COMMENTS OF THE GENERAL ELECTRIC COMPANY ON THE U.S. ENVIRONMENTAL PROTECTION AGENCY'S HUMAN HEALTH RISK ASSESSMENT FOR THE HOUSATONIC RIVER SITE – REST OF RIVER

Prepared for General Electric
by
AMEC Earth and Environmental, Inc.
and
BBL Sciences

July 28, 2003

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

ATTACHMENTS (Separately Bound)

EXECUTIVE SUMMARY

The General Electric Company (GE) is providing these comments to the U.S. Environmental Protection Agency (EPA) and the Peer Review Panel on EPA's public review draft of its *Human Health Risk Assessment, GE-Housatonic River Site, Rest of River* (HHRA), with the objective of presenting additional information, views, and analyses that may help to refine the HHRA to be more reflective of the potential for exposures and risks due to polychlorinated biphenyls (PCBs) in the Rest of River portion of the Housatonic River and its floodplain. These comments generally follow the structure of the Peer Review Charge developed by EPA. As such, they first discuss each of the three risk assessments in the HHRA – the Direct Contact Assessment, the Fish and Waterfowl Consumption Assessment, and the Agricultural Products Consumption Assessment – focusing primarily on the inputs affecting exposure. They then discuss more general issues, including the approaches and values used in the HHRA to assess toxicity, which are critical issues affecting all three risk assessments. (The following sections of this Executive Summary reference in parentheses the corresponding sections of the comments where the points are discussed in more detail.)

Site-Specific Empirical Data Affecting Exposure and Health (Section 2.3)

At the outset, it should be noted that, although the HHRA relies on hypothetical exposure and risk estimates, there are some actual empirical data that relate to exposures and health risks in the Housatonic River area. For example, in 2002, GE consultants conducted an intensive Floodplain User Survey of recreational use of the floodplain areas in the most contaminated part of the floodplain (the stretch between the East/West Branch Confluence and Woods Pond) (TER, 2003). Much of the floodplain in this stretch (apart from residential properties) consists of wetlands or backwaters and/or dense vegetation, with only certain specific areas, such as trails and other recreational areas, more accessible to human use. Consistent with these physical characteristics, the survey showed that the majority of floodplain areas receive little or no recreational use and that only a relatively few areas have regular recreational use.

In addition, data collected by the Massachusetts Department of Public Health (MADPH, 1997) on PCB levels in blood collected from individuals in the area who were identified as having a high potential for PCB exposure showed that the blood PCB levels in non-occupationally exposed individuals in this survey were, in fact, within the normal background range. Further, the latest cancer incidence data reported by ATSDR (2002) for the local area show that cancer rates for the towns adjacent to the most contaminated portion of the river are not elevated and not associated with areas having high PCB concentrations.

These site-specific real-world data provide an important backdrop for evaluating the hypothetical assumptions contained in the HHRA.

Direct Contact Assessment (Section 3)

GE's principal concern with the exposure estimates contained in the Direct Contact Assessment for the various identified Exposure Areas (EAs) is that, for all the key exposure parameters, the HHRA uses either upper-bound values or values that appear to be unrealistically high, at least for the Reasonable Maximum Exposure (RME) estimates. This approach is not consistent with EPA (1992a) guidance. The combination of these values results in exposure estimates that go well beyond what can reasonably be expected to occur. The most significant of these parameters are as follows:

- <u>Selection of Exposure Scenarios</u>. For some EAs and subareas, the HHRA's assignment of exposure scenarios is not consistent with current and reasonably anticipated land use. For example, the angler scenario is inappropriately applied to certain riverbank areas with limited access and dense vegetation, where no anglers were observed in GE's Floodplain User Survey. In addition, the future residential scenario is applied to certain non-residential areas where future residential use is not reasonably anticipated.

- <u>Exposure Point Concentrations (EPCs)</u>. The HHRA calculates the EPC only for the portion of each EA within the 1 ppm PCB isopleth. This is appropriate for areas where the main use is within that isopleth, but not for areas where the assumed use is expected to occur both within and outside that isopleth. The EPCs need to be adjusted in the latter cases to reflect the fraction of use inside the isopleth. In addition, in calculating the EPCs, the HHRA uses the 95% upper confidence limit (UCL) on the mean of the sampling data, calculated by various statistical techniques, or else the maximum detected concentration (if lower than the UCL). The technique used to calculate the UCL for areas downstream of Woods Pond – the Land H-statistic – is not appropriate for environmental data sets, especially those with small sample sizes, such as the data sets for these areas.

- Exposure Frequencies and Durations. For several exposure scenarios and EAs, the assumed RME exposure frequencies and durations are unrealistically high. For example:

  ⇒ For many EAs, the HHRA assumes an exposure frequency of 90 days/year for general recreation. For the majority of these areas, that assumption is not realistic given physical constraints, and is inconsistent with the empirical results of GE's Floodplain User Survey. For instance, there are 27 EAs for which the HHRA assigns this frequency but at which the survey showed either no recreational users or six or fewer total users over the season, despite the extensive coverage of the survey.

  ⇒ The HHRA assumes that an angler will fish in the same floodplain location 60 days/year (2 days/week for 7 mos/year) for 60 years and that a waterfowl hunter will hunt in the same area 48 days/year (3 days/week for every week of the 16-week hunting season) for 60 years. These assumptions are not realistic, not consistent with site conditions, and not supported by available survey information.

  ⇒ Similarly, the HHRA assumes that an adolescent dirt biker will ride in the same area (limited to the 1 ppm isopleth) 3 days/week for 7 months/year for 12 years, which fails to take account of schedule conflicts during the school year and changes in behavior during adolescence.

- Soil Ingestion Rates. The HHRA uses upper-bound soil ingestion rates derived from studies by Calabrese and colleagues. However, newer and improved soil ingestion studies by the same investigators show that these rates are overstated, and support rates of about half those used in the HHRA.

- PCB Dermal Absorption Factor. The dermal absorption factor used in the HHRA for PCBs (14 percent) is based on a monkey study (Wester et al., 1993) that used soil with very low organic carbon content, which is not typical of floodplain soils and would promote bioavailability. A newer, similar study (Mayes et al., 2002) using floodplain soil with more typical organic carbon content shows a lower dermal absorption rate (around 4 percent).

Thus, the principal exposure assumptions used in the Direct Contact Assessment were all selected to be upper-bound values and, in several cases, values are unrealistically high and/or not supported by more recent data. The combination of using all these parameter values results in exposure and thus risk estimates that are overstated and not representative of actual exposures in the floodplain, even for reasonably maximum exposed individuals.

Fish and Waterfowl Consumption Assessment (Section 4)

The HHRA includes both point estimate analyses and probabilistic analyses (using both simple and Microexposure Event [MEE] Monte Carlo techniques) for the Fish and Waterfowl Consumption Assessment. GE has substantial concerns with both approaches.

For the point estimate analyses, the HHRA again selects upper-bound or unrealistically high RME values for the key exposure parameters, as illustrated below:

- Exposure Point Concentration. The HHRA uses the 95% UCLs of the fish and duck tissue data as the EPCs. For certain of these data sets, it uses the Land H-statistic to calculate the EPCs. This technique can greatly overstate the true upper bound on the mean and thus should not be used, at least for the smaller data sets (< 30 samples).

- Fish Consumption Rates. While GE supports the HHRA's use of the Maine angler survey by Ebert et al. (1993) as the basis for deriving fish consumption rates, the HHRA has not selected the most applicable set of fish consumption rates from that survey:

  ⇒ It assumes that anglers do not share their catch with other household members, which is not reasonable and is not consistent with the survey data.

  ⇒ It uses the consumption rates for fish caught from "all waters," which included multiple rivers and lakes. These rates overestimate consumption from a single river reach. The HHRA should use the rates for either rivers or lakes, as appropriate.

- Failure To Account for Waterfowl Migration. The HHRA assumes that all waterfowl consumed are local residents. This is not reasonable since most resident ducks begin migration near the beginning of the hunting season and a large fraction of waterfowl shot and consumed are migrants. EPA should adjust either the EPC or the waterfowl consumption rate to account for migration.

- Cooking Loss. The HHRA does not take account of PCB loss due to cooking in its RME scenarios. It should do so since cooking loss is a well-documented parameter.

- Exposure Duration. The HHRA assumes that consumers eat fish and waterfowl from the Housatonic River for 60 years, based on data from a survey that asked people how many years they had been consuming freshwater fish from *all* sources (including store-bought fish and fish from other waterbodies). These responses cannot provide a reliable estimate of the duration of eating fish or game from this specific river.

Again, due to the combination of these upper-bound and unrealistically high exposure assumptions, the HHRA's point estimate analyses almost certainly overestimate exposures and thus risks due to fish and waterfowl consumption.

The HHRA's probabilistic analyses should, but do not, represent a significant improvement over the point estimate analyses. This is because these analyses use some of the same assumptions, do not make full use of the available data, do not adequately address correlations among parameters, and artificially expand some of the distributions by the inclusion of hypothetical maximum values that are not plausible. For example:

- The analyses continue to use only the 95% UCLs as point estimate EPCs, rather than using distributions of the full range of fish and duck tissue concentrations.

- They use the same "all waters-no sharing" fish consumption rates used in the point estimate analyses.

- They inflate the distributions of fish consumption rates and waterfowl meal sizes by adding hypothetical upper-bound maximums that are not based on the data. Some of these are wholly unrealistic and implausible – e.g., the assumption that a person would eat more than 1,100 half-pound fish meals from the Housatonic River every year for 70 years.

- They do not take adequate account of correlations among certain input variables.

- They do not consider or evaluate uncertainties in the toxicity values, which can be done in probabilistic analyses and would be useful at least as a sensitivity analysis.

To evaluate the effect of these issues, AMEC has performed an alternative MEE analysis of fish consumption, making modifications to address the above issues. That alternative analysis includes: (1) one set of model runs using distributions for the exposure parameters but EPA's point-estimate toxicity values (AMEC MEE 1); and (2) as a further sensitivity analysis, another set of model runs using distributions for both the exposure parameters and the toxicity values

(AMEC MEE 2). In both cases, this alternative MEE analysis shows substantially lower predicted risks that those in the HHRA, as demonstrated in the comparisons of results shown in Table ES-1. As that alternative analysis illustrates, the probabilistic analyses contained in the HHRA significantly overestimate the exposures and risks due to fish and waterfowl consumption.

Agricultural Products Consumption Assessment

The Agricultural Products Consumption Assessment is unique in that, rather than evaluating specific areas or actual data from the site, it evaluates two pre-determined example concentrations of total PCBs (tPCBs) – 0.5 and 2 ppm – along with the concentrations of other chemicals of potential concern (COPCs) (dioxin-like PCB congeners, dioxins, and furans) determined by regression analyses to be associated with those tPCB concentrations. In addition, it relies almost entirely on modeling, with very little site-specific supporting data, to estimate COPC uptake from soil to plants to animals to humans.

GE's initial concern with this assessment is that the HHRA assumes that 100 percent of the pasture and cultivated areas are within the 1 ppm isopleth. In fact, this assumption does not apply to any of the farms in the floodplain, at which only fractions of the pasture/cultivated lands fall within the 1 ppm isopleth. Recognizing this fact, the HHRA explains briefly how to make an adjustment so as to apply its calculations to areas where only a portion of the farm land is within this isopleth. That adjustment, however, does not address the problem that EPA's modeling assumes that farm animals are at steady-state conditions relative to COPCs in the soil, which is not true, especially if only a portion of the animals' diet comes from within the 1 ppm isopleth. Moreover, since the HHRA's assumption does not apply to any actual properties, the resulting risk estimates provide a misleading picture of site risks. While GE does not disagree with the approach of using pre-determined example soil concentrations, GE believes that EPA should use a more realistic assumption for its basic calculations – e.g., that 15 percent of pasture/cultivated lands are in the 1 ppm isopleth (the average in Reach 5) – and explain how to adjust those results based on the actual portion in the floodplain at a given property.

The HHRA also apparently assumes that the agricultural scenarios could apply, as future use, to virtually the entire floodplain in Massachusetts. That is not realistic due to the unlikelihood of future farm development and legal restrictions on such development.

Table ES-1. Comparison of PCB Cancer and Non-Cancer Risk Estimates: EPA's MEE Analysis vs. AMEC's MEE Analyses

River Reach	Cancer Risks Adult/Child					
	50th %ile			95th %ile		
	EPA HHRA	AMEC MEE		EPA HHRA	AMEC MEE	
	MEE	MEE 1	MEE 2	MEE	MEE 1	MEE 2
5 to 6	2E-03	5E-05	2E-05	8E-03	8E-04	4E-04
8	2E-03	4E-05	1E-05	6E-03	6E-04	2E-04
11 to 12 (trout)	3E-04	9E-06	3E-06	1E-03	2E-04	6E-05
11 to 12 (bass)	2E-04	5E-06	2E-06	7E-04	8E-05	3E-05
14 to 15	1E-04	5E-06	2E-06	5E-04	8E-05	3E-05

River Reach	Non-Cancer Hazard Indices											
	Adult						Child					
	50th %ile			95th %ile			50th %ile			95th %ile		
	EPA HHRA	AMEC MEE		EPA HHRA	AMEC MEE		EPA HHRA	AMEC MEE		EPA HHRA	AMEC MEE	
	MEE	MEE 1	MEE 2	MEE	MEE 1	MEE 2	MEE	MEE 1	MEE 2	MEE	MEE 1	MEE 2
5 to 6	40	5.4	0.42	500	69	7.4	91	3.1	0.33	1000	49	6.6
8	29	4.3	0.33	330	44	4.6	61	2.7	0.28	720	28	3.9
11 to 12 (trout)	3.7	0.95	0.07	64	12	1.3	7.9	0.65	0.07	110	7.8	1.1
11 to 12 (bass)	3.7	0.50	0.04	44	6.2	0.65	7.7	0.36	0.04	91	4.0	0.57
14 to 15	2.4	0.56	0.04	31	5.8	0.60	5.3	0.35	0.04	61	3.6	0.50

Most significantly, the HHRA relies on a series of modeling steps to predict the transfer of COPCs from soil to plant matter (e.g., grass, corn), from soil and plants to animal products (e.g., milk, beef, chicken, eggs), and from plant and animal products to human consumers. Very little site sampling data were available to support these modeling steps. In fact, despite EPA's apparent concern about these pathways, it did not collect any site-specific data on the animal products. Instead, the modeling was based on assumptions selected from literature values or extrapolated from very limited site data on soils and plants. Many of these modeling assumptions and estimates are highly uncertain and likely overstated. For example:

- The HHRA's conversion of the pre-set tPCB concentrations in soil to concentrations of the other COPCs is based on very limited data, which are much higher than the pre-set tPCB concentrations, resulting in unreliable estimates for the other COPCs.

- The HHRA's soil-to-grass transfer factors are based on data from a one-time sampling event, which was conducted during optimum conditions for uptake (e.g., hot and dry) and thus did not take account of other factors affecting uptake (e.g., meteorological conditions). Literature data indicate that these factors are overstated.

- Since PCBs were not detected in corn ears, soil-to-corn transfer factors were based only on limited and uncertain data from corn stalks. These results are unreliable and overestimate PCB transfer to corn.

- The HHRA's assumption that the farm animals are at steady state with COPC concentrations in the soil is not valid since the animals would, from day to day, graze or eat food from different areas, much of it outside the floodplain. This assumption likely overstates bioaccumulation.

- The HHRA's assumption that the chemicals in ingested material are 100 percent bioavailable is not supported by the literature.

- The HHRA's use of the maximum bioconcentration factors (BCFs) from the literature for PCBs in milk and body fat is not appropriate.

The net result of using these multiple layers of modeled assumptions, many of which are very uncertain and likely overstated, is a set of exposure and risk estimates that are both unreliable and almost certainly overestimated.

Toxicity Assessment (Section 6.1)

To assess risks, the HHRA uses toxicity values – i.e., Cancer Slope Factors (CSFs) for potential cancer risks and a Reference Dose (RfD) for non-cancer hazards – based exclusively on laboratory animal studies. To begin with, GE believes that the HHRA should recognize two recent weight-of-evidence evaluations of the human epidemiological and clinical studies, which demonstrate that there is little credible evidence that PCBs cause cancer in humans or that PCBs at environmental levels cause adverse non-cancer effects in humans. Further, accepting the use of animal-based toxicity values, GE has a number of serious concerns with the toxicity approaches and values used in the HHRA.

The first relates to the HHRA's use of the dioxin Toxicity Equivalency (TEQ) approach, in which concentrations of the so-called dioxin-like PCB congeners, as well as dioxin and furan compounds, are converted into TEQs of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) through the use of Toxicity Equivalency Factors (TEFs) and then are assessed for potential cancer risks using a CSF for TCDD. GE believes that, at the present time, this approach should not be applied to PCB congeners for several reasons:

- The application of the TEQ approach to PCBs is highly controversial and remains under review. The documents cited in the HHRA in support of that approach do not establish that it is an accepted EPA approach. In fact, that issue is a key aspect of the Agency's ongoing Dioxin Reassessment, which is not final, and is required by a Congressional directive to be reviewed by the National Academy of Sciences (NAS).

- Analyses based on empirical bioassay data show that the TEQ approach does not accurately predict the carcinogenic potency of PCB mixtures.

- This approach requires use of a highly uncertain CSF for TCDD, which is unnecessary given the availability of empirically based CSFs for PCBs.

- Application of both the PCB CSF and the TCDD CSF results in double counting the carcinogenic potential of the dioxin-like congeners in the PCB mixtures, since they are included in total PCBs. While the HHRA makes an adjustment (in the food consumption assessments) in an effort to account for this double counting, that adjustment is both inadequate and incorrect.

- The HHRA's predictions of PCB congener concentrations from tPCB data, based on limited comparisons, are highly uncertain and unreliable due to variability among river/floodplain reaches and the use of different laboratories for the comparisons and the main tPCB analyses.

GE's second concern relates to the CSF selected to assess the cancer risks of TCDD. There is no accepted CSF for TCDD at this time. The CSF used in the HHRA is based on an outdated interpretation of the pathology results for one tumor type from the underlying rat study, combined with an outdated rat-to-human scaling factor. Even accepting a linear non-threshold cancer model (which is questionable), correction for these factors would lead to a lower CSF for TCDD. Moreover, the HHRA should not cite at all, even its uncertainty analyses, the proposed TCDD CSF recommended in the draft Dioxin Reassessment, since that document is not final, is not Agency policy, and is required by Congressional directive to be reviewed by the NAS.

GE's third concern relates to the chronic Reference Dose (RfD) used in the HHRA to estimate non-cancer hazards of PCBs. That RfD was developed by EPA based on the application of various uncertainty factors (UFs) to the results of a long-term monkey dosing study. Two of those UFs, however, are inappropriate: (1) the UF to adjust for inter-species extrapolation, because the empirical data indicate that monkeys are *more* sensitive than humans to these PCB effects; and (2) the UF to adjust for use of a supposedly subchronic study to estimate chronic effects, because the monkey study was essentially a chronic study. Even accepting use of the same underlying study used by EPA to develop its RfD, correction for these UFs would result in an RfD that is 10 times higher. GE believes that that revised chronic RfD should be used in the HHRA. Under a court settlement with GE, EPA has an obligation to consider this alternative RfD. Moreover, the HHRA applies a chronic RfD to all exposure scenarios, regardless of length. However, consistent with EPA (1989a) guidance, subchronic exposures (e.g., those less than 7 years or highly intermittent exposures) should be evaluated using a subchronic RfD.

Risk Evaluation (Section 6.3)

The HHRA uses a Hazard Index (HI) of 1 as a benchmark for assessing non-cancer effects, implying that an HI over 1 is indicative of unacceptable non-cancer hazards. However, while HIs less than 1 are considered "safe," HIs greater than 1 are not necessarily indicative of unacceptable hazards due to the conservatism built into the RfD. The HHRA should recognize that.

Overall Conclusions (Section 7)

GE believes that the HHRA substantially overestimates potential risks at the site for the following principal reasons: First, it repeatedly and consistently selects upper-bound and, in many cases, unrealistically high exposure assumptions which, when combined, result in exposure profiles that are overstated and not representative of actual exposures. Second, where EPA has tried to refine the risk estimates through probabilistic analyses (i.e., in the Fish and Waterfowl Consumption Assessment), it has not used the full range of data and has introduced additional unnecessary conservatism, resulting in risk estimates that are not a significant improvement on the point estimates. Third, the HHRA uses toxicity approaches and values that are overly conservative and, in some cases (e.g., application of TEQ approach to PCBs), scientifically questionable. Fourth, the HHRA fails to consider site-specific empirical data (e.g., GE's Floodplain User Survey, the MADPH blood survey data for the Housatonic area, the latest local cancer incidence data) indicating that actual exposures and risks in the area are not as great as predicted in the HHRA.

GE believes that if the HHRA is revised to use the modifications and improvements suggested in these comments, the resulting exposure and risk estimates will provide a more representative, but still conservative, characterization of potential exposures and risks at the site. However, if changes are not made to address these concerns, GE believes that the HHRA will not present an accurate estimate of risks due to PCBs at the site and cannot serve as a supportable basis for making a remedial action decision for the site.

SECTION 1: INTRODUCTION

The General Electric Company (GE) appreciates the opportunity to submit these comments to the U.S. Environmental Protection Agency (EPA) and the Peer Review Panel on EPA's public review draft of the *Human Health Risk Assessment, GE-Housatonic River Site, Rest of River* (hereinafter "HHRA"), dated June 6, 2003. The objective of these comments is to provide EPA and the Peer Review Panel with additional information, viewpoints, and analyses that may help to refine the current HHRA to be more reflective of the potential for exposures and risks due to PCBs in the Rest of River portion of the Housatonic River.

These comments generally follow the structure of the Peer Review Charge developed by EPA, with relevant charge questions noted in parentheses at the beginning of each comment to assist the peer review panel members in focusing their review. These comments address most but not all of the charge questions. The main points of these comments are presented in text, with reference to a number of attachments that provide a more detailed discussion of certain specific issues and the basis of GE's recommendations on those issues. These comments discuss those areas where the HHRA can be improved and offer specific recommendations for its improvement. In some cases, the comments are relevant to only certain exposure areas or scenarios and the way that they are specifically being evaluated. In other cases, comments are provided on the general approach used in the HHRA.

It should be noted that, because the comments are organized to follow the Peer Review Charge, they are not necessarily discussed in order of importance. Consistent with the Charge, the comments first discuss each of the three risk assessments contained in the HHRA – the Direct Contact Assessment, the Fish and Waterfowl Consumption Assessment, and the Agricultural Products Consumption Assessment. The comments on these three risk assessments focus primarily on the inputs affecting exposure. We then provide comments on more general issues, including EPA's toxicity assessment and its selection of toxicity approaches and values. Although these toxicity issues are addressed toward the end of these comments (for consistency with the Peer Review Charge), they are critical issues that affect all three risk assessments. For example, these comments show that, at the present time, the dioxin toxicity equivalency (TEQ) approach should not be applied to evaluate the potential cancer effects of PCB congeners, given that doing so remains under scientific review and does not appear to accurately predict the carcinogenic potency of PCB mixtures. Thus, GE believes that the HHRA should be revised to limit its evaluation of the potential cancer risks of PCBs to the standard approach based on total PCBs (tPCBs).

In the following sections, for the convenience of the reader, the key points in each section are listed in bullets at the beginning of the section.

SECTION 2: OVERVIEW OF SITE

Key Points

- The primary exposure area evaluated in the HHRA consists of the 10-mile section of the Housatonic River and its floodplain between the Confluence of the East and West Branches of the river and Woods Pond Dam.

- The physical characteristics of the floodplain in this reach dictate the types and frequency of human use.

