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Re: Upper Hudson River--Comments on EPA Response to Comments on the Human Health Risk Assessment Scope of Work

Dear Doug:

Attached is the reply of the General Electric Company on the responsiveness summary issued by the U.S. EPA for the Hudson River Human Health Risk Assessment Scope of Work (SOW). We were disappointed to see that a number of weaknesses in the original SOW have not been addressed. A major problem is that the EPA models which will be used to estimate future PCB exposure for the risk assessment are not reliable and the output of such models should not be used in the risk assessment. GE recently submitted comments detailing the problems with the EPA model.

Additionally, since the SOW has been issued new information on the toxicity of PCBs has been produced that needs to be factored into the risk assessment. Dr. Renate Kimbrough, just published the results of the largest study of PCB exposed workers ever conducted. This study found no association between PCB exposure and mortality due to cancer or any other disease. Another concern is that the best designed and most applicable study to be used for estimating fish consumption will not be used. Instead results from studies with major limitations will be substituted.

Please place a copy of these comments and associated attachments into the site administrative record. GE requests that these comments and concerns be addressed before EPA releases the human health risk assessment for the upper Hudson River Superfund site.

Yours truly,

John G. Haggard

JGH/bg
Attachment

cc: Richard Caspe, USEPA
Bill McCabe, USEPA
Doug Fischer, USEPA
Walt Demick, NYDEC
Anders Carlson, NYDOH
Bob Montione, NYDOH

General Electric Company's Reply to
The United States Environmental Protection Agency's
Responsiveness Summary for Phase 2
Human Health Risk Assessment Scope of Work

General Electric Company ("GE") submits this reply to the United States Environmental Protection Agency's ("EPA" or "Agency") "Responsiveness Summary for Phase 2 – Human Health Risk Assessment Scope of Work" ("Responsiveness Summary"). This reply highlights the significant problems with the Agency's planned Human Health Risk Assessment ("HHRA") for the Hudson River PCBs Superfund Site ("Site").[1]

The major problems with the Agency's planned approach for the HHRA are several-fold. First, the Responsiveness Summary reveals EPA's intent to use the output of its fate, transport and bioaccumulation models before subjecting those models to public comment and peer review and before correcting the obvious problems admitted to by EPA and also detailed in GE's recently-submitted comments on the Baseline Modeling Report. These comments demonstrate that EPA's models, in their current state, are not capable of accurately and reliably predicting future PCB concentrations in sediment, water and fish. As a result, the models will not provide valid and appropriate inputs for the HHRA. Before relying on the models for the HHRA, the Agency must consider public and peer reviewer comments and make appropriate and necessary changes to the models.

Second, in assessing PCB toxicity, EPA must consider the findings of both Kimbrough, et al. (1999), which reports no increase in cancer mortality for workers exposed to significantly higher levels of PCBs than those EPA will consider in the HHRA, and ATSDR's recently issued draft Toxicological Profile (ATSDR 1998).

Third, EPA must be careful in how it uses the data from the various surveys of New York anglers reported in Connelly, et al. (1990, 1992, 1996) and the Barclay (1993) to derive consumption rates and species preferences for Hudson River anglers. Each of these studies has significant limitations. Indeed, elements of EPA's basis for rejecting the Ebert, et al. (1993) survey of Maine anglers could easily apply to most of these New York/Hudson River studies.

Finally, EPA has not clearly explained why it will not consider the Monte Carlo modeling materials submitted by GE in the HHRA. EPA is obligated to provide a reasoned explanation for this decision.

We review these issues in turn.

[1] This reply is limited to GE's significant concerns. By not raising an issue, GE is not withdrawing from EPA consideration any of the issues raised in GE's original comments.

I. EPA should not rely on the Output of Fate, Transport and Bioaccumulation Models that Have Not Been Subjected to Public Comment and Peer Review.

A critical component of the HHRA is estimating potential risks to human health. To perform this task, EPA will need valid and reliable estimates of future PCB concentrations in water, sediment, and fish. The only reliable tools to provide such estimates are properly calibrated and validated fate, transport and bioaccumulation models. Indeed, the Responsiveness Summary states that the Agency intends to use the output of the fate, transport and bioaccumulation models presented in the Baseline Model Report ("BMR") (Responsiveness Summary at 16), and GE generally concurs with this approach.

