis conducted here includes a 1-D Monte Carlo analysis of the variability of exposure as a function of the variability of individual exposure factors." The HHRA, however, provides no demonstration of the alleged insufficiency of information to perform at 2-D model. Although the HHRA discusses many sources of uncertainty, it does not explain how these sources prevent the development of a combined measure of uncertainty and variability. For example, the Agency does not explain that any specific source of uncertainty in fish consumption rates makes it impossible to produce a joint distribution of uncertainty and variability. Numerous techniques exist for merging the results from multiple studies, ranging from meta-analysis to simple systems of weighting the studies, but the HHRA does not discuss such techniques.

The Agency's failure to perform a 2-D Monte Carlo analysis compromises the assessment. First, it prevents a quantitative evaluation of uncertainty. A sensitivity analysis can be useful for identifying those factors that make a significant contribution to the uncertainty of the final estimates of risk, but it cannot be used to characterize uncertainty. Second, it leads to the differences in measurements resulting from uncertainty being embedded in the measures of variability.

Failure to Model Chronic Dose Rates Properly

A third problem in model design is the incorrect calculation of chronic risks for anglers with short-term exposures. The HHRA, (at 36) defines averaging time (AT) in days as the exposure duration multiplied by 365 days. This approach systematically overestimates the chronic dose for anglers whose exposure duration is less than seven years.

The reference dose for PCBs is intended to evaluate chronic exposures. Therefore, only chronic exposures (seven years or greater) should be used in the non-carcinogenic risk assessment. EPA's approach results in evaluating anglers who are exposed for only one or two years as if those exposures occurred over seven years.

This error significantly affects the risk estimate on the upper tail of the risk distribution for non-carcinogenic endpoints, because those exposures will be the highest during the first few years. For example, if an angler has a high fish consumption rate but only consumes fish for a single year (e.g., 1999), then his dose will be determined by the fish concentration in that initial year. Because the PCB levels are highest in the initial year, the modeled dose received by the individual will also be high. A second angler who has the same consumption rate but who fishes for seven years will have a lower estimate of dose, because his dose will be based on the average concentration over the seven years. Clearly, the second angler has the higher chronic dose yet the model will rank his risk as being lower than the risk to the first angler. In fact, the first angler should not be considered at all in the evaluation of chronic non-carcinogenic risk from PCBs, because his exposure does not occur over a sufficient duration to warrant a comparison to the chronic PCB reference dose.

The model's structural limitations prevent the investigation of inter-year variation in fish consumption and preclude the quantitative characterization of uncertainty. As a result, the Agency's model is incapable of providing the information necessary to make a remedial decision for the Upper Hudson River.

Evaluation of Uncertainty in Probabilistic Assessment

EPA Chose an Inappropriate Methodology and Misrepresented the Implications of Its Findings

EPA's evaluation of uncertainty in the estimates of fish consumption is inadequate, and its reliance on a sensitivity analysis to characterize uncertainty is inappropriate. A sensitivity analysis is a useful technique for identifying which inputs and which types of uncertainty in specific inputs have the greatest impact on the results of an analysis. EPA (1997a) identifies this technique as a useful tool for focusing a probabilistic assessment on significant pathways and parameters, but it is not as powerful as Discrete Probability Analysis (DPA) and two-dimensional Monte Carlo models.

In a sensitivity analysis, one determines the impact of varying model inputs on the results of the model. The results of this analysis determine whether a model's outputs are sensitive to a change in inputs. The findings of the sensitivity analysis are strictly limited by the choice of what types of changes are made to the model's inputs. In contrast, DPA is performed by expressing the choice of inputs as a series of discrete values or options that have been selected so as to span all possible values of interest (Morgan and Henrion, 1990). For example, if the range of values of a model's inputs is divided into three categories of high, medium, and low, then it is possible to run the model for every permutation of high, medium, and low for each of the inputs to the model. The resulting set of outputs provides insight into the range and relative distribution of the uncertainty in the model outputs.