  ⇒ Much of the floodplain in this reach (excluding residential properties) consists of wetlands or backwaters and/or dense low-lying vegetation, which limit accessibility to and use of these areas. Certain specific areas, such as trails and some recreational areas, are more conducive to human use.

  ⇒ This is illustrated by an empirical Floodplain User Survey conducted by GE consultants from April through October 2002, including extensive, nearly daily observations of recreational use in these floodplain areas. This survey showed that the majority of floodplain areas receive little or nor recreational use, while only a relatively few areas have regular recreational use.

- The river in this reach is a shallow meandering stream with numerous backwaters. Access to the river is limited to bridges, a dirt road along a portion of the floodplain, a number of trails, and some boat launch areas. This section of the river is used for canoe racing and, in some locations, fishing.

- Since the historical releases of PCBs occurred decades ago, PCBs have been in the environment for many years. Thus, predicted effects can be checked against actual data. Empirical data on exposure and health effects in this area have been collected and provide a point of comparison for the HHRA.

  ⇒ Data collected by the Massachusetts Department of Public Health (MADPH) on PCB levels in blood from individuals identified as having a high potential for exposure indicate that the blood PCB concentrations in such non-occupationally exposed individuals do not exceed background levels.

  ⇒ The latest cancer incidence data collected for a number of towns along the Housatonic River indicate that cancer rates are not elevated and are not associated with areas having elevated PCB concentrations.

SECTION 2: OVERVIEW OF SITE

The Rest of River site spans from the Confluence of the East and West Branches of the Housatonic River in Pittsfield, Massachusetts, to Long Island Sound in Connecticut. It includes the Housatonic River and its associated sediments, riverbanks, and floodplain soils. In some areas the floodplain is very narrow, while in other areas it is extensive. Land uses adjacent to the river include residential, recreational, commercial, and agricultural uses.

2.1 Description of the Floodplain

The section of the floodplain that receives the greatest attention in the HHRA is the approximately 10-mile stretch between the Confluence and Woods Pond Dam, since the PCB concentrations in soil are the highest in that stretch. Much of this floodplain area consists of wetlands or backwaters and/or dense low-lying vegetation, as shown, for example, in Figures 1-4. These characteristics limit the accessibility to such areas and thus human use of the areas. Certain specific areas within the floodplain are more conducive to human use. These include portions of residential properties, trail networks along easements, the Canoe Meadows property (which provides boating access, hiking trails, and opportunities for bird-watching), and the Housatonic Valley Wildlife Management Areas (which provide opportunities for hiking and hunting). Examples are shown in Figures 5-8.

The physical characteristics of the floodplain dictate the types and frequency of human use of these floodplain areas. This is illustrated by an empirical Floodplain User Survey conducted by Triangle Economic Research (TER) at GE's request from April through October 2002. That survey, which is described in more detail in Attachment A to these comments, included intensive, almost daily observations of recreational use of most of the floodplain exposure areas identified by EPA between the Confluence and Woods Pond Dam. As discussed in Attachment A, this survey revealed that there are a few specific areas that are used regularly for recreational purposes, but that many floodplain areas receive little or no recreational use, consistent with the physical characteristics of those areas.

Figures 1-4. Areas with difficult access to the river and floodplain

Figure 1. EA10 – Heavy vegetation in off trail area of Audubon-Canoe Meadows

Figure 2. EA17 – View from trail on EA12 into EA 17 showing heavy vegetation

Figure 3. EA38 – Heavy vegetation along bank in EA that is being evaluated for the bank angler scenario

Figure 4. EA43 – Heavy vegetation along bank in EA that is being evaluated for the bank angler scenario

Figures 5-8. Areas with easy access to the river and floodplain

Figure 5. EA12 – Easily walkable trail with heavy vegetation on both sides of the trail

Figure 6. EA37 – Easily walkable trail with heavy vegetation on both sides of trail

Figure 7. EA40 – Easily walkable trail with heavy vegetation on both sides of trail

Figure 8. EA60 – Easy access to canoe launch at Woods Pond near footbridge

Moreover, there are several characteristics of the floodplain in this stretch that make it unlikely that, for many areas, future uses would be substantially different from or greater than current uses. These include the following:

- There is poor access due to slopes and wetlands in many areas. Since these characteristics will not change over time, frequency of use of these areas is unlikely to increase.

- Significant areas of floodplain are bordered by large State forests that are more conducive to recreational activities than the floodplain (e.g., October Mountain State Forest, Bear Town State Forest). The presence of these areas makes it more likely that individuals will use them, rather than the floodplain, for certain recreational activities.

- Approximately 75 percent of the floodplain between the Confluence and Woods Pond consists of publicly owned land. As noted in the HHRA (Vol. IIIA, p. 4-8), the State of Massachusetts has agreed to future use restrictions of these areas, which will ensure the continuation of current uses and prevent future development in these areas.

2.2 Description of River

The physical characteristics and uses of the Housatonic River change considerably as one moves downstream. Between the Confluence and Woods Pond Dam, the river is a small, shallow, meandering stream with numerous backwater areas. Woods Pond is a shallow 54-acre impoundment that was formed when a dam was constructed in the late 1800s. There is fairly limited access to the river from the Confluence south to Woods Pond. Three bridges and a dirt road along a portion of the floodplain provide the main access points. There are also a number of trails and utility easements that provide some access to the river. Further access to the channel is generally via canoe or small car-top boat, which can be launched from one of several access points along the river. The area between New Lenox Road and Woods Pond is used for canoe race training for a portion of the year. In addition, fishing may occur from canoes or small boats in the river, and there is limited access for bank fishing at New Lenox Road, at some points along October Mountain Road, and at Woods Pond.

Downstream of Woods Pond Dam in Massachusetts, the river is somewhat larger but still limited in size, is somewhat faster moving, and includes Rising Pond, which is a fairly large impoundment. This section provides more opportunity for bank fishing along the river itself and at Rising Pond. In Connecticut, the river increases in size and velocity. The Trout Management Area in northern Connecticut is regularly stocked with trout for catch-and-release fishing only.

There are two large lakes downstream in Connecticut, Lakes Lillinonah and Zoar, which are close to more developed urban areas and are used for a variety of recreational activities.

2.3 Empirical Data on Exposure and Health

The primary chemicals of potential concern (COPCs) for this HHRA are PCBs, which resulted mainly from historical releases from the GE facility in Pittsfield.[1] These historical releases occurred decades ago, beginning in the early 1930s and continuing until 1977 (one year before PCB use was generally prohibited by the Federal Government). As a result, PCBs have been present in the river sediment and floodplain soil for decades. Because little was known about the potential toxicity of PCBs prior to the 1970s, there were no restrictions in place relating to PCBs in the river or its biota until fish consumption advisories were first established (in 1977 in Connecticut and in 1982 in Massachusetts). In these circumstances, exposures to PCBs in sediment, soil, and biota likely occurred for many years. Thus, predicted effects of exposure to PCBs can be checked against actual data. A number of studies have been performed by government agencies to look at factors relevant both to PCB exposure and to cancer incidence in the area.

In 1997, the Massachusetts Department of Public Health (MADPH) issued a report on an exposure assessment conducted on individuals who lived in the Housatonic River valley (MADPH, 1997). This report included the results of a blood survey on PCB levels measured in blood samples taken from individuals who were identified as having a high potential for exposure based on their characteristics and activities (e.g. age and length of residence near the Housatonic River, recreational activities associated with the river). The blood serum PCB levels measured in non-occupationally exposed individuals in this survey were within the normal background range for non-occupationally exposed individuals nationwide, thus indicating that

[1] In addition to PCBs, all three risk assessments include evaluations of polychlorinated dibenzo-p-dioxins (PCDDs or dioxins) and polychlorinated dibenzofurans (PCDFs or furans) in the TEQ analyses. As shown in GE's recent RCRA Facility Investigation Report (BBL & QEA, 2003), review of the spatial distribution of the PCDD/PCDF sediment data indicates that there are multiple sources of these constituents, both upstream of the Confluence and within the Rest of River area (particularly downstream of Woods Pond). Further, the Fish and Waterfowl Consumption Assessment includes evaluations of several organochlorine pesticides and mercury, for which there is no evidence of a link to releases from the GE facility.

even individuals identified as having a high potential for exposure in this area did not have elevated serum PCB levels relative to those in the general population.

Another study conducted by the MADPH, under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR), evaluated the cancer incidence data for the communities along the Housatonic River to determine whether the presence of the GE facility and PCBs in the environment had resulted in an increased chance of cancer in the potentially exposed population (ATSDR, 2002). The cancer incidence data indicated that, from 1982 to 1994, residents of the Housatonic River area did not experience excessive rates of cancer incidence for the majority of cancer types evaluated and that, when cancer incidence was considered relative to known areas of PCB contamination, the "pattern of cancer incidence in the HRA did not suggest a relationship to PCB exposure" (p. 29). While two census tracts (CTs) in Pittsfield showed statistically significant elevations in cancers of the bladder, breast, and non-Hodgkin's lymphoma, ATSDR stated that "a pattern suggesting that a common environmental exposure pathway played a primary role in the CTs was not observed nor were cases distributed more toward the vicinity of the GE sites" (p. 30). In addition, ATSDR (2002) reported that an update to the study for the years 1995 to 1998 indicated that there were no elevations for any of the cancer types evaluated. During this period, the incidence of breast cancer was similar to the state-wide rates and the incidence rates for bladder cancer and non-Hodgkin's lymphoma, overall, were below expected rates for Pittsfield during that period. Thus, the updated data indicated that the elevation in certain cancer types that were observed between 1982 and 1994 did not persist during the 1995 to 1998 time period. In addition, during the 1995 to 1998 period, the other towns evaluated (Lee, Lenox, Great Barrington, and Stockbridge) had lower-than-expected incidence rates for all six cancer types evaluated.

These site-specific real-world data provide an important backdrop for evaluating the hypothetical assumptions contained in the HHRA.

SECTION 3: DIRECT CONTACT ASSESSMENT – PHASE 2

Key Points

- For some exposure areas (EAs) and subareas, the HHRA's assignment of exposure scenarios is not consistent with current and reasonably anticipated land use. For example, the angler scenario is inappropriately applied to certain riverbank areas with limited access and dense vegetation, where no anglers were observed in GE's Floodplain User Survey. In addition, the future residential scenario is applied to certain non-residential areas where future residential use is not reasonably anticipated.

- The HHRA limits the exposure point concentration (EPC) to the portion of each EA within the 1 ppm PCB isopleth. This is appropriate for areas where the main use is within that isopleth, but not for areas where the assumed use is expected to occur both within and outside that isopleth. The EPCs need to be adjusted in the latter cases to reflect the fraction of use inside the isopleth.

- In calculating the EPCs, the HHRA uses various statistical techniques to calculate the 95% upper confidence limit (UCL) on the mean. The technique used for areas downstream of Woods Pond – the Land H-statistic – is not appropriate for environmental data sets, especially those with small sample sizes, such as in these areas.

- For several exposure scenarios and EAs, the HHRA's assumed exposure frequencies and durations are unrealistically high. For example:

  ⇒ For many EAs, the assumed frequency of 90 days/year for general recreation is not realistic given physical constraints, and is inconsistent with the empirical results of GE's Floodplain User Survey, in which little or no recreational use was observed in 27 of these areas.

  ⇒ The assumed angler exposure frequency (60 days/year for 60 years), waterfowl hunter frequency (48 days/year for 60 years), and dirt biking/ATVing frequency (90 days/year for 12 years) in the same floodplain area are not realistic, not consistent with site conditions, and not supported by available information.

- The general upper-bound soil ingestion rates used in the HHRA are overstated, as shown by newer soil ingestion studies by the same investigators, which support rates of about half those used by EPA.

- The dermal absorption factor used in the HHRA for PCBs is based on a monkey study that used soil with very low organic carbon content, which is not typical of floodplain soils and would promote bioavailability. A newer, similar study using soil with more typical organic carbon content shows a lower dermal absorption rate.

- Overall, the HHRA uses upper-bound, and sometimes unrealistically high, values for all key exposure parameters. This is inconsistent with EPA guidance and, when combined, results in exposure estimates that are not representative of actual exposures in the floodplain, and consequently in risk estimates that are overstated.

SECTION 3: DIRECT CONTACT ASSESSMENT – PHASE 2

EPA conducted its Direct Contact Risk Assessment in two phases. Phase 1 consisted of developing screening risk-based concentrations (SRBCs) for different types of usage, identifying individual exposure areas (EAs) along with their current and likely future uses, and then comparing the maximum or upper-bound PCB concentration in each EA to its applicable SRBC. This process was used to screen out certain EAs from the need for further evaluation. The EAs that were not screened out in Phase 1 were retained for the Phase 2 direct contact risk assessment. The following comments and their accompanying attachments are focused exclusively on the Phase 2 assessment.

3.1 Exposure Scenarios, Receptors, and Exposure Areas (Question B.1)

Consistent with EPA (1995) guidance, the HHRA states that the application of exposure scenarios to the 90 floodplain EAs and the subareas evaluated in the Phase 2 Direct Contact Assessment was based on "current and reasonably anticipated future land uses" (Vol. I, p. 4-5). EPA further recognizes, in the Peer Review Charge (Question B.1), that in evaluating this issue, it is appropriate to consider, in addition to such land uses, the physical conditions and accessibility of the areas, the locations and distribution of the chemicals of potential concern, and the ages of the selected receptors. GE believes that, for the majority of the EAs and subareas, EPA has appropriately considered these factors in assigning the applicable exposure scenarios and receptors. (This does not mean, however, that GE agrees with the exposure frequencies and durations assigned to these areas, which is separate issue discussed below.) For some EAs and subareas, however, GE believes that the HHRA has not properly considered the relevant factors, and as a result has assigned current-use scenarios and/or receptors that are not realistic or future use-scenarios that are not "reasonably anticipated."

<u>Current-use scenarios.</u> Attachment B to these comments lists the EAs and subareas where GE believes that the HHRA has incorrectly assigned exposure scenarios and/or receptors. As an example, the HHRA applies the bank angler scenario to several subareas (EAs 38A, 41A, 42A, and 43A) where there is no access for fishing due to the presence of dense vegetation and/or wetland areas (see, e.g., Figures 3 and 4 regarding EAs 38A and 43A), or where GE owns the property and does not allow access (EA 41). Indeed, during the intensive floodplain user survey conducted in 2002 (mentioned above and described in Attachment A), no anglers were observed in any of these areas despite the extensive observations. Hence, GE believes that

the angler scenario should not be evaluated for these areas. As another example, the HHRA assumes that young children (ages 1-6) would be recreating in certain undeveloped areas that have no designated access points and are covered with dense vegetation with no trails (e.g., EAs 70 and 87), as well as in certain other heavily vegetated areas (e.g., the off-trail portion of EA 10, as shown on Figure 1). It is unrealistic to assume that 1-6 year old children would spend any considerable time "bush whacking" through these areas.

<u>Future-use scenarios.</u> There are a number of EAs that are not currently residential properties but for which the HHRA evaluates a "future residential scenario" based on the assumption that future residential development is possible. For some of these areas, it seems clear that future residential use cannot be considered "reasonably anticipated" based on "realistic assumptions," as provided in EPA (1995) guidance. These include EA 6 (a portion of Miss Hall's School for Girls, which has been in operation since 1898), EA 78 (which consists of a retail store and two hotels), and EA 86 (a portion of the Stockbridge Golf Course). As discussed in Attachment B, it is highly unlikely that these parcels will be converted to residential property in the foreseeable future, and hence the future residential scenario should be dropped for these EAs.

<u>Sediment exposure scenario.</u> In addition to evaluating the floodplain EAs, the HHRA includes a river sediment exposure scenario. According to the HHRA, this scenario "was developed to evaluate sediment exposure from a variety of activities that could result in contact with sediment such as wading, swimming, fishing, waterfowl hunting, canoeing, and other related activities" (Vol. IIIA, p. 4-11). To evaluate this scenario, the entire upper reach of the river was divided into three distinct reaches: Confluence to New Lenox Road, New Lenox Road to the Woods Pond backwaters, and Woods Pond.

GE believes that the inclusion of this exposure scenario is unnecessary and redundant. The HHRA already includes the evaluation of more intensive activities that will occur in specific areas of these reaches, including canoeing, waterfowl hunting, and fishing, all of which may include wading in the river. In addition, any risks or hazards calculated for the sediment exposure scenario are not area-specific but instead apply to very large reaches of the river, making the information of little use to risk managers who will need to identify and characterize the "problem" areas associated with the river. Other information provided in the risk assessment is more meaningful. For example, the exposure point concentrations (EPCs) used for the waterfowl hunter are inclusive of sediment exposures in those areas and thus provide more meaningful and useful information for risk estimates.

GE recommends that the sediment exposure scenario be dropped from the HHRA as it provides no additional, meaningful information. In any event, these sediment exposures should not be added to the direct soil contact exposures that have been derived for canoeists, waterfowl hunters, anglers, etc. unless the soil and sediment exposure scenarios are modified (e.g., the total soil ingestion rate and contact rate are divided among the two pathways). If EPA is concerned that risks to these individuals occur as a result of exposure to both media and that this is not adequately considered in the activity-specific exposure scenarios, GE recommends that new EPCs, which are based on a combination of the data from the two media in each EA, be used to derive a single estimate of risk or hazard.

3.2 Calculation of Exposure Point Concentrations (Question B.3)

To derive exposure point concentrations (EPCs) for the Direct Contact Assessment, EPA calculated 95 percent upper confidence limits (95% UCLs) on the mean concentrations using one of three methods: 1) the Student's t-statistic if the underlying data were determined to be normally distributed; 2) Land's H-statistic if the data were determined to be lognormally distributed; and 3) Hall's bootstrap if goodness-of-fit testing indicated that data were neither normally nor lognormally distributed (Vol. I, p. 4-7; Vol. IIIA, pp. 4-17 - 4-21). If the 95% UCL for a given EA was greater than the maximum observed concentration, then the maximum concentration was used as the EPC. In all cases, the calculation of the EPC was based only on inclusion of sampling data that were contained within the 1 ppm PCB isopleth, even if the activities that occur within the EA are likely to occur both within and outside of the 1 ppm isopleth. GE has two main concerns about these approaches, as discussed below.

3.2.1 Use of the Land H-Statistic To Calculate 95% UCLs

In the HHRA, EPA has used the Land H-statistic to calculate the 95% UCLs for use as EPCs when goodness-of-fit testing indicates that the data are lognormally distributed. This approach has been used for the majority of the floodplain EAs downstream of Woods Pond Dam and some of the sediment EAs (see Vol. IIIA, Tables 4-3 & 4-4).

The validity of Land H-procedure is discussed in Attachment C to these comments. As noted there, EPA guidance (EPA, 2002a) suggests that the Land's H-statistic may be used to calculate the 95% UCL if the data are lognormal, but warns that this method may yield upper bounds that are much too large if the data are not truly lognormal. As also shown in Attachment C, it is widely recognized that when a data set departs even slightly from a true lognormal distribution, even when it tests as lognormal on statistical tests, the Land H-statistic can greatly overstate the true upper bound on the mean (Singh et al., 1997; Ginevan and Splitstone, 2002). Thus, these authors recommend against use of the Land H-statistic to calculate the 95% UCL for environmental data, which are seldom truly lognormal. Further, as explained by Singh et al. (1997) in an EPA technical report cited in EPA (2002a), even when such a data set is lognormal, the Land H-procedure can produce greatly overstated UCLs and thus should be avoided, especially if the sample size is small (i.e., less than 30). EPA (2002a) guidance reiterates this point, noting that when sample sizes are small (less than 30), the Land H-procedure is "impractical even when the underlying distribution is lognormal."

In the HHRA, all but one of the EAs for which EPA has used the Land H-statistic to calculate the 95% UCL have 13 or fewer data points, and in most cases they have less than 10. Indeed, given the overestimates in this procedure, EPA has defaulted to the use of the maximum concentration as the EPC for most of these EAs (see Vol. IIIA, Tables 4-3 & 4-4) – which cannot be considered representative, particularly given the sampling scheme that tended to bias the sampling locations to areas where concentrations were expected to be high. Consistent with EPA guidance (EPA, 2002a; Singh et al., 1997), the Land H-procedure should not be used to calculate the 95% UCL in these cases. By contrast, as also discussed in Attachment C, Hall's bootstrap procedure, which uses a transformation to correct for bias and skewness, appears to be fairly reliable regardless of the underlying distribution. Thus, GE believes that, in cases where the Land H-procedure was used, Hall's bootstrap should be used to derive the 95% UCL. This should result in more realistic upper bounds and may avoid the need to use the maximum concentration as the EPC.[2]

[2] In areas where the upper bound on the mean would still exceed the maximum, GE recommends that more sampling data be collected to allow more representative EPCs to be derived.

3.2.2 Limitation of EPC to Data from Within 1 ppm Isopleth

In developing the EPC for each EA, EPA has included only the data collected from those portions of the EA that fall within the 1 ppm isopleth. (For areas below Woods Pond, where the 1 ppm isopleth has not been defined, the EAs and EPCs are confined to sampling that has occurred within the 100-year floodplain.) This approach is reasonable for those areas or subareas where the activity being evaluated will actually be confined to the 1 ppm isopleth area (e.g., fishing on the river bank). However, for areas where the activity in question is likely to occur both inside and outside the 1 ppm isopleth, this approach will overstate exposures because it does not take into account the portion of soil contact that occurs in the areas outside the 1 ppm isopleth (with lower or non-detectable concentrations of PCBs). For example, at EA 29 (which is subject to the general recreational scenario), it appears that less than 10 percent of the parcel on which that EA is located is included within the 1 ppm isopleth and most of that area is classified as "difficult/wadable" (HHRA, Vol. IIIB, Figure 5-22). Thus, individuals using this parcel for recreation will likely spend the majority of their time in areas that are not within the 1 ppm isopleth. Similarly, none of the subareas subject to the dirt biking/ATVing scenario (EAs 22A, 27A, and 28A) can be accessed without following a trail that passes through other portions of the larger EAs and substantial areas outside the 1 ppm isopleth. Because dirt biking is a high-speed activity, it is likely that dirt bikers will pass through areas both inside and outside of the 1 ppm isopleth during their riding time and thus will contact soils from both types of areas.

This issue is discussed in more detail in Attachment D to these comments. As shown there, the HHRA's limitation of the areas for which EPCs are calculated to areas within the 1 ppm isopleth, without taking into account the portions outside the 1 ppm isopleth where the activities are equally likely to occur, is not consistent with EPA guidance for defining exposure areas (EPA, 1989a) and does not reflect actual exposure patterns. To address this issue and thereby avoid the overestimates associated with EPA's current approach, GE recommends that an adjustment factor be incorporated for the individual EAs to reflect the fraction of total exposure during an activity that is likely to be contained within the 1 ppm isopleth. Attachment D provides a table summarizing GE's recommended adjustments for the individual EAs.

3.3 Exposure Assumptions (Questions B.4 & B.5)

EPA has used a number of default and site-specific exposure assumptions in conducting its risk assessment for the direct contact pathways. While most of the central tendency estimate (CTE) values are reasonable, many of the reasonable maximum exposure (RME) exposure parameters and assumptions are unlikely to be reflective of actual or reasonably anticipated exposures. These parameters and assumptions are discussed below.

3.3.1 Defining Reasonable Maximum Exposure

Some conservatism is appropriate in the development of exposure assumptions to ensure that risks are not underestimated and that resulting remedial decisions are protective. However, the input parameters selected to evaluate exposures must be sufficiently realistic to represent plausible exposure scenarios. EPA (1992a) guidance recognizes this and defines the methodology for calculating "high-end" exposures, such as the RME. Specifically, it recommends that the risk assessor identify one or more of the most sensitive parameters, set them at their upper bounds, and then use average values for all other parameters to ensure that plausible exposures are not exceeded.