EPA issued the BMR on May 18, 1999, and in public meetings described it as a "work in progress." The comment period on the BMR closed on June 23, 1999. GE's comments on the BMR show that there are significant problems with the models, particularly the bioaccumulation models. For example, the models grossly overstate PCB levels in fish at Stillwater, which will result in an exaggerated and unrealistic assessment of risk.

EPA intends to issue its response to comments and initiate its peer review of the models in January 2000, with the actual peer review to occur in March 2000. Under this schedule, it is likely that EPA will not be able to complete the models until the summer of 2000, at the earliest. Nevertheless, EPA's schedule calls for release of the HHRA in August 1999, and it is likely that near-final drafts of the HHRA report are now being reviewed within the Agency. This means that, for one of the most important parameters to be considered in the HHRA – future PCB concentrations –, EPA will be using the output of models that do not reflect changes that might result from public comments and the peer review. The HHRA should incorporate data based on final and complete models, not ones that are very likely to be changed.

To use incomplete and non-final models, models that EPA itself has acknowledged need modifications, will result in a misleading and incorrect assessment of risks to human health in the future. An HHRA on which one cannot rely has little utility for a risk manager.

II. Toxicity Issues

A. Findings in Kimbrough, et al. (1999)

GE's comments on the HHRA SOW contained substantial information about the potential health effects of PCBs, including a proposed alternative cancer slope factor based upon epidemiological study's. The Responsiveness Summary rejects much of this information on alleged weaknesses in the epidemiological database. Since submission of GE's comments, the largest study of PCB exposed workers ever conducted found was published, finding no association between actual human exposure and mortality from cancer or any other diseases (Kimbrough et al. 1999). The cohort consisted of 4,062 men

and 3,013 women who worked between 1946 and 1977 in GE's Hudson Falls and Fort Edward capacitor manufacturing plants. 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%) deceased males and 432 (14%) deceased females. Death certificates were available for 98.5% of the decedents and only 1.3% of the cohort was lost during follow-up. For comparison, standardized mortality rates (SMRs) were calculated using both U.S. and local county mortality tables. The major findings of the study are as follows:

- The workers' exposure to PCBs resulted in significantly higher blood concentrations of PCBs than found in the general population based on historical data from the worker population.

- 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 increases or decreases in mortality associated with length of employment or latency.

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

These findings are consistent with four previous studies of the same cohort or related cohorts (Brown and Jones 1981; Brown 1987; Nicholson et al. 1987; Taylor 1988) and studies of other cohorts (Cogliano, 1998; Danse et al. 1997; Kimbrough 1988; Kimbrough 1995; Longnecker et al. 1997; Smith 1997; Swanson et al. 1995; Vater et al. 1995; Ward et al. 1997; Chase, et al. 1989). EPA must consider this epidemiological information in the HHRA.

B. Findings of ATSDR's Draft Toxicological Profile

Since issuance of the SOW, the Agency for Toxic Substances and Disease Registry ("ATSDR") released an updated draft of its Toxicological Profile for Polychlorinated Biphenyls. ATSDR's conclusions on PCB are relevant to and need to be considered in the HHRA for the Hudson River:

- Death: "No studies were located regarding death in humans after exposure to PCBs by any route. . . . The acute lethality data do not suggest that PCBs would be acutely toxic in humans." (pages 205 and 206)

- Cancer: "The weight of evidence does not support a causal association for PCBs and human cancer at this time." (page 227)

- Respiratory effects: "[Respiratory] effects cannot be definitely attributed to PCBs due to study limitations such as lack of control data, co-exposure to other chemicals, insufficient corroboration, and lack of confirmation in follow-up evaluations. . . . Overall, there is inconclusive evidence that the respiratory tract is a target of PCBs in humans." (page 206)