Both sensitivity analysis and DPA are performed by running the same model multiple times and each time varying the inputs. However, in the case of DPA there are additional requirements on the range of input values. In DPA, the analyst must show that the categories of the values for input (high, medium, and low) fully bound the range of all reasonable values. In addition, the analyst must show that the values selected from arranged values provide a representative spacing across the range of plausible values. (For example, if adult height is an input to the model, values of 4½, 5½, and 6½ ft might be reasonable spacing for low, medium and high values for heights in the general population, while values of 6¼, 6½, 6¾ ft. would not be reasonable). In contrast, in a sensitivity analysis, the analyst merely selects among the various options for input values and examines the impact of the selection on the model's results.

The HHRA only includes a sensitivity analysis but presents the outputs of the 72 different model runs as if they were the results of DPA. The Agency uses terms such as "base", "low", and "high" (Tables 5-38, 5-39, B-1 through B-9, and in the table on page ES-6) as if to suggest that the selection of the alternative sets of assumptions could be viewed as bounding the uncertainty in the estimates of variability. The comparison of the 72 values to the results of the point estimates of the RME carries the same implication.

This is a misuse of the analysis. The Agency has not demonstrated that the factors selected for evaluation in the uncertainty analysis fully capture all on the sources of bias and uncertainty in the estimates of dose and risk. In addition, the Agency has not demonstrated that the choice of options or values for each of the inputs investigated in the 72 model runs represents an equal spacing across the range of plausible values.

For example, the Agency investigated the impact of the choice of four studies for the distribution of fish consumption rates (pages 59 and 79). The results of the model runs demonstrate that the choice of study affects the estimate of the 95th percentile of risk by about a factor of four. EPA's preferred study (Connelly et al., 1992) falls in the middle of this range. Thus, the analysis suggests that the choice of study is important to the estimate of risk and that the Agency's Base Case is a moderate choice. However, the Agency offers no documentation that the selection of these four studies represents the entire range of plausible distributions of fish consumption or that the four studies represent an equal spacing across the plausible range of distributions. Without demonstrating these points, the 72 model runs do not necessarily characterize the range or distribution of uncertainty in the dose estimates for percentiles of the dose and risk distributions.

The results of EPA's sensitivity analysis depend on 1) the choice of studies included in the sensitivity analysis and 2) the decision to exclude consideration of other factors that influence the estimate of fish consumption. EPA included in the sensitivity analysis a study of Michigan anglers who fish the highly productive Lake Michigan (West et al., 1989a,b). The relevance of this study to the Hudson River is questionable. In fact, in the discussion of relevant angler studies given in Section 3.2.1.1 of the HHRA, the West et al. (1989a,b) study is not even considered. If this study were removed from the sensitivity analysis, then the findings would be considerably different. Of the remaining three studies, two studies would give very similar answers and the third study (Connelly et al., 1992) would give risks that are two-fold higher. This would suggest that EPA's base case is an overestimate of risk. If the assessment also included the findings of ChemRisk (1991) (West Branch Penobscot River) and Ebert et al. (1996) (Connecticut reaches of the Housatonic River), then the Connelly et al. (1992) data would appear to be an even more of an outlier.

The Agency should have performed a two-dimensional Monte Carlo analysis, focusing on those factors that contribute the most to the dose estimates of the most highly exposed individuals. Sensitivity analysis should only be used to identify the critical sources of uncertainty.

<u>The Agency Has Failed to Justify Its Decision not to Perform a Two-Dimensional Monte Carlo Model of Variability and Uncertainty</u>

EPA asserts that there are insufficient data to characterize uncertainty and variability jointly using parametric uncertainty. While the Agency indicates that it views uncertainty in distributions in terms of parametric uncertainty, nowhere does the Agency actually define the uncertainty in the parameters of the distributions of variability or identify what factors or data gaps prevent it from defining the parameters and their uncertainties.

There are other mechanisms for characterizing uncertainty in distributions of interindividual variation besides parametric uncertainty that could have been explored. For example, it is possible to develop empirical distributions of uncertainty and variability using two-dimensional matrices (Cullen and Frey, 1999). In addition, where the data are in the form of a series of discrete distributions (such as the findings of different surveys of anglers), techniques such as meta-analysis or systems of weights can be used to characterize uncertainty.

The Agency Has Failed to Make Proper Choices for the Selection of the Sources of Uncertainty Evaluated in the Sensitivity Analysis

EPA has made poor and inappropriate choices in the selection of factors to investigate in the sensitivity analysis. The Agency has examined the impact of alternative decisions in four areas: fishing location, fish ingestion rates, exposure duration, and cooking loss. The choice of two of these factors is highly questionable.