This approach, however, has generally not been followed in the HHRA. Instead, EPA has used upper-bound values for nearly all parameters, including not only the EPC and the assumption that all exposure occurs within the 1 ppm isopleth, but also (as discussed below) the exposure frequency, the exposure duration, the soil ingestion rates, the fraction of soil ingested that comes from the site, the exposed skin surface areas for dermal contact, and the dermal absorption factor for PCBs. This combination of upper-bound estimates for most parameters results in exposure estimates that cannot reasonably be expected to occur. For example, just the combination of two 95^{th} percentile values, which are each protective of all but five percent of the receptor population, yields a result that is protective of 99.75 percent of the population (0.05 * 0.05 = 0.0025 or 0.25 percent). When multiple upper-bound values are combined, as in the HHRA, the resulting estimates go well beyond plausible exposures and are not, therefore, consistent with the goals outlined in EPA (1992a) guidance or with the policy outlined by EPA headquarters (Browner, 1995), which states that core assumptions need to fall within a "zone of reasonableness" and should not be unrealistically conservative. GE's specific concerns and recommendations are discussed below.

3.3.2 RME Exposure Frequencies and Durations

In an effort to ensure that exposures are not underestimated in the direct contact risk assessment, EPA has selected a number of highly conservative estimates of exposure frequency and duration. In many cases, GE does not believe that these assumptions reflect likely levels of usage, either for a particular scenario or for the physical characteristics of the specific EAs being evaluated.

3.3.2.1 General recreation

For the general recreational scenario, the HHRA has assigned RME exposure frequencies of either 30 days/year or 90 days/year to the EAs subject to this scenario. For many floodplain areas, however, the assumed exposure frequency of 90 days/year, which translates to 3 days/week, every week, from April through October, is unrealistically high, given the physical constraints (e.g., extensive wetlands, vegetation, and topography) in such areas and the site-specific recreational Floodplain User Survey, discussed above, conducted by TER from April through October 2002. As discussed in more detail in Attachment A to these comments, the objective of that survey was to collect site-specific data on recreational use within the Housatonic River floodplain between the Confluence and Woods Pond Dam. During intensive observations of most of the recreational EAs in this stretch (nearly daily for many), surveyors collected information about the numbers of recreators in each EA and the types of activities in which they were engaged. The resulting data, summarized in Attachment A, demonstrate that many of the exposure frequencies assigned in the HHRA substantially overestimate current use. For example, there are 27 EAs for which the HHRA assigns an RME exposure frequency of 90 days/year for a single individual but at which the survey showed either no recreational users or six or fewer total recreational visits over the season, despite the extensive coverage of the survey. Clearly, if the frequency of usage envisioned by EPA in those EAs were occurring, the survey would have observed more usage.

GE recognizes that the HHRA needs to take into account reasonably anticipated future use as well as current use, and that future use could be somewhat higher than current use in some areas. However, it is not reasonable to anticipate that future use in areas with such low current use would rise to the level assumed in the HHRA (90 days/year), particularly for EAs that are remote and have difficult access due to the presence of steep slopes, wetlands, and/or dense vegetation. For example, EAs 13, 16, 17, 18, 19, 20, 21, 33, 35, 49, and 51 are isolated, remote areas that are not readily accessible (see, e.g., Figure 2 regarding EA 17), and EAs 10, 32, 41, 43, 49 have difficult access due to the presence of large wet areas. Given these characteristics, the level of usage is not likely to change considerably in the future.

Table 1 provides a summary comparison of the survey observations with the exposure scenarios and frequencies in the HHRA and identifies a number of EAs where GE believes that the frequencies and/or scenarios should be revised. Although a detailed report on this survey was submitted to EPA in January 2003, the HHRA states that EPA did not have time to consider it in its exposure estimates (Vol. IIIA, p. 4-9). Accordingly, EPA should now consider these site-specific empirical data, along with the physical characteristics of individual EAs, to derive revised recreational exposure frequencies for the EAs.

3.3.2.2 Dirt biker/ATVer

The HHRA uses an exposure frequency of 90 days/year and a duration of 12 years (ages 7 to 18) to evaluate potential risks to the dirt biker/ATVer, based on the assumption that an individual between the ages of 7 and 18 years participates in dirt-biking/ATVing 3 days/week, 7 months/year for 12 years (Vol. IIIA, p. 4-38). In addition, EPA's approach assumes that this activity is confined to very small subareas of each EA evaluated and that 100 percent of the soil contacted on each day of exposure is derived exclusively from that subarea.

First, the age group considered (ages 7 to 18 years) and duration of exposure are not realistic for the type and intensity of the scenario being evaluated. It is unlikely, due to safety concerns, that 7 to 9 year old children would be permitted to regularly drive a dirt bike or ATV. For this age group, the frequency would be considerably less than that being assumed for this scenario. In addition, while individuals aged 16 and older might occasionally ride a dirt bike or ATV, the availability of other activities for older adolescents and access to automobiles is likely to mean that they will not choose to continue to engage in dirt biking/ATVing with such a high frequency. GE recommends that a more reasonable age group of 10 to 16 years be used instead to evaluate this scenario.

Second, EPA has assumed an exposure frequency of 3 days/week for 7 months of the year. While it is possible that adolescents ride their bikes/ATVs in these EAs 3 days/week during their summer vacations, this frequency is not likely to occur during the remaining four months when these individuals are involved in school, sports, and other after-school activities. As an upper-bound estimate, it seems more reasonable, but still conservative, to assume 3 days/week from late June through August and 2 days/week for the remaining period for the RME analysis.

Table 1. Comparison of HRFUS Observations with Current Use Scenarios in HHRA

EA	Obs. Days	Obs. Visits	Observed Activities	Current Use in HHRA	HHRA Freq. (d/yr), RME/CTE	Comments
1	181	20	Walking, fishing, ATV/biking	Gen. Rec.	30/15	
2	181	3	Wild crop gathering	Gen. Rec.	30/15	
4	178	14	Walking, ATV, general recreation	Gen. Rec.	90/30	
5	181	1	Walking/hiking/running	Gen. Rec.	90/30	Freq. inconsistent w/ observations
6	60	0	None	Gen. Rec.	30/15	
7	181	2	General recreation	Gen. Rec.	90/30	Freq. inconsistent w/ observations
8	60	0	None	Canoeing	60/30	
9	60	0	None	Gen. Rec.	90/30	Freq. inconsistent w/ observations
10	60	0	None	Gen. Rec.	90/30	Freq. inconsistent w/ observations
11	181	0	None	Gen. Rec.	90/30	Freq. inconsistent w/ observations
12	181	49	Walking, ATV, gen. rec., farming	Gen. Rec.	90/30	
13	181	0	None	Gen. Rec.	90/30	Freq. inconsistent w/ observations
16	181	0	None	Gen. Rec.	90/30	Freq. inconsistent w/ observations
17	181	0	None	Gen. Rec.	90/30	Freq. inconsistent w/ observations
18	60	0	None	Rec./Res.	90/30	Freq. inconsistent w/ observations
19	181	0	None	Gen. Rec.	90/30	Freq. inconsistent w/ observations
20	181	2	Walking	Gen. Rec.	90/30	Freq. inconsistent w/ observations
21	60	0	None	Farming	10/10	
22	181	0	None	Rec./ATV	90/30	Freq. inconsistent w/ observations
23	178	1	General recreation	Gen. Rec.	90/30	Freq. inconsistent w/ observations
24	181	1	General recreation	Gen. Rec.	90/30	Freq. inconsistent w/ observations
25	60	1	Walking/hiking/running	Gen. Rec.	90/30	Freq. inconsistent w/ observations
26	182	45	Walking, ATV, general recreation, hunting, riding, farming	Gen. Rec. Farming	90/30 10/10	
27	60	0	None	Rec./ATV	90/30	Freq. inconsistent w/ observations
28	60	1	Walking/hiking/running	Rec./ATV	90/30	Freq. inconsistent w/ observations
29	60	0	None	Gen. Rec.	90/30	Freq. inconsistent w/ observations
30	60	0	None	Res.	90/30	Freq. inconsistent w/ observations
31	118	6	Walking, general recreation	Gen. Rec.	90/30	Freq. inconsistent w/ observations
32	118	4	Walking, general recreation	Gen. Rec.	90/30	Freq. inconsistent w/ observations
33	181	2	Hunting, general recreation	Gen. Rec.	90/30	Freq. inconsistent w/ observations
34	60	0	None	Farming	10/10	
35	181	2	Walking, bird watching	Gen. Rec.	90/30	Freq. inconsistent w/ observations
36	181	0	None	G. Keeper Farming	30/15 10/10	

EA	Obs. Days	Obs. Visits	Observed Activities	Current Use in HHRA	HHRA Freq. (d/yr), RME/CTE	Comments
37	182	36	Walking, hunting, riding, general recreation	Gen. Rec. Angler	90/30 60/20	
38	118	6	Walking, bird watching	Gen. Rec. Angler	90/30 60/20	Angler scenario inconsistent w/ obs. Freq. inconsistent w/ obs.
39	181	12	Walking, fishing, birding, hunting	M. canoe	150/90	
40	181	70	Walking, hunting, bow shooting, general recreation, fishing	Gen. Rec. Angler	90/30 60/20	
41	181	0	None	Gen. Rec. Angler	30/15 60/20	Angler scenario inconsistent w/ obs.
42	118	6	Walking, bird watching, general recreation	Gen. Rec. Angler	90/30 60/20	Angler scenario inconsistent w/ obs. Freq. inconsistent w/ obs.
43	60	1	Walking/hiking/running	Gen. Rec. Angler	90/30 60/20	Angler scenario inconsistent w/ obs. Freq. inconsistent w/obs.
44	60	1	Walking/hiking/running	Gen. Rec.	90/30	Freq. inconsistent w/ observations
45	118	11	Walking, general recreation	Hunting	48/16	
46	118	5	Hunting, general recreation	Hunting	48/16	Freq. inconsistent w/ observations
47	118	4	Walking, general recreation	Canoeing	60/30	
48	118	9	Walking, hunting, gen. rec.	Hunting	48/16	Freq. inconsistent w/ observations
49	60	0	None	Hunting	48/16	Freq. inconsistent w/ observations
50	60	0	None	Gen. Rec. Hunting	30/15 48/16	Freq. inconsistent w/ observations
51	60	0	None	Gen. Rec. Hunting	30/15 48/16	Freq. inconsistent w/ observations
52	118	3	Walking, fishing	Canoeing	60/30	
53	118	26	Walking, fishing, hunting, gen. rec.	Canoeing	60/30	
54	118	9	Walking, fishing, gen. rec.	Hunting	48/16	Freq. inconsistent w/ observations
55	118	28	Walking, picnicking, general recreation	Gen. Rec. Hunting	30/15 48/16	
56	118	2	Biking	Gen. Rec. Hunting	30/15 48/16	Freq. inconsistent w/ observations
57	118	31	Walking, fishing, birding, gen. rec.	Hunting	48/16	
58	118	25	Walking, fishing, ATV, gen, rec.	Angler	60/20	
59	118	34	Walking, fishing, hunting, bird watching, general recreation	Gen. Rec. Angler	90/30 60/20	
60	118	198	Walking, ATV, biking, fishing, bird watching, general recreation	Canoeing Gen. Rec.	60/30 90/30	

Source: Table 4, Attachment A
Abbreviations: Gen. Rec = general recreation, Res. = future residential, G. Keeping = groundskeeping, M. canoe = marathon canoeing

3.3.2.3 Bank fishing angler

In its direct contact pathway for anglers, the HHRA uses a generic RME exposure frequency of 60 days/year (Vol. IIIA, p. 4-44), which it indicates is based on data from a creel survey by ChemRisk (1994) of the Housatonic River. In that survey, individuals who were observed fishing in one of two reaches of the river (the area between Pittsfield and Woods Pond and the area from Woods Pond to the Connecticut border) were interviewed once about their fishing and fish consumption behaviors.

As discussed by Price et al. (1994) and EPA (1997a), anglers encountered during a creel survey are likely to be the most avid anglers who use the fishery most frequently, due to the fact that the most frequent anglers are most likely to be surveyed and the less frequent anglers will be under-represented in the survey data. Thus, the reported activities of the surveyed anglers will overestimate the activities of the majority of anglers who fish there. In fact, it is likely that the averages reported in that survey do not reflect the average behavior within the entire angler population that uses these portions of the river, but rather represent the average among the most highly exposed individuals. Thus, in estimating the RME, it is not appropriate to attempt to select an upper-bound value from the results of a survey that already represents the most highly exposed individuals and then to apply that value to a single section of riverbank.

Other relevant freshwater fish consumption survey data indicate that the RME frequency used in the HHRA is unlikely. According to angler survey data from Maine (ChemRisk, 1992), the 95th percentile fishing frequency among anglers who fished rivers was 30 days/year. For lake fishing, the 95th percentile was 40 days/year. Both of these estimates included fishing multiple locations and thus are likely to overestimate fishing frequency from a single waterbody. The survey for the West Branch Penobscot River (ChemRisk, 1991), which is an extremely popular landlocked salmon fishery in Maine, indicated that the 95th percentile trip frequency was 26 trips/year. GE recommends that EPA modify its generic exposure frequency to 30 days/year in order to reflect these less biased seasonal trip frequencies.

Moreover, GE does not believe that the generic exposure frequency should be applied to all EAs. Instead, area-specific frequencies should be selected based on the characteristics and accessibility of the particular EA for fishing. For example, while a frequency of 30 days per year may be reasonable for certain EAs (e.g., Woods Pond EAs 58 and 59), it is not reasonable for other areas, such as EAs 37A and 40A, where the dense vegetation along the banks during the late spring, summer, and early fall make such a frequency highly unlikely. Further, as discussed in Section 3.1, there are other EAs where GE believes that the bank angler scenario should not be applied at all.

The HHRA also uses an exposure duration of 60 years for the angler scenario, based on the 95th percentile of the lengths of time that respondents to an MADPH survey (MADPH, 2001) reported that they had consumed freshwater fish (Vol. IIIA, p. 4-45). Those data, however, do not support such a conclusion because, while individuals were asked how many years they had been consuming freshwater fish, they were never asked how many years they had been consuming either sport-caught freshwater fish or fish obtained from the Housatonic River or how long they had been fishing in the Housatonic River. It cannot be assumed that the number of years that an individual reported having eaten any type of commercial or sport-caught freshwater fish is the same as the number of years fishing or consuming fish from the Housatonic River. For example, an individual who had eaten freshwater fish from the grocery store or other sources for 60 years but had seldom, if ever, fished in or eaten fish from the Housatonic River would have reported 60 years as a response to the question. Consequently, these data are too unreliable to be used to estimate exposure duration for the bank angler scenario.

3.3.2.4 Waterfowl hunter

The exposure frequency and duration for the RME waterfowl hunter – 48 days/year for 58 years (Vol. IIIA, pp. 4-47 - 4-48) – are likewise unrealistically high. This scenario assumes that one begins hunting regularly at age 12, hunts for all species of birds available for migratory bird hunting (total season of 16 weeks), goes hunting 3 times/week every week during hunting season in the same locations, and continues this activity every year until age 70. In addition, unlike all other exposure scenarios, this scenario contains no provision for differences in exposure that are likely to occur as the weather gets colder. It is instead assumed that the same level of contact occurs in late November and December as occurs in early Fall, despite the fact that the floodplain is likely to be frozen and snow-covered and that hunters need to wear warm gloves and more clothing during the colder months. While some of these assumptions are possible, all of them combined are highly improbable and thus likely overestimate potential direct contact even for the most active waterfowl hunters.

The 48 day/year exposure frequency assumes that the individual hunter goes hunting 3 days/week in the same spot in the floodplain during every week of hunting season, thereby targeting all migratory birds in the area, including those that are not particularly desirable as food. This level of activity is likely to be rare due to the fact that most hunters will have jobs that will make it impossible for them to hunt more often than two days/week during the hunting season, and likely considerably less often than that since hunting is not allowed on Sundays (MDFW, 2002). While it is possible that someone who is unemployed or retired may hunt more frequently, this is very unlikely to occur with much frequency and certainly won't occur throughout a lifetime. In addition, since older children are in school during hunting season and evenings are dark, the only times that they would likely have the time and opportunity to hunt would be on Saturdays. In light of these factors, as well as the lower exposures during the winter months, it appears that an assumed exposure frequency of 30 days/year or less would be a more realistic and yet highly conservative estimate of upper-bound direct-contact exposure to waterfowl hunters.

The HHRA's assumed exposure duration of 58 years is based on information on residence time in the area that was collected as part of the MADPH (2001) survey. As such, it assumes that residence time is equivalent to the number of years during which bird hunting occurs. However, thus is likely not to be the case, as individuals may go waterfowl hunting sporadically throughout their residence time, and even the most avid hunters may have some years when they are unable to hunt.

3.3.2.5 Sediment exposure

The HHRA also includes a sediment exposure scenario, which assumes that exposure to sediments in the river occurs 3 days/week during the three summer months and continues for a duration of 64 years (Vol. IIIA, p. 4-49 - 4-50). As discussed in Section 3.1, GE believes that a separate evaluation of sediment exposure for large reaches of the river is unnecessary and redundant and should be eliminated. If it is retained, however, the exposure frequency and duration appear to unrealistically high for exposure to the river bottom sediments.

3.3.3 Soil Ingestion Rates

In characterizing RME exposure to floodplain soils, the HHRA generally uses upper-bound soil ingestion rates of 200 mg/day for young children and 100 mg/day for older children and adults, based on studies conducted prior to 1997 and discussed in EPA's *Exposure Factors Handbook* (EPA, 1997a) (HHRA, Vol. IIIA, p. 4-28). However, as discussed in more detail in Attachment E to these comments, improved, more recent studies of soil ingestion by both children and adults, which were conducted by the same investigators (Calabrese, Stanek, and colleagues) who conducted the studies on which EPA's upper-bound estimates are based, indicate that these daily soil ingestion rates are overestimated. Specifically, two recent studies published in the peer-reviewed literature by these investigators, using improved methodologies, indicate that more reasonable upper-bound soil ingestion rates should be around 100 mg/day for young children (based on Stanek and Calabrese, 2000) and about 50 mg/day for older children and adults (based on Stanek et al., 1997). Attachment E provides the detailed bases for these recommendations and includes a letter from Dr. Calabrese supporting these rates.[3]

In addition, in the RME evaluations for the recreational scenarios, the HHRA includes no adjustment to account for the fraction of total daily soil ingestion that comes from areas that are not in or near the floodplain (e.g., home, school, work, etc.) (Vol. IIIA, p. 4-25). For recreational activities that are relatively short in duration (so that the majority of soil ingested daily comes from other activities), such an adjustment needs to be made to reflect that fact that the total volume of soil ingested in a day will be derived from a combination of the floodplain areas and areas wholly unrelated to the floodplain that are contacted during each day of exposure. As also discussed in Attachment E, such an adjustment is supported by EPA (1989a) guidance and was previously made by EPA Region I, using a "fraction ingested" factor of 0.5, in developing its PCB cleanup standards for areas outside the river at the GE-Pittsfield/Housatonic River Site

[3] In addition, the HHRA uses "enhanced" soil ingestion rates of 200 mg/day for the farmer scenario and 330 g/day for the utility worker scenario. As discussed in Attachment E, a review of the available data on adult ingestion rates, as well as recent information on the adherence of soil to the hands of farmers and utility workers, indicates that these enhanced soil ingestion rates should be similar and should be reduced to 137 mg/day.

(see EPA, 1999a). GE believes that a similar adjustment should be made in evaluating the recreational scenarios in the Rest of River floodplain.[4]

3.3.4 Dermal Absorption of PCBs

To evaluate dermal exposures to PCBs, the HHRA uses an absorption factor of 14 percent, based on a study of rhesus monkeys by Wester et al. (1993) (see HHRA, Vol. I, p. 2-20; Vol. IIIA, p. 4-25). That study used soil with a large particle size and a very low organic carbon content (0.9 percent), neither of which is typical of floodplain soils, including those in the Housatonic River floodplain, and also used PCB mixtures (Aroclors 1242 and 1254) which are different from the predominant mixture found at this site (Aroclor 1260). As discussed in detail in Attachment F to these comments, based on both theoretical and empirical considerations, these factors very likely resulted in an overestimate of dermal absorption of PCBs compared to absorption that would occur with the smaller, higher organic-carbon content soils and PCB mixture found at this site.

Indeed, since the time that the Wester et al. (1993) study was published, an additional dermal absorption study has been reported (Mayes et al., 2002), which used the same general protocol as Wester et al. (1993) but used soil with more typical organic carbon content and particle size for Housatonic River floodplain soils and sediments and specifically evaluated dermal absorption of Aroclor 1260. That study, which is also discussed in Attachment F, reported a dermal absorption factor of around 4 percent. While the HHRA points out some limitations of the Mayes et al. (2002) study (Vol. I, p. 2-21; Vol. IIIA, p. 4-26), we have provided responses to those criticisms in Attachment F. In short, although both studies have limitations, GE believes that the Mayes et al. (2002) results are more representative of site-specific soils at this site and thus should be used as the basis for the dermal absorption factor in the HHRA.

---

[4] This adjustment is separate from the adjustment discussed in Section 3.2.2. That adjustment was intended to reflect the fact that, where the overall exposure area includes portions both within and outside the 1 ppm isopleth, a portion of the receptor's exposure *within that overall exposure area* in or near the floodplain will occur outside the 1 ppm isopleth. The adjustment being discussed here is intended to reflect the fact that, in most cases, a large portion of the receptors' daily soil intake will occur in areas that have nothing to do with the floodplain (i.e., home, school, work, etc.).

3.4 Uncertainty Analysis (Question B.6)

The HHRA generally recognizes many of the uncertainties associated with the Direct Contact Assessment (Vol. I, Sec. 4.6.2.2; Vol. IIIA, Sec. 7.2.2). In some cases, however, it does not appropriately recognize the implications of the uncertainties. For example, it states that the exposure frequencies and soil ingestion rates used may either over- or under-estimate risks, and it also states that the exposure durations used are likely to be reasonable (Vol. I, p. 4-27; Vol. IIIA, pp. 7-5 - 7-6). In fact, for the reasons discussed above, the exposure frequencies and durations used, as well as the soil ingestion rates (including the lack of an adjustment for the fraction ingested from the site), are far more likely to overestimate than to underestimate exposures and thus risks. In addition, the HHRA does not mention the uncertainties in the dermal absorption factor for PCBs, which, as also shown above, is likely to overstate dermal absorption from these floodplain soils. If these factors are not modified in the final HHRA, their tendency to overestimate exposures and risks should be directly acknowledged.

3.5 Overall Reasonableness of Approach (Question B.7)

Overall, as discussed above, the principal exposure input assumptions to the Direct Contact Assessment were all selected to be upper-bound values and in several cases go beyond realistic upper bounds and/or are not supported by more recent information. For example, for most of the EAs subject to the general recreational scenario, the HHRA assumes that the RME individual: (a) spends his/her entire time within the 1 ppm isopleth; (b) is exposed to the 95% UCL PCB concentration in that area (or the maximum measured concentration if lower); (c) recreates in this same area 3 days per week for 7 months per year for virtually his/her entire life; (d) ingests soil from that area at a rate higher than those shown by more recent studies and does not ingest soil from any other areas outside the floodplain during each day of exposure; and (e) gets soil on his/her hands, forearms, lower legs, feet, and head during each exposure event and absorbs the PCBs from that soil at a rate higher than shown by a study using the most representative soil type The combination of these parameter values results in exposure estimates that are not representative of potential exposures but are clearly overstated. As discussed above, this approach is not consistent with EPA (1992a) guidance for evaluating high-end exposures, which recommends use of upper-bound values for only a couple of parameters, with average values used for the rest.