- Gastrointestinal effects: "Nonspecific symptoms such as a loss of appetite, nausea, epigastric distress and pain, and intolerance to fatty foods have been experienced by workers exposed to PCBs . . . . No apparent gastrointestinal effects were reported in environmentally exposed populations . . . . The human data for gastrointestinal effects of PCBs are inconclusive and the relevance of the animal data seems questionable since most animal studies used doses much higher than current background levels for the general population and presumably also higher than those experienced by workers exposed to PCBs for months or years." (page 207)

- Hepatotoxicity: "Considering the generally small increases, inconsistencies, and other issues associated with the serum enzyme and lipid data, and the uncorroborated report of hepatomegaly, there is weak evidence that occupational inhalation exposure to PCBs causes hepatotoxicity in humans." (page 30)

- Renal effects: "There is no evidence of an association between PCB exposure and renal toxicity or kidney disease in occupationally . . . or environmentally . . . exposed subjects. . . . The relevance of the renal effects observed in animals treated with high doses of PCBs to human health is unclear since the exposure levels were much higher than current background levels for the general population and higher than those to which workers may have been exposed." (page 215)

- Developmental effects: "Results from some studies in the United States in which exposure to PCBs was assumed to have been by consumption of contaminated fish have raised the possibility that exposure to PCBs causes development effects in humans. . . . The overall evidence suggesting that PCBs may represent a developmental hazard for human health is inconclusive." (pages 223 and 225)

- Neurological effects: "Limited information exists regarding neurological effects in adult humans following exposure to PCBs. . . . The toxicological significance of the reported neurological effects in rats is unknown, in particular since no apparent clinical signs of neurological damage were observed in the chronic study. The information is insufficient to assess the potential for neurological effects in adult humans exposed to PCBs." (pages 220 to 221)

- Dermal effects: "Dermal lesions including skin irritation, chloracne, and pigmentation of nails and skin have been observed in humans following occupational exposure to PCBs . . . . Overall, the existing evidence suggests that it is unlikely that

adverse dermal effects will appear in the general population due to background exposure to PCBs. Exposure to PCBs through contaminated fish consumption . . . , contaminated sludge use . . . , or residence near a PCB waste site . . . have not shown any significant dermal effect or chronic skin disease." (pages 216 to 217)

- Reproductive effects: "Conclusive information on reproductive effects of PCBs in humans was not located. . . . The conclusive evidence for reproductive effects in animals indicates that PCBs are a potential reproductive toxicant in humans." (pages 221 and 223)

- Cardiovascular effects: "Evidence of increased blood pressure or an association between serum levels of PCBs and hypertension in populations with occupational or environmental exposure to PCBs is negative or inconclusive. . . . The existing data are insufficient to infer possible cardiovascular toxicity of PCBs in humans." (page 207)

- Hematological effects: "Conclusive hematological alterations have not been observed in workers who were chronically exposed to PCBs . . . or in individuals environmentally exposed . . . . [T]here have been no reports of increased incidence of any particular abnormality or generalized syndrome suggesting hematotoxicity in individuals presumably exposed to PCB levels higher than those experienced by the monkeys." (page 208)

- Musculoskeletal effects: "The only information regarding musculoskeletal effects in humans exposed to PCBs is the report of joint pain in 11% of the workers exposed to a variety of Aroclors at concentrations of 0.007-11 mg/m^3 . . . . Information on the cause of this pain or whether it is related to duration of exposure was not provided in the study. . . . Based on the existing data, it is not possible to infer that similar skeletal effects could occur in exposed humans." (page 215)

- Body weight effects: "No information was located regarding body weight effects in humans after exposure to PCBs. Body weight loss and/or reduced body weight gain are commonly seen effects of PCB exposure in animals. . . . The relevance of the animal data to body weight effects in humans is unknown." (page 217)

- Immunotoxic effects: "Although the limited data on humans are inconclusive, the available evidence does not suggest that occupational exposures to PCBs were immunotoxic, and no association has been found between PCB exposure and excess mortality from infectious diseases." (page 220)

- Genotoxic effects: "The generally negative results of in vitro and in vivo genotoxicity studies suggest that the PCB mixtures tested do not pose a genotoxic threat to humans." (page 227)

ATSDR's conclusions confirm what the available data show:

- No person has ever died from exposure to PCBs, even at high occupational exposure levels. There is no evidence that PCBs cause blood-related diseases or kidney disease in humans. There is no evidence that PCBs can cause changes in or interfere with the human genetic code. Further, the weight of evidence supports the conclusions that PCBs do not cause cancer or cardiovascular, muscle or skeletal disease in humans, or interfere with the human immune system.