Investigating the impact of different exposure durations is also a poor choice. As discussed on pages 56 and 57, there is little difference between the distributions of exposure duration that are based upon residential mobility and those that are based jointly upon residential mobility and cessation of angling. As a result, the Agency should have concluded that exposure duration has minimal impact on the final estimates of risk.

The choice of cooking loss is inappropriate because the impact of the three identified options is obvious and does not require separate model runs. An average cooking loss of either 20 or 40 percent has a direct and linear effect on the final exposure and risk estimates. In addition, cooking loss is best modeled as a function of an individual's preference for cooking method and the species consumed. Because these factors differ across individuals, a single value should not have been used; rather the value should have been defined separately for each angler.

EPA should have considered other sources of uncertainty in its estimates of fish consumption rates. First, the Agency should have investigated the impact of the recall bias associated with twelve-month recall surveys. As discussed by Connelly and Brown (1995), twelve-month recall surveys have been shown to overestimate fish consumption rates by a factor of two among anglers who fish more than six days in a year. In contrast, consumption rates are only slightly overestimated for less avid anglers. EPA should have investigated this bias among high anglers should be investigated by the Agency for both the Maine angler survey (Ebert et al., 1993) and the Connelly et al. (1992) survey.

The Connelly et al. (1992) study is the basis for the Agency's baseline Monte Carlo assessment. In deriving a distribution of fish consumption rates, the Agency has been forced to perform a number of manipulations on the Connelly et al. (1992) data. These manipulations require a number of assumptions on the part of the Agency. The impact of these assumptions should have been investigated in the uncertainty assessment. The assumptions include:

- The decision to use consumption rates from multiple bodies of flowing water to evaluate the consumption rate of Upper Hudson River anglers,
- The decision to apportion fish meals obtained from unidentified bodies of water into flowing and non-flowing water categories, based upon the ratio of flowing to non-flowing waters,
- The assumption that "unknown" bodies of water in angler records with only flowing waters must also be flowing, and
- The assumption that all anglers who completed a survey form but did not indicate that they consumed fish were non-consuming anglers (catch-and-release anglers).

The Agency should have developed a distribution of consumption rates by randomly selecting the record of fish consumption from a single flowing body of water for each angler. This distribution is likely to reflect more accurately the potential consumption rates for Upper Hudson River anglers because the Upper Hudson River is a single source.

To investigate this point, GE conducted an analysis of the Connelly et al. (1992) data in which rates of consumption from single, flowing waterbodies were estimated for all anglers who consumed at least one fish meal from a flowing water. To do this, each flowing water angler was included and the first flowing waterbody reported by that individual was selected. Based on the number of meals consumed from that waterbody, a single-water body consumption rate was derived for that individual. Results of the analysis are provided in Table C-1.

Table C-1. Distribution of Single Waterbody Consumption Rates for Connelly et al. (1992) Anglers Who Consumed Fish from Flowing Waters

Percentile of Consumption	Single Waterbody Consumption Rate
25^{th}	1.24
50^{th}	2.49
75^{th}	6.22
90^{th}	18.04
95^{th}	29.54
Arithmetic Mean	8.91

On page 42 of the HHRA the Agency used an equation to assign the fish meals from unidentified waterbodies into either flowing or non-flowing waterbody categories. The Agency should also have investigated the impact of assuming that the unknown waters were either all non-flowing or all flowing. One of the implications of the equation on page 42 is the assumption that anglers who consumed fish from non-flowing waters and unidentified waterbodies did not consume any fish from flowing waters. This assumption is arbitrary because there is no reason why that angler could not have fished a flowing waterbody. This suggests that when the Agency investigates the impact of the alternative assumption of considering all unknown waters as flowing waterbodies, all anglers with consumption rates from unidentified waters should be included in the analysis.