GE recommends that the Direct Contact Assessment be revised to select a more realistic set of exposure parameter values, as discussed above, in order to ensure that, when taken as a whole, the combination of assumptions results in a more credible and representative estimate of exposures to individuals in these areas.

SECTION 4: FISH AND WATERFOWL CONSUMPTION ASSESSMENT

Key Points

- The HHRA's use of the Land H-statistic to calculate the EPCs for certain fish/duck tissue data sets can greatly overstate the true upper bound on the mean and thus should not be used, at least for the smaller data sets (< 30 samples).

- While GE supports the HHRA's use of the Maine angler survey by Ebert et al. (1993) as the basis for deriving fish consumption rates, EPA has not selected the most applicable set of fish consumption rates from that survey.

  ⇒ Its assumption that anglers do not share their catch with other household members is not reasonable and not consistent with the survey data.

  ⇒ EPA should not use the rates for "all waters" (which included multiple rivers and lakes) since they overestimate consumption from a single river reach. It should use the rates for either rivers or lakes, as appropriate.

- It is not reasonable for the HHRA to assume that all waterfowls consumed are local residents, since most resident ducks begin migration near the beginning of the hunting season and a large fraction of waterfowls shot and consumed are migrants. EPA should adjust either the EPC or the waterfowl consumption rate to account for migration.

- The HHRA should take account of PCB loss due to cooking in both the RME and CTE scenarios, since cooking loss is a well-documented parameter.

- The HHRA's probabilistic analyses do not make full use of the available data and use some inappropriate and unnecessarily inflated inputs, thus producing results that are not much different from the point estimate results. For example:

  ⇒ They use point estimate EPCs, rather than distributions of all fish/duck tissue data.

  ⇒ They not only use the same "all waters-no sharing" fish consumption rates used in the point estimate analyses, but further inflate those distributions to include hypothetical upper bounds, which are wholly unrealistic (e.g., more than 1,100 fish meals from the Housatonic per year).

  ⇒ They do not take adequate account of correlations among input parameters.

- Alternative Microexposure Event (MEE) analyses of fish consumption performed by AMEC with modifications to correct for the deficiencies in EPA's analyses show substantially lower predicted risks.

- Overall, due to the combination of upper-bound or unrealistically high exposure assumptions in the point estimate analyses and an inappropriate application of probabilistic modeling techniques, the HHRA overstates exposures and thus risks due to fish and waterfowl consumption.

SECTION 4: FISH AND WATERFOWL CONSUMPTION ASSESSMENT

The HHRA includes both deterministic analyses (using point estimates of the exposure parameters) and probabilistic analyses (using both simple and Microexposure Event Monte Carlo techniques) for the Fish and Waterfowl Consumption Assessment. GE has substantial concerns with the approaches used to conduct both of these types of analyses.

4.1 Calculation of EPC (Question C.1)

Use of Land H-statistic. To calculate the EPCs in fish and waterfowl tissue, the HHRA uses the 95% UCLs of the available fish and duck data, calculated using the same statistical techniques used in the Direct Contact Assessment – i.e., the Student t-test, the Land H-statistic, or Hall's bootstrap method, depending on whether the distribution of the underlying data was determined to be normal, lognormal, or neither (Vol. IV, pp. 4-18 - 4-20). The HHRA uses the Land H-statistic to calculate the EPCs for several fish tissue data sets (Vol. IV, Tables 2-18, 2-20, 2-21, 2-22) and the duck tissue data set (Vol. IV, Table 2-26). As noted in Section 3.2.1 above and discussed in detail in Attachment C, GE does not believe that the Land H-statistic should be used to calculate the 95% UCLs due to its marked sensitivity to slight deviations from lognormality in the underlying distribution and the fact that environmental data are seldom truly lognormal, even if they test lognormal on statistical tests. This is particularly true for sample sizes less than 30. Thus, at a minimum, EPA should re-calculate the 95% UCLs for the fish/duck data sets with less than 30 samples (i.e., the yellow perch data for Rising Pond and all the duck data) using Hall's bootstrap procedure instead of the Land H-procedure.

Failure to account for waterfowl migration. For the waterfowl consumption pathway, the EPC used was based exclusively on tissue data from resident ducks. This EPC is not representative of the tissue concentration of the waterfowl consumed by local hunters because many of those birds are migratory and have little potential for exposure. According to H. Heussman, wildlife biologist with MDFW (personal communication), it is unlikely that the waterfowl harvested in Berkshire County after the first week of waterfowl hunting season are resident birds because most of the resident birds begin migrating within a few days of the start of hunting season. During the remainder of the season, most of the birds that waterfowl hunters harvest are migratory birds. Thus, resident bird tissue concentrations are not representative of a large fraction of the birds that are harvested each year.

The HHRA acknowledges that a portion of the hunter's bag is likely to contain less contaminated non-resident ducks (Vol. I, p. 5-32). It supports its approach, however, by stating that, based on duck data from reference areas, ducks from other locations are also likely to contain elevated PCB concentrations, and that the EPC is also intended to account for consumption of resident Canada geese. GE believes that, even if elevated, the reference duck data should be used to evaluate a portion of the waterfowl consumed in the HHRA in order to reflect actual harvest. Further, while some Canada geese harvested are resident birds, a substantial number harvested by local hunters will be non-resident birds migrating through the area since goose hunting season coincides with their migration patterns.

GE believes that EPA should take account of migration in its evaluation of waterfowl consumption. To do so, the EPC can be adjusted to reflect the ratio of resident to non-resident birds. As reported by H. Heussman (personal communication), approximately 80 percent of the waterfowl harvested in Berkshire County are harvested during the first two weeks of hunting season. At the same time, most of the resident birds leave the area within the first week of hunting season. By combining these estimates, one can conclude that approximately 60 percent of the waterfowl harvested during the season (40 percent during the first two weeks and an additional 20 percent during the remainder of the season) are not resident birds. Thus, the EPC can be calculated as a weighted average by assuming that 40 percent of the birds harvested are resident birds and that the remaining 60 percent have PCB concentrations at background or reference area levels.

Alternatively, if EPA chooses to not make adjustments to the EPC to reflect the fraction of birds that are migratory, this adjustment can instead be made in developing the consumption rate estimates for hunters who eat waterfowl. This potential adjustment is discussed in Section 4.2.2 below.

4.2 Exposure Assumptions for Point Estimate Analyses (Questions C.2 & C.3)

The consumption advisories that are currently in place for both fish and ducks prevent current risks from such consumption. GE recognizes, however, that a baseline risk assessment needs to consider the potential risks in the absence of such advisories. Thus, it is appropriate to make assumptions about what level of consumption might occur if there were no advisories. At the same time, however, the assumptions about such consumption need to be plausible and reasonably representative of expected conditions along the river. While some of the assumptions used in the fish and waterfowl risk assessments are appropriate, others are not, as discussed below.

4.2.1 Fish Consumption Rates

The HHRA uses an RME fish consumption rate of 32 g/day (equivalent to 52 fish meals per year) for adults for the river sections in Massachusetts and for consumption of warm water fish in Connecticut (Vol. IV, Sec. 4.5.2.2). This value is based on the Ebert et al. (1993) survey data for state-wide fish consumption by Maine's freshwater recreational anglers and represents the 90th percentile consumption rate for fish consumed from "all waters" in the state combined, assuming that no sharing of the harvested fish among family members occurs. For consumption of trout in Connecticut, the HHRA uses a consumption rate of 14 g/day, based on the 90th percentile of the Ebert et al. (1993) values for fish consumed from "rivers and streams," again assuming no sharing. Small children (aged 1 to 6 years) were assumed to eat fish at rates that were one-half the adult fish consumption rates, based on an assumption of sharing. In addition, it was assumed that 100 percent of the fish consumed during each year were obtained from each individual reach of the Housatonic River.

GE supports the use of the Ebert et al. (1993) survey data as the basis for selecting conservative fish consumption rates for the HHRA. In using these data, however, even assuming the absence of fish consumption advisories, several of the assumptions made in the HHRA are unrealistically high and are not supported by either the Ebert et al. (1993) data or EPA (1989b) guidance for estimating fish consumption rates based on harvest data. These points are discussed in detail in Attachment G to these comments and are summarized below.[5]

4.2.1.1 Assumption of no sharing

The HHRA assumes that no sharing occurs – i.e., that the angler alone consumes every fish caught. In the Ebert et al. (1993) survey, however, the "no sharing" data provided were part of a sensitivity analysis to show the absolute upper bounds of consumption rates; they were not intended to be representative of actual exposure conditions. To the contrary, the anglers who provided the data clearly indicated, for the most part, that they shared the fish that came into their households with other fish consumers there. In addition, because of the way in which the

5 The lead author of the Ebert et al. (1993) study is the lead author of this section of the present comments and of Attachment G.

survey was designed, the survey respondents' consumption rates were based not only on the fish that they themselves had caught for consumption but also on the fish that other family members had brought into the household and shared with them, as well as fish that had been given to them by other individuals outside of the household. Thus, the assumption of no sharing is not consistent with the data and substantially overestimates consumption by individuals.

In addition, only 138 (14 percent) of the 1,007 fish consumers in that survey indicated that only one person consumed all of the harvested fish brought into the household (i.e., no sharing); and the fish consumption rates for those individuals were included in the general distribution of fish consumption rates reported by Ebert et al. (1993). Thus, that general fish consumption distribution already includes rates for individuals who did not share their fish as well as those who reported that they shared harvested fish with other individuals. As such, there is no justification for using the separate set of "no sharing" rates that were provided only as a sensitivity analysis

EPA's assumption of "no sharing" is also inconsistent with EPA guidance on assessing risks due to the consumption of fish and shellfish. According to EPA (1989b) guidance, when consumption estimates are derived based on fish harvest, as was done by Ebert et al. (1993), the average daily consumption rate should be derived by dividing the edible portion of the fish harvested by the number of people in that household (p. 56). This recommended approach is similar to but slightly less conservative than the approach used by Ebert et al. (1993), who based their consumption rates only on the number of individuals in the household who consumed freshwater fish.

Further, the HHRA's approach to sharing is internally inconsistent. Although the fish consumption rates for adults have been selected based on an assumption that fish are not shared within a household, the HHRA evaluates fish consumption by small children based on the assumption that the angler will share with them (Vol. I, p. 5-12). It is unreasonable to assume that the anglers will not share their catch except with small children.

4.2.1.2 Selection of "all waters" consumption rates

In addition, EPA's application of the "all waters" fish consumption rates from the Ebert et al. (1993) survey to the individual river reaches evaluated in the HHRA is not appropriate. These consumption rates represent total consumption by Maine's freshwater anglers and included fish caught in multiple rivers, streams, lakes and ponds in the state. The vast majority of anglers who participated in the survey fished from multiple waterbodies and waterbody types during the year. Thus, these consumption rates overestimate consumption from a single waterbody like the Housatonic River and certainly overestimate consumption from a single reach of the river.

GE recommends that, given the nature of the different reaches of the Housatonic River, different consumption rate distributions from the Maine angler survey should be used to evaluate them. Specifically, as discussed in Attachment G, GE recommends that the river/stream consumption rates from Ebert et al. (1993) be used for the river reaches with flowing water, and that the lake/pond consumption rates from that survey be used for the impoundments that have characteristics more similar to lakes and ponds. Regardless of the distribution selected, the rates used will be very protective for a single small fishery, given that they are derived from data provided by anglers who fished multiple fisheries during the year.[6]

4.2.2 Waterfowl Consumption Rates

The HHRA uses a waterfowl consumption rate of 5 g/day to represent RME consumption of resident waterfowl by adults (Vol. IV, p. 4-78). GE does not regard this consumption rate as unreasonable for waterfowl consumption generally. However, as discussed in Section 4.1, it is unreasonable to assume that all waterfowl consumed are resident birds that have been raised primarily on the Housatonic River, since a large portion of them will in fact be migrants from other areas. If this factor is not taken into account by adjusting the EPC (as discussed in Section 4.1), then the consumption rate should be adjusted to reflect the fraction of waterfowl consumed which are assumed to be local residents.

---

[6] In addition to these changes in the adult fish consumption rates, GE recommends that the fish consumption rate for children aged 1 to 6 years be changed from 50 percent of the adult rates to 40 percent of the adult rates, based on data provided by Rupp et al. (1980), for the reasons given in Attachment G to these comments.

4.2.3 Cooking Loss

While the HHRA incorporates cooking loss as a factor in the CTE analysis of fish consumption, it assumes no cooking loss in the RME analyses of either fish or waterfowl consumption, based on the assumption that individuals could use the pan drippings to make sauce (Vol. IV, pp. 4-48, 4-80). However, cooking loss of PCBs is a well-documented parameter, which is related directly to preparation method (Daubenmire, 1996; Puffer and Gossett, 1983; Skea et al., 1979; Smith, 1972; Smith et al., 1973; Wang and Harrad, 2000; Zabik et al., 1996). Although it is possible that some individuals may consume some of the pan drippings or use them to make sauce or gravy, it is highly unlikely that they will consume or use 100 percent of the pan drippings and will do so at every meal eaten. For example, in *The Joy of Cooking,* Rombauer and Becker (1975) offer recipes for fish preparation that generally do not incorporate pan drippings.[7] Similarly, when gravy is made for waterfowl consumption, most of the fat is separated off from the pan drippings to ensure that the gravy will not be greasy (Rombauer and Becker, 1975). Thus, it is not reasonable to assume that <u>no</u> cooking loss occurs. In addition, since cooking loss is a function of the cooking method rather than the consumption frequency, it would not be expected that consumption of the pan drippings is correlated with high fish or waterfowl consumption rates. Hence, a cooking loss factor should be incorporated into both the CTE and RME analyses.

Further, where the HHRA does consider cooking loss, it does not consider all of the cooking methods reported in the Maine angler survey (Ebert et al., 1993) and does not consider all of the relevant peer-reviewed publications on cooking loss. There are additional studies that have evaluated PCB losses after frying and baking fish with low lipid levels (Smith, 1972; Smith et al., 1973; Skea et al., 1981; and Puffer and Gossett, 1983). GE recommends that EPA expand its cooking loss factors to include all cooking methods reported by the survey respondents and to reflect all available, relevant data. In addition, according to the Maine angler survey data (which were provided to EPA), Maine anglers had specific cooking method preferences for certain

[7] For bland fish, Rombauer and Becker (1975) recommend making sauces, which generally do not incorporate pan drippings. For fish that have a strong flavor, it is recommended that the butter or cooking oil in which they are cooked be discarded. Even when use of pan drippings is suggested, they recommend that the fat in the pan be poured off and water added to the remaining solidified pan scrapings to make a sauce. Other popular preparations, such as grilling, preclude the use of pan drippings.

species of fish. For trout, bass, perch, bullhead, and sunfish, combined, Maine anglers reported the following preferences: broil/grill = 14 percent; bake = 17 percent; fry = 65 percent; boil/poach/soup = 3.4 percent; raw = 0.78 percent. These cooking preference factors can be used to weight the cooking loss factors to derive a point estimate for cooking loss.

4.2.4 Exposure Duration

The HHRA uses an RME exposure duration of 60 years to evaluate the fish and waterfowl consumption pathways (Vol. IV, pp. 4-56, 4-80). This estimate is the 95th percentile of data collected in the MADPH (2001) survey on the number of years that individuals reported eating freshwater fish. For the reasons discussed in Section 3.3.2.3, these survey data do not provide a reliable basis for deriving an exposure duration estimate for fish consumption from the Housatonic River because they relate to freshwater fish consumed from *any* source, including recreational or commercial sources that are not associated with the Housatonic River or any single waterbody. Thus, an individual who reported eating freshwater fish for a long time in that survey may have only done so from the Housatonic River for a small portion of that time. Moreover, the MADPH data on which the HHRA bases its estimate have nothing at all to do with waterfowl consumption.

4.3 Probabilistic Analyses of Fish and Waterfowl Consumption (Questions C.4 & C.5)

In addition to its point estimate analyses, the HHRA includes both a simple Monte Carlo Analysis (MCA) and a Microexposure Event (MEE) analysis for each of the fish and waterfowl consumption risk assessments. While EPA has undertaken these analyses in an effort to provide risk managers with a more complete evaluation of potential risks due to these exposure pathways, there are a number of problems with its approach. The net result is that the probabilistic analyses presented in the HHRA do not reflect the full range of the data or the relationships among the variables. As a result, the outputs from these analyses are not substantially different from, and do not represent an improvement over, the results of the point estimate analyses and thus do not provide risk managers with any more useful information. For example, for tPCBs in Reaches 5 and 6, the RME cancer risk estimate for fish consumption using the point estimate approach yielded a hypothetical cancer risk of 1E-02 while the MEE analysis yielded a risk estimate of 8E-03 (Vol. IV, Table 8-1). These risk estimates only differ by approximately 20 percent. The RME estimates for tPCBs for the waterfowl consumption scenario also only differ by 20 percent (Vol. IV, Table 8-1). Because probabilistic analyses can take into account the full distributions of values for the key input variables, rather than being limited to single point values, they would be expected to produce substantially more refined results than a point estimate analysis.

4.3.1 Probability Bounds Analysis (PBA)

The HHRA uses Probability Bounds Analysis (PBA) as a means of estimating the uncertainty around the input distributions used for these exposure scenarios. The PBA approach begins with the raw data, reduces it to summary statistics, makes assumptions about the shape of the underlying distributions, adds values to provide additional conservatism for certain distributions, and then develops a new data distribution. This is an artificial way to evaluate the uncertainty in the data. It requires that data be summarized and that assumptions (which cannot be verified) be made about the shape of the underlying data distribution. In addition, the selection of additional upper-bound estimates to ensure that all "possible" risks are evaluated is highly subjective and not based on actual data.

While the theory of PBA and similar statistical methods has been presented in the published literature, this is not a mainstream approach for risk assessment and is not discussed as a means of evaluating uncertainty in the Agency's probabilistic assessment guidance documents (EPA, 1999b, 2001a). In addition, this complicated and subjective approach is not necessary or warranted when raw data distributions can be used directly in a probabilistic model. GE believes that PBA is a poor substitute for a properly conducted MCA or MEE analysis in that it tends to over-predict exposures and does not adequately account for internal correlations between input parameters.

Thus, PBA is neither necessary nor appropriate when a Monte Carlo or MEE model is properly conducted. In such a model, uncertainties can be minimized by including all of the raw data in the input distributions so that the variability is captured and there is no need to make assumptions about the data. The full range of possible solutions to the exposure/risk equations will be captured in the output of the model, providing risk managers with a full range of risk and hazard estimates upon which to base remedial decisions. Accordingly, GE recommends that the PBA be discarded and the MEE approach be used, with the modifications discussed in the next section, to evaluate uncertainties in the risk estimates.

4.3.2 Evaluation of EPA's Monte Carlo and Microexposure Event Analyses

GE has serious reservations about the way in which EPA has conducted its MCA and MEE analyses for the fish and waterfowl consumption scenarios. MEE analysis models exposures as a series of separate exposure events for individuals, thereby allowing estimation of the variation in exposures for individuals in the exposed population. As such, MEE analysis is expected to produce different results from both a point estimate analysis and a simple MCA (EPA 2001a; Simon, 1999). The more refined and representative the model, the better the estimation of risks. While the calculated values from the less refined models, like the point estimate and Monte Carlo, will be found within the output distributions from the MEE, the model will provide a more robust representation of the range of potential exposure that will occur.

In its probabilistic analyses in the HHRA, EPA has, in many cases, reduced the available data down to summary statistics and has made assumptions about the distributions of the underlying data and the uncertainties around those data to develop its input distributions. In some cases, these distributions have been artificially "expanded" by the inclusion of non-empirically based data points. In addition, relationships among parameters have not been adequately addressed. All of these steps are unnecessary when robust data distributions are available for input parameters and correlations between parameters are understood. Further, the approach used introduces unnecessary conservatism into the input to the models, thereby biasing the model results. This is not consistent with EPA (2001a) guidance for conducting probabilistic models.

The principal limitations and shortcomings of the probabilistic models presented in the HHRA are discussed in detail in Attachment H to these comments and are summarized below. In addition, Exhibit H.1 to Attachment H provides an alternative MEE analysis that includes the recommended modifications discussed below and demonstrates the differences in results when such modifications are incorporated into the MEE.

4.3.2.1 Failure to use distributions for EPCs

Instead of using the full distributions of the actual data on concentrations in fish and duck tissue, EPA has chosen to derive single upper-bound concentrations, based on the 95% UCLs of the data, and use those concentrations as single-point inputs to the MCA and MEE models. There is no need to reduce the available sampling data to a single upper-bound estimate when full distributions of tissue concentrations are available. Use of the distributions themselves more closely approximates exposures that what would occur when individuals catch and consume fish or waterfowl, because they will catch fish or waterfowl that have a variety of tissue concentrations.

4.3.2.2 Selection of fish consumption rates

As with the point estimate analysis, EPA has used the Ebert et al. (1993) data from the Maine angler survey as the basis for the fish consumption rate distribution but has selected the "all waters" fish consumption rates and has assumed that no sharing of fish occurs. As shown Section 4.2.1 and Attachment G of these comments, use of the "all waters" fish consumption rate distribution and the assumption of "no sharing" are not reflective of actual conditions. The same applies to the probabilistic analyses.

4.3.2.3 Expansion of the distributions to include hypothetical upper bounds

In an effort to ensure that input parameter distributions do not exclude any possible values, EPA has intentionally incorporated additional levels of conservatism into the input distributions by expanding the actual distributions to include calculated upper probability bounds, well above the actual reported ranges. As discussed in Attachment H, this addition of hypothetical upper-bound values has been done for both the fish consumption rate distribution and the calculated distribution of waterfowl meal sizes without concern for reapportioning the shape of the input distributions. Thus, not only are these upper-bound values conjectural, but they are also given inappropriate weight in the distribution.

EPA's expansion of these distributions results in implausible estimates that are no longer based on the data. For example, for the distribution of fish consumption rates, EPA has established a hypothetical maximum fish meal frequency of 1,042 half-pound fish meals per year for 70 years (Vol. IV, p. 6-13), which would equate to 2.9 half-pound sport-caught fish meals from a single reach of the Housatonic River every day of the year. EPA has then further inflated its new distribution for fish consumption by an artificial uncertainty bound of 10 percent (Vol. IV, p. 6-14), thereby raising the hypothetical maximum by an additional 10 percent – i.e., to 1,146 fish meals per year, which equates to more than three half-pound fish meals (over 1.5 pounds of fish from the Housatonic River) every day of the year for 70 years. Such estimates are wholly implausible.

Similarly, EPA has expanded the distribution of waterfowl meal sizes, based apparently on its own extrapolations of empirical data. EPA selected a maximum waterfowl meal size of 675 g/meal (1.5 pounds of duck per meal), purportedly based on data for poultry meal sizes from Pao et al. (1982) as reported in the EPA (1997a) *Exposure Factors Handbook* (see HHRA, Vol. IV, p. 6-50). However, Table 11-23 of the *Exposure Factors Handbook*, which reports the findings of Pao et al., provides no maximum meal size. Instead, the 99th percentile meal size for poultry is reported to be 388 g/meal, while the average and 50th percentile meal sizes are 128 g/meal and 112 g/meal, respectively. Nevertheless, EPA has created a distribution for meal sizes that assumes a maximum value of 675 g/meal and a central estimate of 188 g/meal using its PBA approach. This distribution no longer resembles the empirical data reported by Pao et al. and demonstrates the degree to which EPA has added unwarranted conservatism to this input distribution for the waterfowl consumption risk assessment.