- There is some evidence that high-dose occupational exposure to PCBs in the past may have caused minor respiratory and gastrointestinal effects, as well as changes in some chemicals in the liver. Such effects are highly unlikely in the future, since human receptors no longer experience the exposure levels typical of those received by individuals that worked with PCBs when they were utilized in commerce.

- Although some studies have suggested that PCBs have caused minor developmental and/or neurological effects in the past, other studies have found no evidence of this. At current exposure levels, it is unlikely that PCBs are causing any such effects in humans.

- In the past, high occupational exposures to PCBs may have caused skin rashes, eye irritation, and other temporary adverse effects on the skin and eyes. At current exposure levels, it is unlikely that PCBs are causing any such effects in humans.

- Although some animal studies have suggested otherwise, there is little evidence that PCBs have caused or are causing reproductive effects in humans. Most human studies have found no association between PCBs and adverse reproductive outcomes. At current exposure levels, it is unlikely that PCBs are causing any such effects in humans.

EPA must consider the Draft Profile in the HHRA for the Hudson River.

III. Limitations of The New York Angler Surveys

In estimating exposure to recreationally-caught fish, it is critical to obtain an accurate assessment of the rate at which anglers consume fish and the distribution of species consumed by them. The Responsiveness Summary reaffirms EPA's intent to rely primarily on data from various surveys of New York and Hudson River anglers to derive these important inputs, downplaying the relevance of a survey of Maine anglers. EPA must recognize, however, that each of the New York angler surveys has its own distinct limitations, and that the Maine data set can provide useful insight and data for estimating the rates and types of fish consumed by Hudson River anglers.

A. Consumption Rates

The Responsiveness Summary (p. 17, 19-20) clarifies EPA's intent to rely primarily on the data from Connelly et al. (1990, 1992 and 1996) and Barclay (1993) and, to a lesser extent, Ebert et al. (1993), to establish consumption rates for the HHRA. EPA appears to favor the Connelly (and Barclay) data because they provide "comprehensive information about thousands of anglers within New York State, including anglers from similar rivers with similar type of fish." Responsiveness Summary at 17. In contrast, the Ebert data are less favored because

> [i]n addition to differences between New York and Maine anglers due to differences in climate, fish species present, general fishing regulations, etc., the Maine data set is further limited in that survey respondents were asked only about total fish consumption from all flowing water bodies, and not from individual water bodies separately. As a result, it was not possible to screen Maine data for more "Hudson-like" rivers and streams, as was possible with the Connelly data

The Responsiveness Summary ignores the limitations of the Connelly, et al. and Barclay data and overstates the difficulty of applying the Ebert data to the Hudson River.

1. Connelly Data Limitations

Although it is not clear from the Responsiveness Summary or the SOW on which of the Connelly data EPA intends to rely in establishing consumption rates, there are significant limitations in applying each the data sets to the Hudson river. We review these briefly below.

a. The Connelly, et al. (1990) Data

Connelly, et al. (1990), report the results from the1988 New York Statewide Freshwater Fishing Survey, which was designed to collect information on the fishing activities, preferences, and fish consumption behaviors of New York's licensed anglers. Given this breadth of coverage (statewide), applying its results to the Hudson is problematic.

First, consumption rates from the survey data do not represent consumption of sport-caught freshwater fish from a single river like the Hudson River. Fish meals consumed included all types (freshwater and marine) of purchased, fresh, canned or frozen fish meals obtained at stores and restaurants, as well as sport-caught freshwater fish.