The Agency assumed that an angler who completed the survey form but who did not indicate consuming a fish meal was a catch-and-release angler. It is plausible that certain anglers who catch and consume fish from flowing waters are not always successful every year. As a result, a certain fraction of anglers completing the form indicating that they did not consume fish are likely to consume some fish during their careers as anglers. These anglers should be viewed as having average consumption rates that are below the minimum detection limit of the survey, i.e., one meal per year. The Agency should consider the impact of this assumption by assigning those anglers an average consumption of one-half meal per year. This would add anglers with low consumption rates to the current 226 anglers. While it is unlikely that all of these anglers are low consumption anglers (i.e., not catch-and-release anglers), the Agency should nevertheless investigate how the estimates of risk would have been impacted by this alternative assumption.

In addition to the assumptions used to derive the distribution of annual fish consumption rates, the Agency should have investigated the impact of year-to-year variation in fish consumption. As discussed on page 74 of the HHRA, the Agency has assumed that the consumption rates for each angler will remain constant from year-to-year. Assumptions concerning the stability of annual consumption rates across years have significant effects on estimates of the upper percentiles of distributions of chronic doses (Price et al., 1996). Therefore, the impact of this assumption should also be considered in the sensitivity analysis.

Finally**,** EPA failed to investigate the uncertainty in measures of toxicity. This decision is unwarranted and results in biased estimates of risk. Information on the uncertainty in the cancer slope factor and in the reference dose is reported by a number of authors in the peer reviewed literature (Evans et al., 1994 a,b; Baird et al., 1996; Swartout et al., 1998). The Agency should have considered this large source of uncertainty (McKone and Bogen, 1991).

References

Baird, S.J.S., J. T. Cohen, J. D. Graham, A. I. Shlyakhter and J. S. Evans, 1996. Noncancer Risk Assessment: Probabilistic Characterization of Population Threshold Doses, HERA. 2(1), 78-99.

ChemRisk. 1991. *Penobscot River Creel Survey*. McLaren/Hart-ChemRisk, Portland, ME. November.

Connelly, N.A., B.A. Knuth, and C.A. Bisogni. 1992. *Effects of the Health Advisory Changes on Fishing Habits and Fish Consumption in New York Sport Fisheries.* Human Dimension Research Unit, Department of Natural Resources, New York State College of Agriculture and Life Sciences, Fernow Hall, Cornell University, Ithaca, NY. Report for the New York Sea Grant Institute Project No. R/FHD-2-PD. September.

Connelly, N.A. and T.L. Brown, 1995. *Use of Angler Diaries to Examine Biases Associated with 12-month Recall on Mail Questionnaires.* Transactions of the American Fisheries Society 124: 413-422.

Cullen, A.C., H.C. Frey. 1999. *Probabilistic Techniques in Exposure Assessment*. Plenum Press, New York, NY.

Ebert, E.S., N.W. Harrington, K.J. Boyle, J.W. Knight, and R.E. Keenan. 1993. Estimating consumption of freshwater fish among Maine anglers. *N. Am. J. Fish. Mgt.* 13:737-745.

Ebert, E.S., S.H. Su, T.J. Barry, M.N. Gray, and N.W. Harrington. 1996. Estimated rates of fish consumption by anglers participating in the Connecticut Housatonic River Creel Survey. *North American Journal of Fisheries Management* 16:81-89.

EPA. 1997. "Policy for Use of Probabilistic Analysis in Risk Assessment at the U.S. Environmental Protection Agency." Office of Research and Development, Washington, DC, USEPA/630/R-97/001.

EPA. 1999. *Phase 2 Report – Review Copy, Further Site Characterization and Analysis. Volume 2F – Human Health Risk Assessment, Hudson River PCBs Reassessment RI/FS.* Prepared by TAMS Consultants, Inc. Gradient Corporation for the U.S. Environmental Protection Agency Region II and U.S. Army Corps of Engineers. August.

Evans, J.S., J.D. Graham, G.M. Gray, and R.L. Sielken, Jr. 1994a. A distributional approach to characterizing low-dose cancer risk. *Risk Anal*. 14, pp. 25-34.

Evans, J.S., G.M. Gray, R.L. Sielken, A.E. Smith, C. Valdez-Flores, and J.D, Graham. 1994b. Use of probabilistic expert judgment in uncertainty analysis of carcinogenic potency, *Reg Tox and Pharm*, 20:15-36.

McKone, T.E. and K.T. Bogen. 1991. Predicting the uncertainties in risk assessment. *Environ. Sci. Technol.* 25(10):1674-1681.