This approach is not consistent with Agency recommendations. EPA (1999b) has indicated that "one should strive primarily for accuracy and that ideally any adjustments that introduce 'conservatism' should be left to decision makers."[8]

4.3.2.4 Lack of adequate correlation among model inputs

The HHRA also assumes, in its MEE analysis, that all exposure events are independent of each other. However, as discussed in Attachment H, there are a number of exposure parameters that are interrelated. These relationships, which need to be incorporated into the MEE analysis, involve the following parameters:

- Fish consumption rates: While fish consumption rates may fluctuate somewhat from year to year, they are likely to remain similar over time.
- Body weights: Body weights are gender- and age-specific and thus will vary somewhat over each subsequent year of exposure modeled.

---

[8] The HHRA indicates concern that the MEE approach may underestimate risks because it may not provide an estimate of risk for an individual "who eats the maximum amount of the most contaminated fish and waterfowl at every meal for an entire lifetime" (Vol. IV, p. 6-8). GE believes that such an exposure estimate is implausible. In any event, a well-designed MEE analysis that includes all values in the input distributions and has an adequate number of iterations will, in fact, estimate risks to just such an individual. However, because the likelihood that such an exposure would actually occur is extremely small, it may take millions of iterations before such an individual is identified.

- Cooking losses: Cooking losses are a function of the cooking method use to prepare the fish. In turn, the method used to prepare the fish is correlated with the species of the fish consumed. These factors need to be considered in selecting cooking loss factors.

4.3.2.5 Development of exposure duration estimate based on MADPH survey data

EPA's current MCA and MEE models use an exposure duration distribution that is based on MADPH survey data concerning the number of years that survey respondents reported eating freshwater fish from *any* source (Vol. IV, pp. 6-22, 4-56). As noted previously, the responses to this question do not provide reliable information regarding the length of time that an individual may consume sport-caught fish or game from the Housatonic River.[9]

4.3.2.6 Failure to take account of uncertainty in toxicity values

In addition to the above limitations in EPA's probabilistic analyses, which relate to the exposure estimates, there is an additional, and potentially greater, source of uncertainty in the risk assessment that is not addressed in EPA's probabilistic analyses – i.e., the uncertainty associated with the dose-response values used to estimate risks. These dose-response values are based on studies of laboratory animals and are extrapolated to predict human toxic response using numerous assumptions and uncertainty factors, all of which have considerable uncertainty associated with them. As discussed in more detail in Attachment H, the magnitude of the uncertainty around the toxicity values can be characterized in a probabilistic analysis by replacing point estimate uncertainty factors with distributions, as outlined by Swartout et al. (1998). While GE recognizes that current EPA guidance for probabilistic risk assessments (EPA, 2001a) does not provide for the use of distributions of toxicity values in human health risk assessments, GE believes that the probabilistic analyses presented in the HHRA would be greatly improved if they included, at least as a sensitivity analysis, a quantitative evaluation of the uncertainties associated with the selected dose-response values. Such consideration is consistent with the recommendations of EPA's Science Advisory Panel (SAP) under FIFRA which, in its evaluation of aggregate risks for pesticides, called for "a more quantitative risk

---

[9] It is possible that some data on this issue may be obtained from fishing or hunting license information or other state recreational information sources. In the absence of such information, it would seem more relevant, but still very conservative, to assume that individuals who catch or shoot and consume fish or game from the Housatonic River may do so during each year that they live near the Housatonic River. Exposure durations could thus be estimated using census data for the appropriate counties, taking into consideration population mobility and mortality rates.

assessment approach in which all of the safety factors are replaced by distributions based on the best available data from well studied cases" (EPA, 1999c, p. 37).

4.3.3 Alternative MEE Analysis of Fish Consumption

To quantify the impact of the issues discussed above, AMEC has performed an alternative MEE analysis of potential risks due to the consumption of fish from each reach of the Housatonic River. This analysis is summarized in Attachment H and presented in detail in Exhibit H.1 to Attachment H. This alternative MEE uses all of the raw tPCB data from each river reach, the consumption rate distributions reported by Ebert et al. (1993) for each waterbody type (with no artificial expansion of those distributions), expanded cooking loss factors to include all cooking methods reported by Maine anglers, correlations to account for inter-dependencies in variables (i.e., correlations of cooking methods to species consumed and year-to-year correlations of fish consumption rates and body weights), and local census data on age- and gender-specific mortality and mobility rates to estimate exposure duration. In addition, this alternative analysis presents the calculated risks both using EPA's single-point toxicity values for cancer and non-cancer (AMEC MEE 1) and, as a further sensitivity analysis, using a distribution of toxicity values (AMEC MEE 2).

As shown in Attachment H, the results of this alternative analysis indicate that incorporation of the changes discussed above results in cancer risks and non-cancer hazards that are substantially lower than the risk estimates presented in the HHRA for the corresponding reaches. Comparisons of the CTE (50^{th} percentile) and RME (95^{th} percentile) cancer risks and non-cancer hazards derived by EPA using its MEE model and those derived by AMEC using its alternative MEE 1 and MEE 2 models are presented in Table 2.[10]

GE requests that EPA have both the probabilistic analyses contained in the HHRA and the alternative MEE analyses presented in Exhibit H.1 reviewed by scientists within the Agency who are experts in probabilistic modeling techniques. GE further recommends that the Monte Carlo and MEE analyses presented in the HHRA be revised to incorporate the modifications described above. (Although the alternative analysis provided by AMEC is limited to the fish

[10] GE notes that since these alternative MEE analyses still use animal-based toxicity values and conservative exposure parameter distributions, they are still likely to overestimate actual risks to humans in the Housatonic River.

Table 2. Comparison of tPCB Cancer and Non-Cancer Risk Estimates: EPA's MEE Analysis vs. AMEC's Analyses

River Reach	Cancer Risks Adult/Child					
	50th %ile			95th %ile		
	EPA HHRA	AMEC MEE		EPA HHRA	AMEC MEE	
	MEE	MEE 1	MEE 2	MEE	MEE 1	MEE 2
5 to 6	2E-03	5E-05	2E-05	8E-03	8E-04	4E-04
8	2E-03	4E-05	1E-05	6E-03	6E-04	2E-04
11 to 12 (trout)	3E-04	9E-06	3E-06	1E-03	2E-04	6E-05
11 to 12 (bass)	2E-04	5E-06	2E-06	7E-04	8E-05	3E-05
14 to 15	1E-04	5E-06	2E-06	5E-04	8E-05	3E-05

River Reach	Non-Cancer Hazard Indices											
	Adult						Child					
	50th %ile			95th %ile			50th %ile			95th %ile		
	EPA HHRA	AMEC MEE		EPA HHRA	AMEC MEE		EPA HHRA	AMEC MEE		EPA HHRA	AMEC MEE	
	MEE	MEE 1	MEE 2	MEE	MEE 1	MEE 2	MEE	MEE 1	MEE 2	MEE	MEE 1	MEE 2
5 to 6	40	5.4	0.42	500	69	7.4	91	3.1	0.33	1000	49	6.6
8	29	4.3	0.33	330	44	4.6	61	2.7	0.28	720	28	3.9
11 to 12 (trout)	3.7	0.95	0.07	64	12	1.3	7.9	0.65	0.07	110	7.8	1.1
11 to 12 (bass)	3.7	0.50	0.04	44	6.2	0.65	7.7	0.36	0.04	91	4.0	0.57
14 to 15	2.4	0.56	0.04	31	5.8	0.60	5.3	0.35	0.04	61	3.6	0.50

consumption pathway as an illustration, GE believes that similar modifications should be made to the MEE for waterfowl consumption.)

4.4 Evaluation of Uncertainties (Questions C.6 and C.7)

The HHRA contains a discussion of the uncertainties in its Fish and Waterfowl Consumption Assessment (Vol. I, Sec. 5.7; Vol. IV., Sec. 7.2). The uncertainties relating to the toxicity assessment, including the Agency's discussion of those uncertainties, are addressed in Section 6.1.5 of these comments. With respect to the exposure assumptions in the RME point estimate analyses, the HHRA does not adequately highlight a number of key factors that have likely resulted in overestimates of exposure and thus risk. These include:

- The use of the 95% UCLs of fish and duck tissue concentrations as representative of PCBs in all fish and waterfowl meals consumed;
- The assumption that all waterfowl consumed consist of resident birds, with no adjustment for migration;
- The use of an upper bound of the fish consumption rates calculated for multiple waterbodies and waterbody types (although the HHRA states that the use of these data may under- or overestimate exposures to anglers, it seems clear that these data will not underestimate exposures for fish consumption from a single reach of the Housatonic River, for the reasons discussed previously);
- The assumption that all fish are consumed only by the angler, with no sharing, despite the fact that most survey respondents in the underlying survey indicated that they shared the fish that they harvested;
- The assumption that consumers eat 100 percent of the pan drippings at every meal so that there is no loss due to cooking;
- The assumption that 100 percent of the sport-caught freshwater fish consumed are from a single section of the Housatonic River; and
- The assumption that this level of consumption will occur every year for 60 years.

To the extent that these assumptions are not modified in the final HHRA, that document should acknowledge and discuss more explicitly their tendency to overestimate risks.

With respect to the quantitative treatment of uncertainty, the HHRA notes that its PBA approach "propagates both variability and uncertainty in the risk assessment" and thus complements the MCA and MEE analyses by "allowing for a comprehensive treatment of the effects of uncertainty" (Vol. I, p. 5-35; Vol. IV, p. 7-15). As discussed above, however, the PBA is neither necessary nor warranted in a well-designed MEE model. Moreover, as discussed in Section 4.3, the MCA and MEE models contained in the HHRA do not fully account for the variability and uncertainty in the inputs and thus do not represent a significant improvement over the point estimate analyses in terms of providing risk managers with a more complete picture of potential risks due to fish and waterfowl consumption.

4.5 Overall Reasonableness of Approach (Question C.8)

Overall, due to the combination of numerous upper-bound and unrealistically high exposure assumptions and parameters, discussed in Sections 4.1 and 4.2 and listed above in Section 4.4, the HHRA's point estimate results substantially overstate potential exposures and thus risks to fish and waterfowl consumers in the Rest of River area. That approach is not consistent with EPA's (1992a) recommended approach for evaluating high-end exposures, which recommends a combination of upper bound and mid-range inputs. Further, as discussed in Section 4.3, EPA's probabilistic analyses do not cure these problems since they use some of the same assumptions, do not make full use of the available data, do not adequately address relationships among parameters, and use inflated distributions with hypothetical maximum values that go far beyond realistic. GE recommends that the HHRA be revised to incorporate the modifications suggested herein for both the point estimate and the probabilistic analyses of fish and waterfowl consumption.

SECTION 5: AGRICULTURAL PRODUCTS CONSUMPTION ASSESSMENT

Key Points

- In providing risk estimates for two example PCB concentrations – 0.5 and 2 ppm – the HHRA assumes that 100% of the pasture and cultivated areas are within the 1 ppm isopleth. This assumption does not apply to any of the farms in the floodplain.

  ⇒ While the HHRA explains briefly how to adjust for this so as to apply its calculations to areas where only a portion of the farm land is within the 1 ppm isopleth, that adjustment does not address the lack of steady-state PCB conditions in the farm animals and does not adequately address the resulting overestimates in risks.

  ⇒ EPA should use a more realistic example – e.g., assuming that only 15% of pasture/cultivated lands are in the 1 ppm isopleth – and explain how to adjust those results based on the actual portion in the floodplain at a given property.

- The HHRA's apparent assumption that the agricultural scenarios could apply, as future use, to virtually the entire floodplain in Massachusetts is not realistic due to the unlikelihood of future farm development and legal restrictions on such development.

- EPA did not collect any site data on animal products. Instead, the HHRA uses models to estimate the uptake of chemicals of concern from soil to plants (grass, corn) and from soil and plants to animal products, and then from animal products to human consumers. Many of these modeled estimates are highly uncertain and likely overstated. For example:

  ⇒ The HHRA's soil-to-grass transfer factors are based on data from a one-time sampling event, which was conducted during optimum conditions for uptake (e.g., hot and dry) and thus did not take account of other factors affecting uptake (e.g., meteorological conditions). Literature data indicate that these factors are overstated.

  ⇒ Since PCBs were not detected in corn ears, soil-to-corn transfer factors are based only on limited and uncertain data from corn stalks. These factors are unreliable and overestimate PCB transfer to corn.

  ⇒ The HHRA's assumption that the farm animals are at steady state with chemical concentrations in the soil is not valid since the animals would from day to day graze or eat food from different areas, much of it outside the floodplain. This assumption likely overstates bioaccumulation.

  ⇒ The HHRA's assumption that the chemicals in ingested material are 100% bioavailable is not supported by the literature.

  ⇒ The HHRA's use of the maximum bioconcentration factors (BCFs) from the literature for PCBs in milk and body fat is not appropriate.

- The net result of using these multiple layers of modeled assumptions, many of which are very uncertain and likely overstated, is a set of exposure and risk estimates that are both unreliable and almost certainly overestimated.

SECTION 5: AGRICULTURAL PRODUCTS CONSUMPTION ASSESSMENT

The Agricultural Products Consumption Assessment is unique among the various pathways evaluated in the HHRA. First, unlike the other pathway assessments, this assessment does not evaluate specific areas or actual data from the site. Rather, it evaluates two pre-determined tPCB concentrations in floodplain soil – i.e., 0.5 ppm and 2 ppm – along with the concentrations of other COPCs (namely, dioxin-like PCB congeners, PCDDs, and PCDFs) determined by regression analyses to be associated with those pre-determined tPCB concentrations. In addition, the Agricultural Products Consumption Assessment relies almost entirely on modeling. Modeling was used to predict the transfer of COPCs from the soil to plant matter, from both soil and plants to animal products, and from plant and animal products to the human consumers. Very little empirical sampling data were available to support these modeling steps, and no site-specific sampling data were collected on the bioaccumulation of COPCs in the animal products themselves. As a result, the modeling was based on a series of assumptions selected from values published in the scientific literature or derived or extrapolated from limited analytical data on soils and plants. Due to these multiple levels of estimated and modeled exposure variables, the calculated risks and hazards for this exposure pathway are highly uncertain and are unreliable for risk management decision-making. The most significant assumptions and exposure models are discussed below.

5.1 Agricultural Exposure Scenarios (Question D.1)

GE has two principal concerns with the agricultural exposure scenarios evaluated, both of which relate to their applicability to the floodplain. These concerns pertain to: (1) the HHRA's assumption that all pasture and cultivation areas are within the 1 ppm tPCB isopleth; and (2) the apparent assumption that these agricultural scenarios could apply to large stretches of the floodplain that are not currently in agricultural use.

5.1.1 Assumption That All Pasture/Cultivation Areas Are Within 1 ppm Isopleth

All exposure and risk calculations in the HHRA for the Agricultural Products Consumption Assessment are based on the assumption that 100 percent of the pasture or cultivation areas are within the 1 ppm tPCB isopleth (Vol. I, p. 4-1). In fact, as illustrated in Figures 2-1a, 2-1b, and 2-1c (in Volume V), in areas where the 1 ppm isopleth has been determined, only small portions of the areas designated as agricultural actually fall within the 1 ppm isopleth. In apparent recognition of this, the HHRA contains a brief discussion of how its assumption can be applied to “actual exposure conditions” where all of the animals’ food source does not originate from within the 1 ppm isopleth (Vol. V, pp. 4-1 - 4-2; see also Vol. I, p. 6-4). It suggests, for example, that the results obtained by assuming that 100 percent of the cultivated land has a soil concentration of 0.5 ppm would likewise apply to a property that has 10 percent of the cultivated land at 5 ppm and 90 percent with no PCBs.

There are at least two problems with this approach. First, since the HHRA’s assumption of 100 percent of the agricultural land in the 1 ppm isopleth does not actually apply to any known agricultural properties in the floodplain, the resulting risk estimates (which don’t apply to any actual properties) tend to misinform readers of the risks from consuming locally raised agricultural products. Second, this approach does not address the fact that the HHRA’s bioaccumulation modeling assumes that the farm animals are at steady-state conditions with COPCs in the soil. As discussed further below, that assumption is not consistent with site conditions, especially if a portion of the animals’ diet comes from outside the 1 ppm isopleth; and the HHRA’s suggested adjustment procedure does not solve this problem.

In these circumstances, while GE does not disagree with the approach of using pre-determined example soil concentrations, it recommends that the HHRA use a more realistic example of portion in the floodplain for its basic calculations. For instance, it could assume that 15 percent of pasture/cultivation areas lie within the 1 ppm isopleth (which, according to the HHRA, is the average in Reach 5 [Vol. V., p. 5-10]), and then provide clear guidance for how to adjust from those results to reflect actual exposure conditions at a given farm property.

5.1.2 Applicability for Future Use

Although the HHRA is not entirely clear on this, it appears to suggest (Vol. V, Table 2-1) that the agricultural consumption scenarios could apply, as future use, to all of Reaches 5, 7, and 9 – which amount to essentially the entire floodplain in Massachusetts. Such an assumption may not have been intended and clearly could not be supported. The HHRA itself recognizes that “commercial agriculture appears to be on the decline in this area” (Vol. I, p. 6-1). Further, the floodplain contains large areas of State-owned properties, for which the State has agreed to deed restrictions locking in current uses if necessary (see Vol. IIA, p. 7-4; Vol. IIIA, p. 4-8), as well as many other areas that are unsuitable as future agricultural land due to extensive wetland areas. In addition, under the Massachusetts Wetland Protection Act (as amended by the Rivers Protection Act), and its implementing regulations, there is a statutory limitation on farming activities in riverfront areas, which (subject to specific exemptions) extend 200 feet from each riverbank. In these circumstances, the HHRA should be revised to make clear that future agricultural use is not expected in most of the floodplain in Massachusetts.

5.2 Approaches Used To Estimate Transfer of COPCs from Soil to Plants (Question D.2)

In estimating the transfer of COPCs from soil to plants, EPA has had to combine a number of theoretical approaches that introduce substantial levels of uncertainty into the calculations. These include both the estimation of soil concentrations for COPCs other than tPCBs and the calculation of soil-to-grass and soil-to-corn transfer factors. These issues are discussed in detail in Attachment I, with summaries provided below.

5.2.1 Methodology for Predicting Concentrations of COPCs in Soil

In addition to evaluating risks associated with exposures to tPCBs, the HHRA has used the dioxin toxic equivalency (TEQ) approach to evaluate dioxin-like PCBs and PCDDs/PCDFs in floodplain soil. To perform this phase of the assessment, the HHRA converted tPCB concentrations of 0.5 and 2 ppm to concentrations of dioxin-like PCB congeners and PCDDs/PCDFs based on regression models developed from supplemental soil data (Tables 2 and 3 of Attachment 2 of HHRA). The data used to establish these regression models were very limited, and therefore the outputs are unreliable. For example, for PCB-126, the regression model was limited to only 19 soil samples, and the average tPCB concentration in that sample set was 11.5 ppm, which is much higher than the pre-determined tPCB concentrations to which the models were applied. Thus, the predicted concentrations could not be verified for the concentrations of interest. This uncertainty has a substantial impact on the risk estimates since PCB-126 is the congener with the highest toxic equivalency factor and the one that accounts for 54 percent of the total TEQ in the extrapolation model. This creates one of the most significant uncertainties in the Agricultural Products Consumption Assessment. Moreover, as discussed further below, GE does not believe that EPA should use the TEQ approach for PCBs at this time. Thus, this conversion should be dropped.

5.2.2 Soil-to-Grass Transfer Factors

These factors were based on single sampling event and only a few samples (n=10) collected in Reach 5. These data are too limited to derive reliable transfer factors. The samples used to establish the transfer factors were collected: 1) during the warmer months of the year; 2) during a period with no heavy rain; 3) from areas adjacent to the river channel where recent inundation with floodwaters was evident; and 4) from areas with high levels of tPCBs (Vol. V, p. 2-16). All of these factors could lead to an overestimation of soil-to-grass transfer relative to other areas of the floodplain and other times of the year. In fact, as shown in Attachment I, other published data (Chaney et al., 1996) show much lower transfer factors for forage and grain crops than those used in the HHRA. In addition, the HHRA assumes a linear relationship between soil concentrations and predicted plant concentrations, even though the data do not support this assumption for either tPCBs or PCB congeners and in fact, for some congeners, show a negative correlation (Vol. V, Fig. 4-4a). Since this is such an important component of the overall exposure model, as it is used to estimate intakes of COPCs by the farm animals, GE recommends that additional data be collected to reduce the uncertainty associated with the current soil-to-grass transfer factors used in the HHRA.

5.2.3 Soil-to-Corn Transfer Factors

Similarly, the HHRA attempted to estimate COPC concentrations in corn silage using the limited data collected from the floodplain. In this case, however, the data showed no detected tPCBs in corn ears and the concentrations of tPCBs in the stalks of the corn plant were either undetected (5 samples) or estimated values (5 samples with a J qualifier) (Vol. V, Table 2-4). EPA based its soil-to-corn transfer factor for tPCBs only on the five corn stalk samples with detected but estimated tPCB concentrations. The HHRA itself recognizes that this procedure “likely overestimates PCB transfer to corn” (Vol. V, p. 4-24). Moreover, EPA guidance indicates that where, as in this case, there are *no* samples in a data set with unqualified measurable concentrations, the data set should not be used for quantitative risk assessment (EPA, 1989a, p. 5-11). In addition, since no data were collected on PCB congeners in corn, soil-to corn transfer factors for PCB congeners were based on the soil-to-grass transfer factors, adjusted to account for the lower transfer observed in the limited tPCB soil-to-corn data (Vol. V, p. 4-24). These practices result in highly uncertain and overestimated transfer factors. In fact, other data in the literature indicate that PCBs are not translocated from soil to corn (e.g., Gan and Berthouex, 1994; Webber et al., 1994; O’Connor et al., 1990). In these circumstances, given the absence of data to verify and allow a reliable estimate of the accumulation of PCBs in corn silage, GE believes that this route of exposure should be dropped.

5.3 Approaches Used To Estimate Bioaccumulation in Animal Tissue (Question D.3)

While limited soil and grass data were obtained to develop transfer factors, EPA did not sample agricultural animals or animal products raised on the floodplain or conduct controlled studies to obtain similar information from animals that could be used as surrogates for the conditions at the site. In the absence of such empirical data, the tissue concentrations of the COPCs in agricultural animal products had to be estimated, and the HHRA relied on modeling assumptions to derive these important levels. The most significant of these modeling assumptions are discussed in Attachment I and summarized below.

5.3.1 Assumption of Steady-State Conditions in the Animals

An most important assumption applied in the bioaccumulation model is that the dairy and beef cattle and chickens are at steady state with COPCs in floodplain soil – i.e., that the intake rate of COPCs by an animal is constant and equals the rate of elimination of these compounds (Vol. V, p. 4-5). This assumption is not consistent with existing conditions in the floodplain. Steady-state conditions require that concentrations in all of the soil, grass, and corn silage are constant and reflect soil concentrations of either 0.5 ppm or 2 ppm tPCBs, which, of course, is not the case. Moreover, since not all pasture and cultivation lands are within the 1 ppm isopleth, the animals' food sources will include some from within that isopleth and some from outside it, with the proportions varying from day to day. This fact will preclude the animals from being at steady state. As previously discussed, the HHRA does contain a brief discussion of how its assumption that 100 percent of the agricultural land is within the 1 ppm isopleth can be adjusted to apply to areas where only a portion of the agricultural land is within that isopleth. However, this does not rectify the flaw in the steady-state assumption. As discussed in Attachment I, the suggestion that 100 percent cultivated land at 0.5 ppm is equivalent to 10 percent at 5 ppm and 90 percent with no PCBs is not correct in terms of dictating body burdens because it does not take account of kinetic parameters. For example, the animals' consumption of feed from a 5 ppm area on one day and feed from a non-detect area on 9 other days would not result in an equivalent body burden as constant consumption of feed from a 0.5 pm area.