Second, the only waterbody specific information for consumption of sport-caught fish involved meals consumed from Lake Ontario. Fish consumption by anglers fishing Lake Ontario is likely not representative of consumption from the Hudson River fishery given the differences in water bodies, fish species and general fishing regulations.

b. The Connelly, et al. (1992) Data

The data reported in Connelly et al. (1992) have limitations that affect their usefulness in assessing consumption rates for the Hudson River. Most fundamentally, the data are from a survey that was not designed to assess consumption by New York sports anglers. In addition, the data contain no portion size information. EPA (1997) identifies these same limitations.

c. The Connelly, et al. (1996) Data

Connelly et al. (1996) also suffers from limitations in this application:

- The study focused on the Lake Ontario fishery and asked anglers to recall their total consumption activities, including non-sport-caught fish as well as sport-caught fish from all waterbodies fished. Because the survey focused on individuals who lived near Lake Ontario, it collected little or no information about fish caught and consumed from tidally influenced areas, like the Middle Hudson.

- The mail survey did not collect information on the sizes of fish caught, sizes of meals consumed, or the number of fish caught that were retained for consumption.

- The first section of the diary survey also did not collect information on the portion sizes of the meals eaten or the number of individuals who consumed each fish. To use these data to develop consumption rates, one would either have to use a weight-length regression to estimate edible mass of each fish eaten and make assumptions about the number of individuals who ate that fish, or one would need to make assumptions about portion size. Because these assumptions could vary substantially from meal to meal and among species, consumption estimates derived in this manner would by highly uncertain.

- The second section of the diary survey, while focused on fishing trips to other New York State water bodies, does not contain any consumption information, such as the species of fish caught, whether the caught fish were retained, or whether they were eaten.

- Although the third section of the diary survey contains good information about consumption on a species-specific basis, including angler estimates of fish meal sizes, there is no water-body specific information. Thus, these data can not be partitioned for "Hudson-like" fisheries.

2. The Barclay Data Limitations

The Barclay (1993) data do not provide a basis for estimating long-term consumption rates.

- While anglers were asked to report what types of fish or crab they had eaten from the river in the past, questions related to meal frequency were not species-specific and combined all species of fish and crabs into a single category. This approach could lead to serious inaccuracies in determining consumption rates. For example, an individual might consume striped bass "2 or 3 times per week" during striped bass season, crab "once a week" throughout the year, and other species "less than once a month." When asked to indicate his/her frequency of eating fish or crab over the season, the angler could respond with any of these frequencies even though none would accurately represent the angler's consumption rate.

- There is no reliable way to relate consumption frequency during the previous week or month to seasonal consumption, which means that one would need to make numerous assumptions about seasonal consistency or variations.

- The data contain no information about meal size.

- The survey data are subject to avidity bias because the survey method used on-site interviews, (Price et al., 1993). That is, given the nature of the sampling method, it is likely that more avid anglers would be sampled more frequently than would more occasional anglers, due to their higher likelihood of being present at the survey site on a given day. This bias would likely result in substantial overestimation of consumption rates.

3. Use of the Ebert Data

As noted above, the Responsiveness Summary dismissed the Ebert, et al. (1993) data because of perceived differences in climate, fish species and fishing regulation in Maine and New York and the difficulty of segregating consumption data for flowing water bodies. Yet, all the Connelly, et al. data suffer from similar and other problems. For example, the Connelly, et al. (1990) report did not distinguish between sport-caught and commercially-obtained fish meals and also did not collect consumption information on a waterbody-specific basis. The Connelly, et al. (1992) report did not provide data about portion size, and the Connelly, et al. (1996) survey did not collect water-body specific consumption information.

Moreover, the consumption rates reported by Ebert, et al. (1993) for All Waters are very similar to the consumption rates reported by Connelly, et al. (1996), indicating consistency among northeastern fisheries.

Percentile of anglers	Sport-caught Consumption Rate (g/day) Connelly et al. (1996)	Ebert et al. (1993)
25th	0.6	0.72
50th	2.2	2.0
75th	6.6	5.8
90th	13.2	13
95th	17.9	26
99th	39.8	na

In addition, the Ebert et al. (1993) study provided additional consumption rate data for flowing waters which indicated that consumption rates from these fisheries were lower than consumption rates from All Waters combined. Thus, it is reasonable to assume, based on the Ebert et al. (1993) analysis, that the Connelly et al. (1996) consumption rate distribution overestimates consumption from the Upper Hudson River.