Morgan, M.G., M. Henrion. 1990. *Uncertainty: A Guide to Dealing with Uncertainty in Quantitative Risk and Policy Analysis*, Cambridge University Press, New York.

Price, P.S., S.H. Su, J.R. Harrington, and R.E. Keenan. 1996. Uncertainty and variation in indirect exposure assessments: An analysis of exposure to tetrachlorodibenzo-*p*-dioxin from a beef consumption pathway. *Risk Analysis* 16(2):263-277.

Swartout, J.C., P.S. Price, M.L. Dourson, H. Carlson-Lynch, and R.E. Keenan. 1998. A probabilistic framework for the reference dose, *Risk Anal*. (18)3, 271-282

West, P., J.M. Fly, R. Marans and F. Larkin. 1989a. Michigan sport anglers fish consumption survey. A report to the Michigan Toxic Substance Control Commission. Natural Resource Sociology Research Lab Technical Report #1. May.

West, P., J.M. Fly, R. Marans, and F. Larkin. 1989b. Michigan sport anglers fish consumption survey. Supplement I – Non-response bias and consumption suppression effect adjustments. A Report to the Michigan Toxic Substance Control Commission. Natural Resource Sociology Research Lab Technical Report #2. September.

Bruce Young/MOYFH/US/BCS/BAYER

10/16/2009 09:39 AM

To gallagher.kathryn@epa.gov
cc
Subject Comments to docket EPA-HQ-ORD-2009-0645

Hi Kathryn,

My colleague (Gary Mihlan) with Bayer CropScience submitted comments yesterday to docket EPA-HQ-ORD-2009-0645:
Notice of Availability of the External Peer Review Draft of Using Probabilistic Methods To Enhance the Role of Risk Analysis in Decision-Making With Case Study Examples: Correction

He didn't receive a confirmation number. Just checking to see if you received the comments.
The deadline was extended to today Oct. 16, 2009.
If you have nothing from us, let me know so we can resubmit the comments.

Best Regards,
Bruce M. Young
Exposure & Risk Assessment Modeler

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ORD Docket
Environmental Protection Agency
Mailcode: 28221T, 1200
Pennsylvania Ave., NW.
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October 15, 2009

RE: EPA Docket Number EPA-HQ-ORD-2009-0645

Dear Sirs,

Bayer CropScience US, Product Safety Management provides the following comments to the draft document *Using Probabilistic Methods to Enhance the Role of Risk Analysis in Decision-Making With Case Study Examples, Prepared by Risk Assessment Forum, PRA Technical Panel Working Groups*, EPA/100/R-09/001. Our comments focus on the application of probabilistic risk assessment (PRA) in human exposure and risk assessment.

1. EPA's Experience with the Use of Probabilistic Risk Analysis

<u>Page 10</u>

Once developed, however, some of the more complex models have been used many times for different assessments. All have stood the test of internal and external peer review. A list of the case study examples presented in Appendix D are provided in Table 1 including categorizations based on type of assessment (i.e., human health or ecological risk assessment); PRA tools used in the assessment; and program office or region responsible for the assessment. In several cases, the examples presented represent components of the overall risk assessment that demonstrate use of multiple PRA techniques.

<u>Pages 17-18</u>

The concept of "validation" of models used for regulatory decision making has been a topic of heated discussion. In a recent report on the use of models in environmental regulatory decision making, NRC recommended the use of the notion of model "evaluation" rather than "validation," suggesting use of a process that encompasses the entire life cycle of the model and recognizes the spectrum of interested parties in the application of the model, which often extends beyond the model builder and decision maker. Such a process can be designed to ensure that judgment of the model application is based not only on its predictive value determined from comparison with historical data but also on its comprehensiveness, rigor in development, transparency, and interpretability (NRC, 2007b).