Hence, EPA should develop a non-steady-state model or at least develop an adjustment factor to account for limited contribution of floodplain soils to diet. In the absence of such a revision, the risk estimates for this pathway will necessarily be overstated.

5.3.2 Assumption of Complete Absorption of COPCs in the Ingested Material

The HHRA explicitly assumes that the COPCs in the material ingested by the animals have a bioavailability of 100 percent (Vol. V, p. 4-9). This is not supported by data available either in the scientific literature or in the HHRA, both of which report bioavailability percentages less than 100 percent for tPCBs, PCB congeners, and PCDDs/PCDFs, as shown in Attachment I. For example, Table 4-4b in Volume V of the HHRA lists "predicted absorption" values for the dioxin-like PCB congeners ranging from 41 to 71 percent. Although specific data for some congeners may not be available, information on structurally similar congeners can be used to develop appropriate estimates of bioavailability. GE believes that the HHRA should be revised to incorporate appropriate lower bioavailability factors based on Table 4-4b for PCB congeners and on the scientific literature for tPCBs and PCDDs/PCDFs.

5.3.3 Bioconcentration Factors (BCFs) for Milk and Body Fat

Once the amount of ingested COPCs was estimated, the distribution into tissue (including milk and beef fat) was determined using bioconcentration factors (BCFs). The BCF selected for tPCBs was the maximum value reported in the scientific literature (Vol. V, p. 4-14). Similarly, upper-bound values were used for PCB congeners (Vol. V, pp. 4-15 - 4-18). The selection of these maximum or upper-bound values results in the maximum or near-maximum predicted tissue concentrations. The HHRA itself acknowledges that BCFs for both PCB mixtures and PCB congeners might be significantly lower, and it provides a sensitivity analysis with those lower values (Vol. V, p. 5-11). Since many of the other model parameters and exposure variables used to estimate human exposure via this pathway are upper-bound estimates, use of the upper-bound estimates for the BCFs as well is not consistent with EPA (1992a) guidance. Use of the same BCFs used in the sensitivity analysis would provide a more reasonable and objective approach.

5.4 Uncertainty Analysis (Question D.7)[11]

The previous sections have highlighted the uncertainty in several modeling and exposure assumptions that were incorporated into the equations used to characterize risks and hazards in the Agricultural Products Consumption Risk Assessment. The HHRA attempts to acknowledge these limitations by providing, in Volume V, a Sensitivity Analysis (Section 5.2) and a specific Uncertainty Analysis (Section 6). In some cases, the uncertainties are quantified and the impact of the alternative exposure variables on the risk estimates is developed and presented in the report (Vol. V, Tables 5-3a & b, Table 5-4). While these potential limitations are described in the Sensitivity and Uncertainty Analyses, no modifications to the final risk calculations have been made. Since this assessment is limited to a deterministic approach, there is no mechanism for a range of variables to be included in the calculations, despite EPA's recognition that the use of single-point estimates for some of the assumptions may have resulted in an overestimation of risk. Thus, the reader is left with a single estimate of cancer risks and non-cancer hazards without an appropriate understanding of the substantial uncertainty and variability (and likely overestimation) inherent in these estimates.

While data sufficient to develop distributions for the different parameters may not be available, and thus a probabilistic analysis cannot be performed, the HHRA needs to illustrate both in the body of the assessment (Vol. V, Sec. 5 – Risk Characterization) and Volume I the uncertainties in the exposure and risk estimates and the range of risks predicted from alternative assumptions. Thus, to the extent that the modifications suggested above are not made in the main risk assessment, they should at least be presented as alternatives. Examples of such alternative assumptions would include an adjustment to account for the portion of grass and corn silage grown within the 1 ppm isopleth, use of different soil-to-grass transfer factors, elimination of the corn silage exposure route, and use of alternative bioavailability factors and BCFs.

Moreover, many of the uncertainties could be reduced by the collection of additional data. Such data could include current site-specific COPC data on the animal products themselves – i.e., milk, beef, chicken, and/or eggs – or at least additional co-located soil and grass data.

---

[11] These comments do not address Peer Review Question D.4 at this time, and GE's comments relating to Questions D.5 and D.6 are discussed in the above sections.

5.5 Overall Reasonableness of Approach (Question D.8)

In its current form, the Agricultural Product Consumption Assessment is based on modeling. The transfer of the pre-determined COPC concentrations from soil to plant matter was modeled based on limited site data collected from a relatively small area of the Housatonic River floodplain or on hypothetical extrapolations from those data. Consumption of COPCs in this plant matter by farm animals, the accumulation of the ingested compounds in the animals, and their distribution into animal tissue consumed by humans (including eggs) were all estimated based entirely on models and not empirical data. As discussed above, the net result of using these multiple layers of modeled assumptions, many of which are very uncertain and likely overstated, is a set of exposure and risk estimates that are both unreliable and almost certainly overestimated. These problems can be addressed, to some extent, by the collection of additional site-specific data or, in the absence of such data, by the other modifications suggested above.

SECTION 6: TOXICITY ASSESSMENT, RISK EVALUATION, AND GENERAL ISSUES

Key Points

- The dioxin TEQ approach should not be applied to PCB congeners because:

  ⇒ The application of this approach to PCBs is still under scientific review and, in fact, has been required by a Congressional directive to be reviewed by the National Academy of Sciences.

  ⇒ Analyses based on empirical bioassay data show that the TEQ approach does not accurately predict the carcinogenic potency of PCB mixtures.

  ⇒ This approach requires use of a highly uncertain Cancer Slope Factor (CSF) for dioxin, which is unnecessary given the availability of empirically based CSFs for PCBs.

  ⇒ Application of both the PCB CSF and the dioxin CSF results in double counting the carcinogenic potential of the dioxin-like congeners in the PCB mixtures, since they are included in total PCBs. The HHRA's effort to adjust for this is both inadequate and incorrect.

  ⇒ The HHRA's predictions of PCB congener concentrations from tPCB data, based on limited comparisons, are highly uncertain and unreliable due to variability among river/floodplain reaches and the use of multiple laboratories.

- There is no accepted CSF for dioxin at this time. The CSF used in the HHRA is based on an outdated interpretation of the pathology results from the underlying rat study and an outdated rat-to-human scaling factor. Even accepting a linear non-threshold cancer model (which is questionable), correction for these factors would lead to a lower CSF for dioxin.

- The chronic Reference Dose (RfD) used in the HHRA to estimate non-cancer hazards of PCBs is based on the use of two uncertainty factors that are unnecessary. Correction for these would result in an RfD that is 10 times higher, which should be used.

- A subchronic RfD should be used for subchronic exposures (less than 7 years).

- A Hazard Index (HI) of 1 should not be used as a bright line to indicate unacceptable non-cancer risks. While HIs < 1 are considered "safe," HIs > 1 are not necessarily indicative of unacceptable hazards due to the conservatism built into the RfD.

- The HHRA should consider certain site-specific information not currently considered, including GE's Floodplain User Survey, the MADPH blood survey data, and the ATSDR cancer incidence data for Housatonic River communities. In addition, from a more general standpoint, the HHRA should consider recent comprehensive weight-of-evidence analyses of human epidemiological and clinical studies on cancer and non-cancer effects of PCBs.

SECTION 6: TOXICITY ASSESSMENT, RISK EVALUATION, AND GENERAL ISSUES

This section of GE's comments addresses issues raised in Parts E ("Integrated Risk Characterization") and F ("General") of the Peer Review Charge. It begins by discussing the important issues relating to the toxicity assessment in the HHRA, which applies to all three of the individual risk assessments. It then discusses other questions listed in Parts E and F of the Charge, apart from the question on the HHRA's overall conclusions (Question F.7), which is addressed in Section 7 of these comments.

6.1 Toxicity Assessment (Questions E.1, E.4, F.1)

To assess both cancer risks and non-cancer hazards, the HHRA uses PCB toxicity values – i.e., Cancer Slope Factors (CSFs) for cancer risks and a Reference Dose (RfD) for non-cancer hazards -- that have been developed by EPA based on animal studies. This reliance on animal studies and default toxicological assumptions, to the exclusion of evidence from human epidemiological studies, may be responsible for one of the greatest sources of uncertainty in the HHRA. It is widely recognized, as well as intuitively plain, that human data, together with animal bioassays and mode-of-action data, are critical to an evaluation of the toxicity of a chemical (Cook, 1982; Dinman and Sussman, 1983; Layard and Silvers, 1989; EPA, 1998a, 1999e). Many chemicals do not have the same effect in humans as they do in animals, and even when similar effects do occur, the potency of a compound in humans often differs from its potency in animals. Both positive and negative epidemiological studies allow a direct determination of these differences. Moreover, for evaluating a body of epidemiological data on a particular chemical, EPA (1998a, 1999e) endorses a weight-of-evidence approach in which the available studies are evaluated in the context of well-accepted criteria for causation.

Formal weight-of-evidence evaluations using this approach have been conducted for both the potential cancer effects of PCBs and the potential non-cancer effects of PCBs. Detailed reports on these evaluations have been submitted to EPA. The cancer report (Golden and Shields, 2001) provides an assessment of the clinical and epidemiological evidence relating to whether PCBs cause cancer in humans, including 19 studies of whether PCBs are associated with an increased risk of any type of cancer in humans and 20 studies that have sought an association between PCBs and breast cancer. The report concludes that the collective weight-of-evidence from these studies demonstrates that exposure to PCBs is not a risk factor for breast cancer, that there is little credible evidence that PCBs have caused any type of cancer in highly exposed occupational cohorts, and that there is virtually no evidence that PCBs could cause cancer in humans at environmental exposure levels. The non-cancer report (Bernier et al., 2001) provides a comprehensive critical assessment of the 24 studies of the six major cohorts of children that serve as the primary source of data for evaluating potential effects of PCBs on growth or neurodevelopment in children, as well as 84 occupational and environmental studies (primarily of adults) that investigated potential associations between PCB exposure and effects on 14 different organs or organ systems. This report concludes that, with the possible exception of dermal and ocular effects in highly PCB-exposed workers, there is no credible evidence of a causal relationship between PCB exposure and adverse non-cancer health effects in humans.

The Executive Summaries of these reports are provided in Attachments J and K to these comments. GE believes that, at a minimum, these comprehensive weight-of-evidence evaluations of the human data should be considered and cited in the HHRA. As illustrated by these evaluations, the human epidemiological studies indicate that potential risks to humans from the PCBs in soils, sediments, and biota in the Housatonic River area are not nearly as great as calculated through the use of toxicity values based on animal studies.[12]

Nevertheless, GE recognizes that the HHRA relies on animal-based toxicity values. Within that context, there are a number of aspects of the toxicity assessment that are of concern to GE. These include the use of the TEQ approach to evaluate potential cancer risks from the so-called dioxin-like PCB congeners, the CSF selected for evaluating dioxin and dioxin TEQs, the RfD selected to evaluate non-cancer effects of PCBs, and the use of a chronic RfD to evaluate subchronic and intermittent exposures. Each of these issues is discussed below.

6.1.1 Use of TEQ Approach To Evaluate PCB Congeners

In all three of the risk assessments in the HHRA, EPA has not only calculated the potential carcinogenic risks associated with tPCBs (Aroclor-based), but has then added to those risks the

[12] For example, studies of worker populations over the past 25 years at GE plants in Hudson Falls and Fort Edward, New York, demonstrate that the cancer and non-cancer toxicity of PCBs is significantly lower than the toxicity values used in the HHRA. These studies focused not on laboratory animals, but on the very workers who were exposed to PCBs on a daily basis. Studies by a broad array of experts – from Dr. Renate Kimbrough to scientists from the National Institute for Occupational Science and Health -- have demonstrated that these workers are as healthy as the rest of the general population (Brown and Jones, 1981; Brown, 1987; Kimbrough et al., 1999; Nicholson, 1987; Taylor, 1988).

additional potential carcinogenic risks associated with TEQs of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). In calculating those TEQs, EPA has included concentrations of PCDDs, PCDFs, and the so-called dioxin-like congeners of PCBs, all of which are converted to TEQs through the use of Toxicity Equivalency Factors (TEFs) developed by the World Health Organization (WHO) (van den Berg et al., 1998). EPA has then calculated the carcinogenic risks of the resulting TEQ concentrations by applying the CSF for TCDD, 150,000 $(mg/kg\text{-}day)^{-1}$ that was previously listed in EPA's Health Effects Assessment Summary Tables (HEAST) (EPA, 1997b). Finally, EPA has added the predicted cancer risks associated with TEQs to those calculated for tPCBs. In support of the TEQ approach, the HHRA cites a 1998 EPA guidance document, the van den Berg et al. (1998) discussion of the WHO's recommended TEF approach, and a statement attributed to EPA's Science Advisory Board (SAB) (EPA, 2001b) in reviewing the Agency's draft Dioxin Reassessment (EPA, 2000), which uses the TEQ approach (see HHRA, Vol. 1, pp. 2-7, 2-12).

GE does not believe that the TEQ approach should be applied to PCB congeners at this time, because: (1) the application of this approach to PCBs is not an accepted Agency approach and, in fact, will be reviewed by the National Academy of Sciences (NAS) at the direction of Congress; (2) data reveal that the TEQ approach does not accurately predict the carcinogenic potency of PCB mixtures; (3) this approach requires use of a highly uncertain CSF for TCDD, which is unnecessary given the availability of a CSF for PCB mixtures based on animal bioassay data; (4) use of the TEQ approach in combination with an assessment of tPCBs using the CSF for PCBs results in double counting the carcinogenic potential of the dioxin-like PCB congeners in the PCB mixtures; and (5) there is high degree of uncertainty associated with EPA's prediction of PCB congener concentrations based on tPCB data, since that conversion was based on very limited data and may not provide representative estimates of actual concentrations. These issues are discussed in more detail in the following sections.

6.1.1.1 Application of TEQ approach to PCBs still under review

The inclusion of PCB congeners in the TEQ approach is highly controversial and remains under scientific review. The documents cited in the HHRA in support of that approach do not establish that it is an accepted Agency approach. First, the 1998 guidance document (cited in Volume I, p. 2-7) does not say anything about the inclusion of PCBs in the TEQ approach; it simply recommends preliminary remediation goals for dioxins and furans (EPA, 1998b). Second, the van den Berg et al. (1998) paper, which describes the TEFs developed by the WHO, does not constitute guidance or policy of the U.S. EPA. Third, the statement in the HHRA (Vol. 1, p. 2-12) attributed to EPA's SAB (2001b), that "EPA is … within the bounds of current science to use a judicious TEF approach until such time as a better approach may be developed," was actually the opinion of *one individual SAB member*. That individual was remarking about the paucity of data on many individual dioxin-like compounds, which was the basis for the development of TEFs. (PCBs, of course, do not lack relevant toxicological data; EPA has evaluated PCB cancer risks using conservative animal models and PCB data for over 20 years using CSFs established by EPA.) Furthermore, the majority of the SAB made a much more relevant statement:

> "Based on the PCB-related data presented at the public meeting (later determined to be based on the work of Mayes et al., 1998), questions were raised about whether the recommended TEF values for selected PCBs are consistent with the experimental carcinogenicity data that are now available on these specific chemicals. Since one of the important foundations for the EPA position that background uptake in the diet poses a significant cancer hazard is based on the TEFs presented in the document, EPA should review these data and make a determination whether a revision of the TEF values for the PCBs is appropriate. This is especially important since PCBs are, in many situations, the predominant source of human exposures." (EPA, 2001b, at p. 29)

More significantly, the underlying draft Dioxin Reassessment (EPA, 2000), which the SAB was reviewing and which contains, as a key aspect, the application of the TEQ approach to PCBs, has not been finalized and remains under scientific review. Hence, neither the SAB's Report nor the draft Dioxin Reassessment constitutes EPA policy or guidance. Although EPA's Peer Review Panel Charge lists the Agency's 2000 draft Dioxin Reassessment in the list of relevant EPA guidance and policy documents, that document has undergone some changes since the 2000 draft, and EPA makes clear on its own website (http://cfpub.epa.gov/ncea/cfm/dioxin.cfm) that the draft Dioxin Reassessment "should not be construed to represent Agency policy or factual conclusions" and "should not be cited or referred to as EPA's final assessment of dioxin risks."

In fact, Congress has directed that the draft Dioxin Reassessment, including its application of the TEQ approach to PCB congeners, be reviewed by the NAS. In February 2002, the chairman of a House subcommittee sent a letter to the EPA Administrator requesting that, due to "substantial questions regarding the scientific underpinning of the Reassessment's conclusions about the toxicity of dioxin and 'dioxin-like' compounds," EPA should undertake an agreement with the NAS to review the draft Dioxin Reassessment, including "the appropriateness of including 'dioxin-like' chemicals in the risk assessment without independent empirical review of their effects" (Walsh, 2002). Thereafter, EPA asked for additional time to convene an Interagency Working Group (IWG) to review these issues. On February 13, 2003, the House and Senate agreed to a conference report (House of Representatives, 2003) on an appropriations bill (later signed by the President on February 20, 2003 as Public Law 108-7) directing that if the IWG did not complete its review within 60 days of enactment, EPA is to contract with the NAS as quickly as possible to review the draft Dioxin Reassessment, including the issues specified in Chairman Walsh's letter. Since the IWG did not complete its review by that date, Congress's directive requires that the draft Dioxin Reassessment be submitted to the NAS for review.

Since one of the key issues to be addressed in the NAS review of the Dioxin Reassessment is the appropriateness of including dioxin-like PCB congeners in the TEQ approach, it is inappropriate for EPA to apply that approach to dioxin-like PCB congeners in a site-specific risk assessment prior to the completion of the NAS review. Doing so before the NAS has examined the "substantial questions regarding the scientific underpinning" of that approach would raise serious data quality issues.

6.1.1.2 Unreliable prediction of the carcinogenic potential of PCB mixtures

To evaluate the validity of the TEQ methodology in estimating the cancer potency of PCB mixtures (such as those found in the environment), AMEC has conducted tests of the approach using the results of two-year cancer bioassays involving four PCB mixtures of known composition that were fed to Sprague-Dawley (SD) rats. Those tests are described and their results are presented in Attachment L to these comments. In the first test, the effective CSFs in SD rats were determined for the TEQ components of each PCB mixture and compared to that of TCDD (based on a two-year cancer bioassay of SD rats that had been fed TCDD). A basic premise of the TEQ method is that a given dose of TEQ has equal biological potency irrespective of the chemical mixture from whence it came (van den Berg, et al., 1998). Thus, each CSF determined in this way should be equivalent to that of TCDD. However, the CSFs for the TEQs in these mixtures varied by up to a factor of 24 across the range of PCB mixtures tested, showing that TEQ is not an accurate predictor of cancer potency for PCB mixtures.

In a second test, the human CSFs for three PCB mixtures were determined using the TEQ methodology and compared to the empirically derived CSFs for those mixtures, as cited in EPA's (1996b) cancer dose assessment of PCBs. If the TEQ method is an accurate predictor of the potency of the dioxin-like PCBs in a PCB mixture, then one would expect the CSFs determined through the TEQ method to be consistent with the CSFs derived empirically for the PCB mixtures, which included both dioxin-like and non-dioxin-like PCB congeners. However, the comparisons showed that the TEQ-based CSFs are greater than the mixture-based CSFs, indicating that the TEQ approach substantially over-predicts the carcinogenic potency of PCB mixtures relative to the actual potencies demonstrated in laboratory bioassays.

As discussed further in Attachment L, there are several potential reasons why the TEQ method is found to overstate the carcinogenic potency of PCB mixtures. Both the empirical findings of these tests and the theoretical reasons discussed in Attachment L indicate that the TEQ approach does not accurately estimate PCB cancer response and thus should not be used for evaluating potential cancer risks of PCBs.

In addition, there is a practical reason why the TEQ approach cannot be reliably applied in site-specific risk assessments, at least those involving fish consumption. This is demonstrated by the observation that, in a fish consumption assessment, the TEQ methodology can predict risks approaching the top of EPA's risk range even for fish samples in which the PCB congeners are *not detected*. The reasons for this are that the proposed TEF for PCB-126 is very high and that there are no commercially available analytical methods that have detection limits at the pg/kg (ppq) levels. For example, using Method 1668, the estimated method detection limit for PCB-126 is 14 ng/kg or 0.000014 mg/kg (EPA, 1999d). Thus, using that method and the approach used in the HHRA to reduce the data, a non-detect sample of PCB-126 would be assumed to be present at a concentration of 0.000007 mg/kg (half the detection limit). If the TEF for PCB-126 (0.1) is applied to this concentration, the result is an estimated concentration of 0.0000007 mg/kg. When this concentration is then included in the assumptions used in the HHRA to evaluate exposures to anglers who consume fish (using 32 g/day, 365 days/year for 60 years and a body weight of 70 kg), the result is an estimated exposure of 3E-10 mg/kg-day. When this is combined with the TCDD CSF of 150,000 $(mg/kg\text{-}day)^{-1}$, the result is a predicted cancer risk of 4E-05 – *despite the fact that PCB-126 was not detected*. This result indicates that the TEQ method lacks sufficient power to discriminate between PCB exposures that may represent potential risks from those scenarios and those that are artifacts of the laboratory analytical protocols.

6.1.1.3 Uncertain CSF for TCDD

Use of the TEQ approach for PCBs requires application of a CSF for TCDD. As discussed in Section 6.1.2 and Attachment M to these comments, the appropriate CSF for TCDD is a matter of substantial controversy within the scientific community and CSFs ranging from 9,000 to 1,000,000 $(mg/kg\text{-}day)^{-1}$ have been proposed. The selection of any CSF within this range is associated with a high level of uncertainty. While such uncertainty might be acceptable in the absence of more reliable data, there is no need to rely on such a highly uncertain and controversial CSF for TCDD in evaluating risks of PCBs since CSFs for PCBs have been developed based on animal bioassays of different Aroclor mixtures, which contain both dioxin-like and non-dioxin-like congeners (EPA, 1996b, 2003a).

6.1.1.4 Double counting

The HHRA recognizes that simply adding the cancer risks estimated using the TEQ approach to those estimated using the CSF for PCBs could result in double counting the cancer risk from dioxin-like PCB congeners in the test material (Vol. I, p. 2-11), since the CSF for PCBs already includes the carcinogenic potential of the dioxin-like PCB congeners in the PCB mixtures tested in the animal studies that form its basis. In an effort to avoid such double counting, EPA has adjusted the TEQ risk to account for the presence of these congeners in the PCB mixtures that were used in the animal studies upon which the PCB CSF is based. This adjustment involved estimating the amount of dioxin-like PCBs already accounted for in the PCB CSF, which EPA states is 7.1 mg dioxin-like TEQ per kg PCBs based on an analysis by Cogliano (1998), and then subtracting this TEQ concentration from the predicted TEQ concentration from dioxin-like PCBs for each food exposure pathway (HHRA, Vol. I, pp. 2-10 - 2-11).

GE has several concerns with this adjustment. First, while the adjustment was made for the food consumption assessments, no adjustments were made in the Direct Contact Assessment. As a result, the double counting that the adjustment was designed to avoid is still present in the Direct Contact Assessment, as the HHRA recognizes (Vol. IIIA, p. 3-7).

Second, EPA's adjustment, where made, does not in fact avoid the double counting of risks. Because the Aroclor mixtures used to develop the PCB CSF contain congeners with assigned TEF values, any attempt to use both the TEQ approach and the PCB CSF method to calculate PCB risks inherently double counts the contribution of the TEQ congeners. Currently, CSFs have only been developed for Aroclor mixtures and no method exists to adjust the CSFs when a congener or series of congeners is removed from an Aroclor mixture. Since the coplanar PCB congeners that are included in the TEQ scheme are considered to be carcinogenic, removing them from any Aroclor mixture will have the effect of decreasing the CSF, but the magnitude of the decrease cannot be determined.