B. Species Preference

The Responsiveness Summary states the EPA intends to rely primarily on Barclay (1993) supplemented by the three Connelly studies to ascertain species preference for the HHRA. Although Barclay (1993) contains information on the species that anglers targeted, anglers were not asked to indicate whether these species were targeted for consumption or whether they were successful in obtaining them. In addition, while anglers were asked to report the type and number of fish caught, they were not asked to report which of those fish were retained or whether they would be consumed. Also, as noted above, Barclay (1993) suffers from avidity bias.

Some of the Connelly data suffer from similar problems:

- Although Connelly, et al. (1990) contains information about fish targeted in specific water bodies, it does not indicate whether these species were targeted for the purpose of consumption. In fact, many of these fish may have been targeted for other purposes (bait, trophy fish, fishing tournaments, etc.). Thus, it can not be assumed that the fish that were targeted in the Hudson River were indicative of the fish preferentially consumed by its anglers.

- The list of species reported in Connelly, et al. (1992) is inadequate to characterize species preference because it does not contain some key Hudson River species. For example, the list does not differentiate among bass species and does not include

important target species in the Hudson, such as striped bass, bluegills, white sucker, bluefish, and Atlantic silverside (NYSDOH, 1999). It is therefore not possible to derive species-specific consumption rates using these data.

Although GE believes that Ebert, et al. (1993) provides the best information on species preference, the data in the first section of the Diary Study in Connelly, et al. (1996) can also be used. In this section of the diary, anglers identified the waterbody fished, the species of each fish caught, its length, and whether or not the fish was kept for consumption. These data can be sorted by waterbody type to identify those species likely to be present in "Hudson-like" rivers, the likelihood that the angler who catches a particular species will consume it, and the sizes of the fish of each species kept for consumption.

IV. Rejection of Proposed Monte Carlo Model

GE's comments on the SOW recommended that EPA use the State-of-the Science Monte Carlo model of exposure to PCBs from fish consumption that GE had previously provided to EPA. (See GE Comments on pages 13-14.) In response, the Responsiveness Summary states that "for the reasons noted in the response to HP1-1, HP1-7, and HG1-1, USEPA will use a probabilistic risk analysis as outlined in the HHRASOW (p. 15) and not conduct the microexposure event analysis described in ChemRisk (1995)." Responsiveness Summary at 20. There are several problems with this response.

First, it is not evident to what microexposure event analysis EPA is referring. The document cited in the Responsiveness Summary (ChemRisk (1995)) is listed as "Determining the Intake of Upper Hudson River by Species." This is not the document and materials cited in GE's comments when it referred to the advanced Monte Carlo model. That document, also authored by ChemRisk, is entitled "Hudson River Superfund Project: Approach for Performing Human Health Risk Assessment – Estimating Potential PCB Exposure from Fish Consumption."

Second, assuming EPA meant to refer to the proper document, EPA's response to "HP1-1, HP1-7 and HG1-1" (Responsiveness Summary at 17-18) provides no "reasons" explaining EPA's decision not to "conduct the microexposure event analysis" recommended by GE. The referenced response addresses a number of issues relating whether the HHRA should or will consider subsistence anglers. This issue is not relevant to microexposure modeling, which is simply a technique to calculate an individual's total exposure to a contaminant by summing the doses received by many individual exposure events. GE again urges EPA to use such microexposure modeling because it allows variation of exposure over an individual's lifetime and thus more realistically reflects real world exposures to PCBs through fish consumption. At a minimum, EPA should provide a cogent explanation for why it will not use such an approach, something the Responsiveness Summary fails to provide.

V. Conclusion

Given data limitations and the fact that EPA's risk assessment will be based on conservative assumptions that do not reflect actual angler behavior on the Hudson River, the HHRA must explain that the risks it describes are hypothetical. Indeed, NYSDOH's recently-released survey (NYSDOH 1999) confirms the fact that little or no consumption of fish is occurring in the upper Hudson. Upon questioning by NYSDOH personnel, every single upper Hudson angler stated that no fish were being consumed. As EPA's HHRA will make assumptions that run counter to these statements, it is clear that the HHRA will not accurately reflect the real-world risks faced by Hudson River anglers.

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