The tools used in the application of probabilistic risk analysis (PRA) for some case studies in Appendix D do not meet the criteria stated on page 10 of having "... stood the test of internal and external peer review". Although selected external peer reviewers have been used by the Agency, all interested stakeholders are not offered the opportunity to assess the PRA tools developed by the Agency. Specifically, the example in Appendix D regarding the application of SHEDS in the risk assessment for chromated copper arsenate (CCA) has provided the PRA tools to selected external peer reviewers but has refused to provide access to interested stakeholders including regulated industries impacted by the PRA model. Although the Agency did obtain limited external peer of the SHEDS PRA model, interested stakeholders continue to be excluded from the review process. We also cite the statements on pages 17-18 of the document that describes the evaluation process for a probabilistic risk assessment model, that should encompass the entire "life cycle" of the model, that is "… based not only on its predictive value determined from comparison with historical data but also on its comprehensiveness, rigor in development, transparency, and interpretability (NRC, 2007b)". Our experience with PRA tools used or developed by the Agency has varied, observing both exemplary and less than ideal examples.

However, the Agency is to be commended for providing transparency, interpretability and public access to other PRA tools including the Dietary Exposure Evaluation Model (DEEM, Appendix D, Case study 4) developed by Durango Software and Novigen Sciences. Critical to the usefulness of the DEEM PRA model is the quality of input data available for conducting a dietary risk assessment. USDA played a critical role in the applicability of the PRA tool through the collection of high quality data including the Continuing Survey of Food Intakes by Individuals (CFSII) and the establishment of the Pesticide Data Program (PDP), a residue monitoring program for raw agricultural commodities. Both U.S. EPA's Office of Pesticide Programs and USDA's Agricultural Marketing Service are to be commended as an example of Federal agencies working together to provide an important PRA tool used in the human risk assessment process. The high quality data and access to PRA tools has provided both the Agency and stakeholders with a useful risk management tool. Both Agencies are to be commended for their policies of providing open access to critical PRA tools and data. The Agency should follow a similar strategy for the development of, and continued use of, other PRA tools and data.

2. Recommendations for Enhanced Utilization of PRA in EPA

Page 22

- *Provide easily available, flexible, modular training for all levels of experience to familiarize employees to the menu of tools and their capacities.*
- *Provide introductory as well as advanced training open to all offices.*

The recommendations on page 22 of the document describe training resources to educate risk managers at the Agency. These recommendations lack any mention of regulated stakeholders. We recommend that the Agency not implement such a program without stakeholder participation. Although not a PRA model, we cite the online BMDS tutorial provided for the Benchmark Dose Software developed by U.S. EPA NCEA for evaluation of dose-response data as a good example of tools that could be developed for all interested stakeholders (http://www.epa.gov/NCEA/bmds/bmds_training/index.htm). Continued development of probabilistic methods for risk analysis decision making in areas such as probabilistic dose–response assessments, should follow the guidance recommendations of transparency, clarity, and consistency. The implementation of any probabilistic risk assessment tool developed by the

Agency should also include adequate documentation in the form of technical manuals and user guides that describe all aspects of the PRA tool.

3. PRA Variability and Uncertainty

Among the models currently used by U.S. EPA, Office of Pesticide Programs (OPP) for cumulative and aggregate risk assessments of pesticides, the SHEDS model appears to provide the most concise and thorough handling of uncertainty and variability of the available models. Specific comments regarding the uncertainty, variability and sensitivity analysis methods used in the model have been addressed by others and we will not comment on those specific techniques at this time but refer those interested to review these documents (e.g. Mokhtari A., and Frey, H.C. *Review and Recommendation of Methods for Sensitivity and Uncertainty Analysis for the Stochastic Human Exposure and Dose Simulation (SHEDS) Models Volume 1: Review of Available Methods for Conducting Sensitivity and Uncertainty Analysis in Probabilistic Models* (2005) prepared by North Carolina State University for Alion Science and Technology, Inc., Durham, NC; Mokhtari A., and Frey, H.C. *Review and Recommendation of Methods for Sensitivity and Uncertainty Analysis for the Stochastic Human Exposure and Dose Simulation (SHEDS) Models Volume 2: Evaluation and Recommendation of Methodology for Conducting Sensitivity Analysis in Probabilistic Models* (2005), prepared by North Carolina State University for Alion Science and Technology, Inc., Durham, NC).

U.S. EPA's Office of Research and Development, National Exposure Research Laboratory is to be commended for providing concise documentation for the SHEDS PRA model and addressing uncertainty and variability in a complex model used to evaluate pesticide exposures and the risk assessment process. We look forward to additional development of graphical data to describe the uncertainty and variability, and hope that the developers open the peer review process to interested stakeholders.

Respectfully submitted,

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