Finally, even if the TEQ approach were appropriate with the type of adjustment proposed by EPA, the value used by EPA to represent the amount of dioxin-like congeners in the PCB CSF is erroneous. As recognized both by EPA (1996b, 2003a) and by Cogliano (1998), the chronic rat feeding study of four PCB mixtures (Aroclors 1016, 1242, 1254, and 1260) performed by Brunner et al. (1996) and later published by Mayes et al. (1998) "provides the best information for distinguishing the cancer potential of different mixtures" (Cogliano, 1998). As reported by EPA (1996b, 2003a), the highest upper-bound CSF from this study was the CSF of 1.5 (mg/kg-day)$^{-1}$ for Aroclor 1254. Aroclor 1260 had an upper-bound CSF of only 0.5 (mg/kg-day)$^{-1}$. Since EPA's range of CSFs for PCB mixtures was based primarily on the potencies observed in the Brunner et al. (1996) study, in which Aroclor 1254 was the most potent (EPA, 1996b, p. 35), the amount of dioxin-like congeners in that Aroclor should be used to represent the amount of congeners in the PCB CSF. While Cogliano (1998) reported an estimate of 7.1 mg dioxin-like TEQ per kg PCBs in Aroclor 1260, he also reported a quantity of 46.4 mg dioxin-like TEQ per kg PCBs in Aroclor 1254. It is thus the latter value that should be used for the adjustment.

Although the HHRA recognizes that the TEQ concentration in Aroclor 1254 is higher than that in Aroclor 1260, it attempts to justify its use of the ratio for Aroclor 1260 as the basis for the adjustment because the latter is the Aroclor "that most closely resembles the environmental mixture at the site" (Vol. I, p. 2-11). While it is true that Aroclor 1260 most closely resembles the PCB mixture at the site, this basis for the selection of the adjustment factor is not appropriate. The HHRA does not use the individual Aroclor 1260 CSF of 0.5 (mg/kg-day)$^{-1}$, derived from Brunner et al. (1996), as the basis for the risk calculations. Instead, it uses the upper-bound CSF of 2 (mg/kg-day)$^{-1}$, which, as noted above, was based primarily on the potencies observed in the Brunner et al. (1996) study, in which Aroclor 1254 was most potent (EPA, 1996b). Thus, to adjust for the double counting of dioxin-like TEQ in the CSF for PCBs, the adjustment should be made for the quantity of dioxin-like TEQ in Aroclor 1254, since it is that mixture which drives the PCB CSF. Hence, even if the TEQ approach were appropriate, instead of using the value of 7.1 in the equation at the bottom of p. 2-10 (Vol. I) in the HHRA, a value of 46.4 should be substituted. This would result in more than a six-fold greater concentration of the designated dioxin-like PCB congeners being accounted for by the PCB CSF.

6.1.1.5 Prediction of congener concentrations based on total PCB data

To use the TEQ approach in the HHRA, EPA had to estimate the concentrations of dioxin-like congeners. For the Direct Contact and Agricultural Products Consumption Assessments, because PCB congener data in floodplain soils were available for only approximately 10 percent of the samples, EPA developed regressions in order to estimate congener content of the Aroclor-only samples (Vol. I, Attachment 2). These regressions, however, were based on very limited data and on samples analyzed by a different laboratory than the one that analyzed the main set of tPCB data. There is a high level of uncertainty associated with these predictions because of differences in physical characteristics of the individual river reaches (which can affect the bioavailability and degradation of individual congeners) and because of different results between the laboratories used.[13] There has not been adequate analysis of analytical differences between the laboratories to allow substantiation of the reliability or applicability of the developed ratios used to predict congener concentrations for the bulk of the tPCB data. In these circumstances, the results of the regressions may not be representative of actual PCB congener concentrations. This additional uncertainty further diminishes the reliability of the TEQ approach.

6.1.1.6 Summary

For the above reasons, GE believes that the HHRA should be modified to remove the inclusion of PCB congeners from the TEQ approach and from the calculation of TEQs for all environmental media.

---

[13] The regressions were based on a comparison of the congener and Aroclor concentrations in samples that were analyzed by GERG, and then applied to the Aroclor concentration data provided by ITS and On-Site (for which there were no corresponding congener data) to estimate the congener concentrations in those samples. However, no inter-laboratory comparisons have been provided to demonstrate that there is no bias in the congener-to-Aroclor ratios that have been developed based on the GERG data. A review of the available analytical data (available in the November 2002 EPA data release) on Aroclor measurements performed by both ITS and GERG on the same floodplain soil samples revealed that the ITS Aroclor-based total PCB results from the same samples were approximately 3.7-fold greater than those analyzed by GERG, as shown on Figure 9. It is not known why the results reported by GERG are lower than those reported by ITS. It is possible, however, that certain peaks in the chromatographs were not counted. If this is the case, then the ratio of individual congeners to total Aroclor concentrations may not be representative of the other laboratory's analytical results.

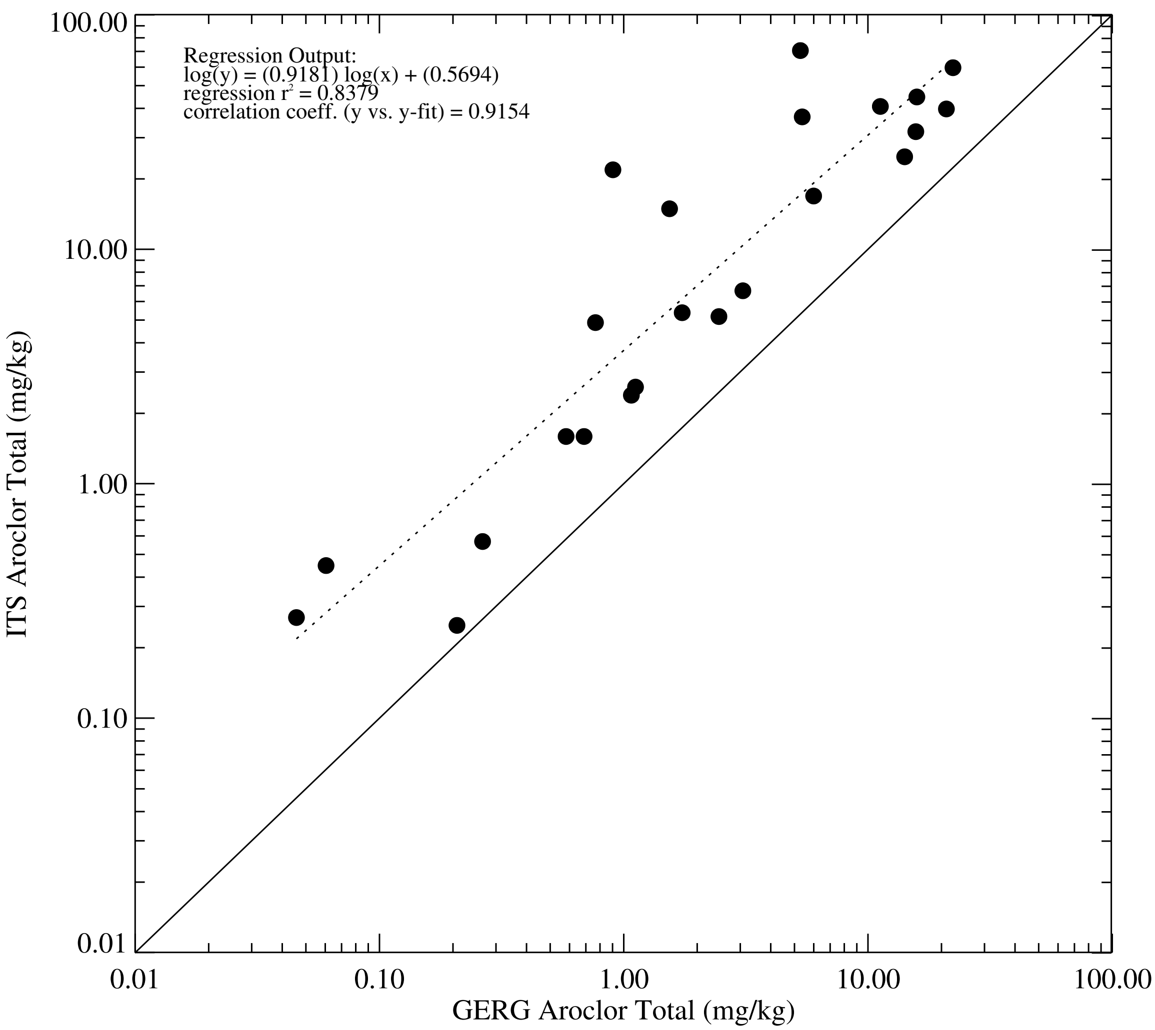

Figure 9. Comparison of Aroclor-based total PCB concentrations in surface floodplain soils.

Data source: US EPA (November 2002 release)

6.1.2 Cancer Slope Factor for TCDD

To assess the potential cancer risks from dioxin TEQs, the HHRA uses a TCDD CSF of 150,000 $(mg/kg\text{-}day)^{-1}$ (Vol. I, p. 2-7), which was derived from an early evaluation by EPA (1985) of the Kociba et al. (1978) rat bioassay data and was previously listed on HEAST (EPA, 1997b). In addition, in its uncertainty analyses, the HHRA discusses the proposed CSF of 1,000,000 $(mg/kg\text{-}day)^{-1}$ that is presented in the draft Dioxin Reassessment (EPA, 2000), noting that it would result in increased risks from TEQs (Vol. I, pp. 2-33, 5-33; Vol. IIIA, p. 7-8; Vol. IV, p. 7-14).

As discussed in detail in Attachment M to these comments, there is substantial uncertainty associated with the CSF for TCDD, and there is no current EPA guidance or policy on the appropriate CSF to use for TCDD. Estimates ranging from 9,000 to 1,000,000 $(mg/kg\text{-}day)^{-1}$ have been derived using a linear non-threshold cancer model. In fact, various CSFs have been derived from the same animal bioassay data (Kociba et al., 1978), with differences resulting from the extrapolation models and scaling methods used. Moreover, the CSF for TCDD is a key issue in the draft Dioxin Reassessment. The SAB could not reach consensus on this issue (EPA, 2001b), and one of the key issues that Congress specified for review by the NAS is "the validity of the non-threshold linear dose-response model . . . and the corresponding cancer slope factor calculated by the Agency through use of this model" (House of Representatives, 2003).

There is considerable controversy in the scientific community as to the validity of the linear non-threshold model for dioxin (JECFA, 2001; Pohl et al., 2002; Starr, 2001). In fact, review of the original data of Kociba et al. (1978) on all tumors in the rats studied – rather than just the liver tumors in females at the two highest doses, as were used by EPA to derive its original CSF – indicates that TCDD had a strongly negative (i.e., anti-carcinogenic) relationship with tumors at the lower doses.

Nevertheless, even within the framework of EPA's linear non-threshold model, GE does not believe that the HHRA should use the CSF of 150,000 $(mg/kg\text{-}day)^{-1}$, because, as shown in Attachment M, that was based on a now-outdated tumor classification scheme and a now-outdated method for scaling results from rats to humans. Instead, if EPA continues to use a linear model, then to assess potential cancer risks of PCDDs and PCDFs, GE recommends use of a CSF of 30,000 $(mg/kg\text{-}day)^{-1}$. As discussed in Attachment M, this factor is based on the same rat bioassay (Kociba et al., 1978) used to develop the CSF of 150,000 $(mg/kg\text{-}day)^{-1}$, but uses revised tumor incidence rates determined by an independent Pathology Working Group (PWG, 1990a,b) for liver tumors in that study and the inter-agency compromise interspecies scaling factor (EPA, 1992b) for scaling results from rats to humans.[14]

In any event, GE believes that the HHRA should not cite or use the proposed new CSF from the draft Dioxin Reassessment even in its uncertainty analyses. As noted above, that draft is not final and EPA has made clear on its website (http://cfpub.epa.gov/ncea/cfm/dioxin.cfm) that the draft Dioxin Reassessment "should not be construed to represent Agency policy." Further, as also noted above, Congress has called for that document to be submitted to the NAS for review, and the issues to be reviewed specifically include the validity of the recommended CSF for TCDD (House of Representatives, 2003). Thus, at the present time, it is unwarranted to use that draft's CSF, even as part of an uncertainty analysis, to suggest that the cancer risks from dioxin TEQs may be underestimated.

6.1.3 Reference Dose for Non-Cancer Effects of PCBs

The RfD used in the HHRA to assess the non-cancer effects of PCBs is the RfD listed on EPA's Integrated Risk Information System (IRIS) for Aroclor 1254, which is 2×10^{-5} mg/kg-day (Vol. I, p. 2-13). This RfD is based on the results of a five-year feeding study on Rhesus monkeys (Arnold et al., 1993a,b; Tryphonas et al., 1989, 1991a,b), in which dermal, ocular, and immunological effects were observed in the monkeys at a Lowest Adverse Effect Level (LOAEL) of 5×10^{-3} mg/kg-day. To derive a chronic RfD from these data, EPA applied several uncertainty factors (UFs) totaling 300 (see EPA, 2003a). These included a UF of 3 to account for extrapolation from monkeys to humans and a UF of 3 to account for use of a subchronic study to develop a chronic RfD.

GE previously provided to EPA a detailed analysis, prepared by AMEC, showing that even accepting the use of the same Rhesus monkey study used by EPA to develop this RfD, two of

[14] GE previously recommended use of this alternative CSF for TCDD to EPA. As discussed further in the next section, under a settlement agreement with GE and a subsequent EPA guidance memorandum issued to the EPA regions pursuant to that settlement (EPA, 1993), if an outside party questions the use of EPA's standard toxicity values during the course of an EPA risk assessment and presents alternative toxicological information that may be used in place of those values, EPA has an obligation to "consider all credible and relevant evidence before it." Thus, EPA had a duty to consider this alternative CSF for TCDD. There is no indication in the HHRA that EPA did so. GE urges EPA to do so now.

the UFs used by EPA are too high (AMEC, 2001). A summary of this analysis is presented in Attachment N to these comments. First, the toxicity data from the long-term PCB oral dosing studies on Rhesus monkeys were compared to PCB toxicity data in humans who had long-term occupational exposures. These human data included a detailed examination of workers who had been exposed to PCBs, particularly Aroclor 1254, at two capacitor manufacturing plants in New York State. These comparisons showed that, for the same types of effects, the monkeys were at least one to two orders of magnitude *more* sensitive to the effects than humans, and that thus there is no need or basis for using a UF of 3 for inter-species extrapolation. Second, this analysis showed the UF of 3 for study duration was unnecessary because the monkeys were dosed for more than 25 percent of their lifetimes and most of them had reached pharmacokinetic equilibrium with respect to PCB concentrations in their tissue and blood. Thus, the study should be considered equivalent to a chronic study, such that no adjustment is needed for exposure duration. Based on replacing these two UFs of 3 with UFs of 1, AMEC (2001) developed a revised chronic RfD for Aroclor 1254 of 2×10^{-4} mg/kg-day – a value that is 10 times higher than the RfD used in the HHRA.

In a lawsuit brought by GE in the early 1990s challenging EPA's adherence to its IRIS values in the absence of a rulemaking proceeding, GE and EPA reached a settlement agreement, which was filed in the court (Settlement Agreement in *General Electric Company v. Browner*, No. 93-1251, D.C. Circuit, October 25, 1993). Under that agreement and a subsequent EPA guidance memorandum issued to the EPA regions pursuant to that settlement (EPA, 1993), if an outside party questions the use of IRIS values during the course of an EPA risk assessment and presents alternative toxicological information that may be used in place of the IRIS values, the EPA region has an obligation to "consider all credible and relevant evidence before it." Thus, EPA Region I had a duty to consider this alternative information regarding the RfD for Aroclor 1254. There is no indication in the HHRA that EPA has done so. GE urges that EPA do so now and recommends that the HHRA be revised to utilize the alternative RfD of 2×10^{-4} mg/kg-day, for the reasons given in Attachment N.

6.1.4 Use of Chronic Reference Dose To Evaluate Subchronic Exposures

The HHRA uses a chronic RfD for all non-cancer calculations. GE believes, however, that, a subchronic RfD should be used for exposure scenarios that would meet the definition of subchronic. EPA (1989a) defines subchronic exposures as those exposures that are shorter than 7 years in duration. Thus, all exposures to 1 to 6 year old children, which involve a duration of six years, meet EPA's definition for subchronic exposures. Indeed, for such children involved in recreational activities, even assuming an RME exposure frequency of 90 days/year, they would only be exposed for a total of 540 days, which is less than 25 percent of the 7-year chronic exposure period established in EPA (1989a) guidance. In addition, certain exposure scenarios are assumed to involve highly intermittent exposures, which makes them similar to subchronic exposures. For example, while exposures to utility workers occur over a period of more than 7 years, EPA has assumed that their exposure occurs 5 days/year for a period of 25 years. Thus, their total exposure would consist of 125 days, which amounts to 1/3 of a year or less than 5 percent of the 7-year chronic exposure period.

EPA has established a subchronic RfD of 5×10^{-5} mg/kg-day (EPA, 1997b). Even if the Agency does not change the chronic RfD as discussed above, GE believes that, at a minimum, the subchronic RfD should be used for the above-described exposure scenarios, which are in fact subchronic or equivalent to subchronic.

6.1.5 Uncertainties in Toxicity Assessment (Question E.4)

Although the various uncertainty analysis sections of the HHRAs discuss some of the uncertainties associated with the toxicity assessment, GE believes that the following, all of which would tend to overestimate risks, have not been identified or adequately discussed:

- The general uncertainties in using toxicity values based on animal studies, despite the existence of a large body of epidemiological evidence;
- The marked limitations and uncertainties associated with the TEQ approach, and particularly its application to PCB congeners – including its apparent over-prediction of the cancer potency of PCB mixtures, assignation of specific TEFs to individual congeners, use of a highly uncertain CSF for dioxin, inability to adequately account for double counting of potentially carcinogenic congeners, and the estimation of congener levels based on a comparison of limited congener and tPCB data;
- The high level of uncertainty in the CSF for TCDD;
- The high level of conservatism associated with the RfD for PCBs, which includes the use of two UFs that are unnecessary or, at least, highly conservative; and
- The use of the chronic RfD to evaluate less than chronic exposure situations.

6.2 Identification of Important Assumptions for Estimation of Dose (Question F.2)

The HHRA identifies the important assumptions associated with each pathway but, in many cases, the assumptions themselves and their underlying justifications are buried in the

Appendices. This makes it difficult for the reader to see clearly the key assumptions that underlie the risk estimates. For example, for the direct contact pathway, while Tables 4-2 through 4-4 (in Volume IIIA) indicate the method used to calculate the EPC, the only place that the actual EPCs are clearly presented is in the individual figures for each EA, which is presented in a separate volume (Volume IIIB). Thus, one must search through all of the figures to determine what EPCs were used for each EA. Similarly, the exposure frequencies for the general recreational scenario are not presented in Volume I at all and are not listed in tabular form even in Appendix B (Volume IIIA). They can be found only by reading the individual sections in that appendix.

This approach is inconsistent with the requirement in EPA's Information Quality Guidelines (EPA, 2002b) that the information presented in Agency documents should be transparent. It prevents a clear understanding by the reader of the principal assumptions on which the risk estimates are based, which may be critical to understanding the reasonableness of those estimates. GE recommends that EPA reorganize sections of the HHRA so that all of the pertinent exposure assumptions for each analysis can be easily accessed and understood by the reader.

6.3 Calculations of Carcinogenic and Non-Carcinogenic Risks (Question F.3)

The HHRA uses a Hazard Index of 1.0 as a benchmark for evaluating predicted non-cancer effects, thus implying that an HI over 1.0 is indicative of unacceptable non-cancer hazards. An HI of 1.0 is often used as a screen to indicate whether there is a potential for adverse effects and HIs less than 1.0 are considered to be "safe," thus requiring no additional evaluation (EPA, 1996c; 2002c). However, HIs greater than 1.0 do not necessarily constitute a matter of concern or indicate that an adverse health effect will occur. They only indicate that a conservative threshold has been exceeded.

The HI is the ratio of the predicted dose to the RfD. The RfD represents a daily intake level (or dose) that will not result in non-cancer health effects. That level is typically calculated by applying multiple uncertainty factors to the no-effect or lowest-effect level in the underlying study. Thus, if the HI is less than 1.0, then the dose is less than the RfD and no risk is predicted. However, given the uncertainty factors and conservatism inherent in the derivation of the RfD, the converse is not true: a calculated HI greater than 1.0 does not necessarily mean that significant hazards are predicted.

The RfD is itself defined by the EPA as "an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime" (EPA, 1988a). With uncertainty spanning an order of magnitude or greater built into the very definition of the RfD, a calculated HI greater than 1.0 cannot and should not automatically be interpreted as presenting an unacceptable hazard or warranting remedial action. EPA has acknowledged this in a recent guidance memorandum (EPA, 2003b), stating that the RfD "does not represent a 'bright-line' between safety and risk. Because of the use of uncertainty factors in deriving the RfD so as not to underestimate the 'safe' level, the specific level at which actual risk from exposure begins above the RfD cannot be precisely calculated."

This view was also expressed in a report by the Presidential/Congressional Commission on Risk Assessment and Risk Management: "[U]se of risk estimates with bright lines, such as one-in-a-million, and single point estimates in general, provide a misleading implication of knowledge and certainty. As a result, reliance on command-and-control regulatory programs and use of strict bright lines in risk estimates to distinguish between safe and unsafe are inconsistent with the Commission's Risk Management Framework …." (EPA, 1997c). Further, there are precedents from other sites in this EPA region indicating that EPA views the non-cancer risk threshold as an HI range from 1 to 10. For example, EPA's Record of Decision (ROD) for the Fletcher's Paint Works and Storage Facility Superfund Site in New Hampshire (EPA, 1998c) and the ROD for the Charles George Reclamation Trust Landfill in Massachusetts (EPA, 1988b) state that EPA's non-cancer risk range is "usually a hazard index between 1 and 10."

GE recommends that the HHRA make this point explicitly. Correspondingly, in discussing the results of the various risk assessments, the HHRA should not consider HIs greater than 1.0 as necessarily indicative of unacceptable non-cancer hazards.

6.4 Overall Uncertainties (Question F.4)

GE's views on the uncertainty analyses presented in the HHRA have been provided in Sections 3.5, 4.4, 5.5 and 6.1.5 of these comments.

6.5 Consideration of Other Pertinent Information (Questions F.5 and F.6)

There are other sources of information available about potential exposures and risks to individuals living and recreating in the Housatonic River valley that provide valuable insight into the estimates of exposure and predicted risks and hazards developed in the HHRA. These information sources, which underscore the level of conservatism associated with the HHRA and its risk estimates, should be discussed in the Risk Characterization to provide perspective and points of comparison with the risks and hazards that have been predicted in the HHRA.

<u>TER Floodplain User Survey.</u> As discussed previously and in Attachment A, the Housatonic River Floodplain User Survey conducted by TER (2003) provides a substantial amount of information about the types and intensity of recreational usage of individual EAs between the Confluence and Woods Pond. Although the HHRA mentions this survey (Vol. IIIA, p. 4-9), it did not take its findings into consideration in developing either exposure scenarios for individual EAs or selecting exposure frequencies for the EAs. The TER survey data are site-specific and highly relevant to the Direct Contact Assessment in that they characterized specific recreational uses of individual EAs during a six-month period (end of April through the end of October) when one would expect use of the floodplain to be at its highest. These data clearly indicate that, despite extensive observations, little to no recreational usage occurred in most EAs and only a relatively few EAs exhibited regular usage. In addition, the survey did not observe some of the types of activities that the HHRA assumes occur in certain EAs with high frequency. Thus, these data indicate that the RME exposure assessments in the HHRA have a substantial tendency to overestimate actual exposures that occur in many of these EAs. GE recommends that the HHRA be revised to consider these survey data and to modify both the designation of exposure scenarios for certain EAs and the selection of recreational exposure frequencies for many EAs to provide more realistic estimates of exposure.

<u>MADPH Blood Monitoring Data.</u> The reasonableness of the exposure assumptions used in the HHRA should also be considered in light of the blood PCB levels that were reported in the same MADPH (1997) study from which EPA has selected some of its exposure parameters (e.g., exposure duration for certain direct contact pathways and for fish and waterfowl consumption). In that study, participants were selected for blood sampling based on factors indicating the highest potential for exposure – e.g., age and length of residence near the Housatonic River and recreational activities associated with the river, including both fishing and fish consumption. PCB serum analysis was also offered to volunteer residents at their request. As reported by

MADPH (1997), the serum PCB concentrations among non-occupationally exposed individuals tested were within the normal background range for non-occupationally exposed individuals nationwide, thus indicating that even in individuals believed to have the highest potential for exposure, serum PCB levels were not elevated relative to those in the general population. GE believes that the HHRA should take into account and explicitly discuss these important real-world site-specific results to provide a context for the estimated risks. Specifically, these results provide an empirical confirmation that the extremely high exposure assumption values currently used in the HHRA, especially for the RME analysis, are unrealistic.

Cancer Incidence Data for Berkshire County. The HHRA predicts cancer risks as high as 8 in 1,000 persons for the fish consumption pathway. However, as discussed in Section 2.3, the latest cancer incidence data reported by ATSDR (2002) for the towns adjacent to the most contaminated portion of the Housatonic River indicate no excessive cancer incidence for the six cancer types evaluated despite a substantial latency period, and no indication that cancer incidence was associated with PCB exposure. This is so even though the historical releases to the Housatonic River occurred decades ago, well before consumption advisories were established. GE believes that the lack of elevated cancer rates in the area should be discussed in the HHRA to provide perspective for EPA's hypothetical cancer risk estimates.

Weight-of-Evidence Evaluations of Cancer and Non-Cancer Effects of PCBs. As discussed at the beginning of Section 6.1, comprehensive weight-of-evidence evaluations, using accepted causation criteria, have been conducted of the human epidemiological and clinical evidence on potential cancer and non-cancer effects of PCBs in humans. The Executive Summaries of those reports are provided in Attachments J and K to these comments. For the reasons discussed in Section 6.1, GE believes that these evaluations should be considered and cited in the HHRA, since they indicate that potential risks to humans from the PCBs in the Rest of River area are not nearly as great as calculated through the use of toxicity values based on animal studies. The same is true of the worker epidemiological studies mentioned in note 12 on p. 6-2.

SECTION 7: CONCLUSIONS

For the reasons discussed in the prior sections of these comments, the current HHRA substantially overestimates potential current and reasonably foreseeable risks to human health from the direct contact pathways, the fish and waterfowl consumption pathways, and the agricultural exposure pathways. This overestimation is the cumulative result of several factors. First, for each assessment, the HHRA consistently and repeatedly selects exposure assumptions that are, at a minimum, upper-bound values and, in numerous instances, are unrealistically high and/or higher than supported by the most recent relevant information. The combination of these exposure assumptions results in exposure profiles that are no longer likely to be representative of actual exposures and thus lead to inflated risk estimates. Second, where the HHRA has attempted to refine its point estimates of exposures and risks through the use of probabilistic analyses, the additional conservatism added to the input distributions for those analyses, coupled with the other methods followed in those analyses, have again overestimated actual exposures and have resulted in risk estimates that are not substantially different from the point estimates. Third, the HHRA uses toxicity approaches and values that are overly conservative (even accepting the use of animal bioassays as the basis for them) and in some cases scientifically questionable (e.g., application of the TEQ approach to PCBs). Fourth, the HHRA does not consider certain site-specific real-life data (e.g., GE's Floodplain User Survey data, the MADPH blood survey data, the ATSDR cancer incidence data), as well as other human epidemiological studies, which indicate that actual human exposures and risks are less than predicted. Finally, the HHRA has not taken account of suggested alternative toxicity values (i.e., the alternative PCB RfD and TCDD CSF suggested by GE) despite its obligation to do so under the prior settlement with GE.

The result of using all these approaches is that the HHRA in its current form presents a skewed and inaccurate picture of the risks due to PCBs at the Rest of River site. GE believes that if the HHRA were revised to use the modifications and improvements suggested in these comments, the resulting exposure and risk estimates would be more representative of the potential for exposure and would provide a more accurate but still conservative characterization of the potential risks that may occur at the site. However, if changes are not made to address these concerns, GE believes that the HHRA cannot provide a supportable basis for making a remedial action decision for the Rest of River.

SECTION 8: REFERENCES

AMEC. 2001. *Development of a Revised Chronic Reference Dose for Polychlorinated Biphenyls (Aroclor 1254) Based on Empirical Data.* AMEC Earth & Environmental, Inc. May 31. Submitted to EPA Region 1 on June 6, 2001.

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. McGruire, D. Burns, J.R. Tanner, R. Stapley, Z.Z. Zawidzka, and D. Basford. 1993b. *Toxicological Consequences of Aroclor 1254 Ingestion by Female Rhesus (Macaca Mullata) Monkeys, Part 1A: Prebreeding Phase, Clinical Health Findings*. Health and Welfare Canada, Health Protection Branch, Toxicology Research Division.

ATSDR. 2002. *Assessment of Cancer Incidence, Housatonic River Area, 1982-1994*. US Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry (ATSDR). April.

Bernier, J.E., J. Borak, D. Palumbo, R.C. James, R.E. Keenan, and J. Silkworth (contributors or reviewers). 2001. *Non-Cancer Effects of PCBs – A Comprehensive Literature Review*. January 4. Submitted to EPA Headquarters by the PCB Panel of the American Chemistry Council, the National Electrical Manufacturers Associated, and the Utility Solid Waste Activities Group. January 4, 2001.

Brown, D.P., and M. Jones. 1981. Mortality and industrial hygiene study of workers exposed to polychlorinated biphenyls. *Arch. Environ. Health* 36:120-9.

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

Browner, C.M. 1995. Memorandum from Carol Browner to Assistant Administrators, Associate Administrators, Regional Administrators, General Counsel and Inspector General, Re: *EPA Risk Characterization Program*. March 21.

Brunner, M.J., T.M. Sullivan, A.W. Singer, M.J. Ryan, J.D. Toft, R.S. Menton, S.W. Graves, and A.C. Peters. 1996. *An assessment of the chronic toxicity and oncogenicity of Aroclor-1016, Aroclor-1242, Aroclor-1254, and Aroclor-1260 administered in diet to rats*. Battelle Study No. SC920192. Chronic toxicity and oncogenicity report. Columbus, Ohio.

Chaney, R.L., J.A. Ryan, and G.A. O'Conner. 1996. Organic contaminants in municipal biosolids: risk assessment, quantitative pathways analysis, and current research priorities. *Sci. Total Environ.* 185:187-216.

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

ChemRisk. 1992. *Consumption of Freshwater Fish by Maine Anglers*. ChemRisk Division of McLaren/Hart, Portland, ME. July 24.

ChemRisk. 1994. *Methodology and Results of the Creel Survey for the Housatonic River*. ChemRisk Division of McLaren/Hart, Portland, ME. March 25.

Cogliano, V.J. 1998. Assessing the cancer risk from environmental PCBs. *Environmental Health Perspectives* 106(6):317-323.

Cook, R.R. 1982. The role of epidemiology in risk assessment. *Drug Metab. Rev.* 13:913-923.

Daubenmire, S.W. 1996. *Use of Great Lake Species as Bioindicators of Environmental Contamination and the Effect of Food Processing on the Reduction of Polychlorinated Biphenyl (PCB) Congeners, Homologs, and Total PCBs*. PhD. Dissertation, Michigan State University. (cited in HHRA)

Dinman, B.D. and N.B. Sussman. 1983. Uncertainty, risk and the role of epidemiology in public policy development. *Journal of Occupational Medicine* 25:511-516.

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. 1980. *Ambient Water Quality Criteria for Polychlorinated Biphenyls,* Office of Water Regulations and Standards, PB81-117798, Criteria and Standards Division, Washington, DC.

EPA. 1985. *Health Assessment Document for Polychlorinated Dibenzo-p-dioxins.* EPA/600/8-84/014F. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment. Washington, D.C.

EPA. 1988a. *Integrated Risk Information System (IRIS). Background Document 1*, National Center for Environmental Assessment, Cincinnati Office, Cincinnati, OH.

EPA. 1988b. *EPA Superfund Record of Decision: Charles-George Reclamation Trust Landfill*. EPA ID: MAD003809266, OU 03, 04, Tyngsborough, MA. EPA/ROD/R01-88/029. September 29.

EPA. 1988c. *Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs),* Final Report, Office of Health and Environmental Assessment, ECAO-CIN-414, Cincinnati, OH.

EPA. 1989a. *Risk Assessment Guidance for Superfund; Volume I: Human Health Evaluation Manual (Part A) – Interim Final*. U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C. EPA/540/1-89-002. July.

EPA. 1989b. *Assessing Human Health Risks from Chemically Contaminated Fish and Shellfish: A Guidance Manual.* U.S. Environmental Protection Agency, Office of Marine and Estuarine Protection, Washington, DC. EPA-503/8-89-002. September.

EPA. 1992a. Final guidelines for exposure assessment. U.S. Environmental Protection Agency, Washington, D.C. *Federal* Register 57(104):22888-22938. May 29.

EPA. 1992b. Draft report: A cross-species scaling factor for carcinogen risk assessment based on equivalence of $mg/kg^{3/4}/day$; Notice. U.S. Environmental Protection Agency, Washington, D.C. *Federal Register* 57(109):24152-24173. June 5.

EPA. 1993. *Use of IRIS Values in Superfund Risk Assessment.* Memorandum from W.H. Farland to Divisional Directors. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response. OSWER Directive 9285.7-16. December 21.

EPA. 1995. *Land Use in the CERCLA Remedy Process.* Memorandum from E.P. Laws to EPA Regional Offices. OSWER Directive 9355.7-04. May.

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

EPA. 1996b. *PCBs: Cancer Dose Assessment and Application to Environmental Mixtures*. U.S. Environmental Protection Agency, Office of Research and Development. EPA/600/P-96/001. Washington, D.C.

EPA. 1996c. *Soil Screening Guidance: Fact Sheet*. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response. EPA/540/F-95/041. July.

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

EPA. 1997b *Health Effects Assessment Summary Tables, FY 1997* Update. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response. EPA-540-R-97-036. July.

EPA. 1997c. *Risk Assessment and Risk Management in Regulatory Decision-Making*. Final Report, Volume 2. The Presidential/Congressional Commission on Risk Assessment and Risk Management.

EPA. 1998a. *Guidelines for Neurotoxicity Risk Assessment*. *Federal Register* 63(93):26926-26954.

EPA. 1998b. Memorandum from Timothy Fields, Acting Administrator, to Regional Directors, Re: *Approach for Addressing Dioxin in Soil at CERCLA and RCRA Sites*. U.S. Environmental Protection Agency, Washington, D.C. OSWER Directive #9200.4-26. April 13.

EPA. 1998c. *EPA Superfund Record of Decision: Fletcher's Paint Works & Storage*, EPA ID: NHD001079649, OU 01, Milford, NH. EPA/ROD/R01-98/124. September 30.

EPA. 1999a Memorandum from Ann-Marie Burke, Toxicologist, to Richard Cavagnero, GE Project Leader, Re: *Protectiveness of Cleanup Levels for Removal Actions Outside the River – Protection of Human Health*. Attachment A to Appendix D to Consent Decree in *United States et al. v. General Electric Company* (No. 99-30225-MAP, U.S. Dist. Court for Dist. of Mass.). August 4.

EPA. 1999b *Report of the Workshop on Selecting Input Distributions for Probabilistic Assessments.* U.S. Environmental Protection Agency, Risk Assessment Forum. EPA/630/R-98/004. January.

EPA. 1999c. *Report on FIFRA Scientific Advisory Panel Meeting, February 23-24, 1999.* 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.

EPA. 1999d. *Method 1668, Revision A: Chlorinated Biphenyl Congeners in Water, Soil, Sediment, and Tissue by HRGC/HRMS*. U.S. Environmental Protection Agency, Office of Water. EPA-821-R-00-002. December.

EPA. 1999e. *Guidelines for Carcinogen Risk Assessment (Review Draft – Interim Final)*. Risk Assessment Forum, Washington, D.C. NCEA-F-0644. July.

EPA. 2000. *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds. Part II. Health Assessment for 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds.* Environmental Protection Agency. NCEA-I-0836. May.

EPA. 2001a. *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. 2001b. *Dioxin Reassessment – An SAB Review of the Office of Research and Development's Reassessment of Dioxin*. U.S. Environmental Protection Agency, Science Advisory Board. EPA-SAB-EC-01-006. May

EPA. 2002a. *Calculating Upper Confidence Limits for Exposure Point Concentrations at Hazardous Waste Sites*. U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response. OSWER 9285.6-10. December.

EPA. 2002b. *Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility and Integrity of Information Disseminated by the Environmental Protection Agency*. U.S. Environmental Protection Agency. EPA/260R-02-008.

EPA. 2002c. *Region 9 PRGS Table 2002 Update.* Memorandum from S.J. Smucker, U.S. EPA Region 9, to PRGs Table Users. October 1.

EPA. 2003a. *Chemical Search for Aroclor 1254*. Integrated Risk Information System (IRIS), Cincinnati, OH.

EPA. 2003b. *Status of EPA's Interim Assessment Guidance for Perchlorate*. Memorandum from M.L. Horinko, Assistant Administrator, U.S. Environmental Protection Agency, to Assistant and Regional Administrators. January 22.

European Commission, Health & Consumer Protection Directorate-General, Scientific Committee on Food (2001). *Opinion of the SFC on the risk assessment of dioxins and dioxin-like PCBs in food*. Adopted 30 May 2001.

Gan, D.R, and P.M Berthouex. 1994. Disappearance and crop uptake of PCB from sludge-amended farmland. *Water Environ. Research* 66: 54-69.

Ginevan, M.E. and D.E. Splitstone. 2002. Bootstrap upper bounds for the arithmetic mean of right-skewed data, and the use of censored data. *Environmetrics* 13: 443-464.

Golden, R. and P. G. Shields, 2001. Technical Report: A Weight-of-Evidence Review of the Human Studies of the Potential Cancer Effects of PCBs. Lombardi Cancer Center, Washington D.C. April 10.

House of Representatives. 2003. *Making Further Continuing Appropriations for the Fiscal Year 2003, and for Other Purposes*. Conference Report H.R. 108-10 to accompany H.J. Res. 2 – Public Law 108-7. 108th Congress, 1st Session. February 13. U.S. Gov't Printing Office, Washington, D.C., pp. 1445-46.

JECFA. 2001. *Joint FAO/WHO Expert Committee on Food Additives (JEFCA): Summary and Conclusions, 57th Meeting*, pp. 24-40. Food and Agriculture Organization/United Nations World Health Organization (WHO), Rome, Italy. June 5–14.

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* 41:161-171.

Kociba, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreon, C.E. Wade, D.A. Dittenber, R.P. Kalnins, L.E. Frauson, C.N. Park, S.D. Barnard, R.A. Hummel, and C.G. Humiston. 1978. Results of a two-year chronic toxicity and oncogenicity study of 2,3,7,8-tetrachlorodibenzo-p-dioxin in rats. *Toxicol. Appl. Pharmacol.* 46:279-303.

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

MADPH. 1997 *Housatonic River Area PCB Exposure Assessment Study.* Final Report. Massachusetts Department of Public Health, Bureau of Environmental Health Assessment Environmental Toxicology Unit. September.

MADPH. 2001. Letter from M. Steele, Bureau of Environmental Health Assessment, Massachusetts Department of Public Health, to B. Olson, U.S. EPA Region 1. September 10.

Mayes, B.A., E.E. McConnell, B.H. Nel, J.J. Brunner, S.B. Hamilton, T.M. Sullivan, A.C. Perters, M.J. Ryan, J.D. Toft, A.W. Singer, J.F. Brown, R.G. Menton, and J.A. Moore. 1998. Comparative carcinogenicity in Sprague-Dawley rats of the polychlorinated biphenyl mixture Aroclors 1016, 1242, 1254, and 1260. *Toxicol. Sci.* 41:62-76.

Mayes, B.A., G.L. Brown, F.J. Mondello, K.W. Holtzclaw, S.B. Hamilton, and A.A. Ramsey. 2002. Dermal absorption in rhesus monkeys of polychlorinated biphenyls from soil contaminated with Aroclor 1260. *Regul. Toxiol. Pharmacol.* 35(3):289-295.

MDFW. 2002. *Masswildlife Migratory Bird Regulations for 2001-2002*. Massachusetts Division of Fish and Wildlife. [online: www.state.ma.us/dfwele]

Nicholson, W. J. 1987. *Report to the Workers Compensation Board on Occupational Exposure to PCBs and Various Cancers. Human Health Effects and Carcinogenic Risk Potential of PCBs*. Ontario Ministry of Labour.

O'Connor, G.A., D. Kiehl, G.A. Eicemen, and J.A. Ryan. 1990. Plant uptake of sludge-borne PCBs. *J. Environ. Qual.* 19:113-118.

Pao, E.M., K.H. Fleming, P.M. Guenther, S.U. Mickel. 1982. *Foods Commonly Eaten by Individuals: Amount Per Day and Per Eating Occasion.* U.S. Department of Agriculture. Home Economics Report No. 44.

Pohl, H.R., H.E. Hicks, D.E. Jones, H. Hansen and C.T. DeRosa. 2002. Public health perspectives on dioxin risks: Two decades of evaluations. *Hum. Ecol. Risk Assess*. 8:233-250.

Price, P.S., S.H. Su, and M.N. Gray. 1994. The effect of sampling bias on estimates of angler consumption rates in creel surveys. *Journal of Exposure Analysis and Environmental Epidemiology* 4(3):355-372.

Puffer, H.W. and R.W. Gossett. 1983. PCB, DDT, and benzo (a) pyrene in raw and pan-fried white croaker (Genyonemus lineatus). *Bull. Environ. Contam. Toxicol.* 30:65-73.

PWG. 1990a. *Pathology Working Group Report on 2,3,7,8-Tetrachlorodibenzo-p-dioxin Chronic Toxicity/Carcinogenicity Study in Sprague-Dawley Rats.* Prepared by R.M. Sauer, PWG Chairperson, PATHCO, Inc., Ijamsville, MD and submitted to R.A. Michaels, Chairperson, Maine Scientific Advisory Panel. March 13.

PWG. 1990b. *Hepatotoxicity in Female Sprague-Dawley Rats Treated with 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD).* PATHCO, Inc., Ijamsville, MD and submitted to R.A. Michaels, Chairperson, Maine Scientific Advisory Panel. April 27.

Rombauer, I.S., and M.R. Becker. 1975. *The Joy of Cooking*. Bobbs-Merrill Co., Inc, Indianapolis.

Rupp, E.M., F.L. Miller, and C.F. Baes. 1980. Some results of recent surveys of fish and shellfish consumption by age and region of U.S. residents. *Health Phys.* 39-165-175.

Simon, T.W. 1999. Two-dimensional Monte Carlo simulation and beyond: A comparison of several probabilistic risk assessment methods applied to a Superfund site. *Hum. Ecol. Risk Assess.* 5 (4): 823-843.

Singh, A.K., A. Singh, and M. Engelhardt. 1997. *The Lognormal Distribution in Environmental Applications.* U.S. Environmental Protection Agency. EPA/600/R-97-006. December.

Skea, J.C., S. Jackling, J. Symula, H.A. Simonin, E.J. Harris, and J.R. Colquhoun. 1979. *Summary of Fish Trimming and Cooking Techniques Used to Reduce Levels of Oil Soluble Contaminants*. Field Toxicant Research Unit, Rome, NY and Hale Creek Field Station, New York State Department of Environmental Conservation, Gloversville, NY. September.

Smith, W.E. 1972. *Effects of Three Cooking Methods on Pesticide Residues in Chinook and Coho Salmon*. Thesis. Michigan State University, Department of Food Science and Human Nutrition.

Smith, W.E., K. Funk, and M.E. Zabik. 1973. Effects of cooking on concentrations of PCB and DDT compounds in Chinook (Oncorhynchus tshawytscha) and (O. kisutch) Salmon from Lake Michigan. *J. Fish Res. BD. Canada* 30(5):702-706.

Stanek, E.J., E.J. Calabrese, R. Barnes, and P. Pekaow. 1997. Soil ingestion in adults – results of a second pilot study. *Ecotoxicology and Environmental Safety* 36:249-257.

Stanek, E.J., and E.J. Calabrese. 2000. Daily soil ingestion estimates for children at a Superfund Site. *Risk Analysis* 20(5):627-635.

Starr. T.B. 2001. Significant shortcomings of the U.S. Environmental Protection Agency's latest draft risk characterization for dioxin-like compounds. *Toxicol. Sci*. 64:7-13.

Swartout, J.C., P.S. Price, M.L. Dourson, H.L. Carlson-Lynch, R.E. Keenan. 1998. A probabilistic framework for the reference dose (probabilistic RfD). *Risk Analysis* 18(3):271-281.

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

TER. 2003. *Housatonic River Floodplain User Survey Summary Report*. Triangle Economic Research (TER), Research Triangle, NC. January 20. Submitted to EPA Region 1.

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.

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.

USFWS. 1998. *1996 National Survey of Fishing, Hunting, and Wildlife-Associated Recreation: Massachusetts.* U.S. Department of the Interior, Fish and Wildlife Service. FHW/96-MA. April.

Van den Berg, M., L. Birnbaum, A.T.C. Bosveld, B. Brunstrom, P. Cook, M. Feeley, J.P. Giesy, A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X. R. van Leeuwen, A.K. Djien 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. *Environmental Health Perspectives* 106(12):775-792.

Walsh, J.T. 2002. Letter from J.T. Walsh, Chairman, Subcommittee on VA, HUD and Independent Agencies Appropriations, U.S. House of Representatives, to Honorable Christine Todd Whitman, Administrator, U.S. Environmental Protection Agency. February 21.

Wang, Y., and S. Harrad. 2000. *Cooking-induced Reductions in Concentrations of Polychlorinated Biphenyls (PCBs) in Fish*: ΣPCB versus ΣTE. Human Exposure Posters – Organohalogen Compounds. Volume 48. Division of Environmental Health and Risk Management, University of Birmingham, Birmingham, U.K. (cited in HHRA)

Webber, M.D., R.I. Pietz, T.C. Granato, and M.L. Svoboda. 1994. Plant uptake of PCBs and other organic contaminants from sludge-treated coal refuse. *J. Environ. Qual.* 23:1019-1026.

Wester, R.C., H.I. Maibach, L. Sedik, and J. Melendres. 1993. Percutaneous absorption of PCBs from soil: In vivo rhesus monkey, in vitro human skin, and binding to powdered human stratum corneum. *Journal of Toxicology and Environmental Health* 39:375-282.

Zabik, M.E., A. Booren, M.J. Zabik, R. Welch, and H. Humphrey. 1996. Pesticide residues, PCBs and PAHs in baked, charbroiled, salt boiled and smoked Great Lakes lake trout. *Food Chemistry* 55: 231-239.