U.S. Army
Corps of Engineers

New England District
Concord, Massachusetts

U.S. Environmental
Protection Agency

New England Region
Boston, Massachusetts

RESPONSIVENESS SUMMARY TO PUBLIC COMMENTS ON NEW INFORMATION HUMAN HEALTH RISK ASSESSMENT FOR THE GE/HOUSATONIC RIVER SITE REST OF RIVER

DCN: GE-051605-ACRT

June 2005

Environmental Remediation Contract
GE/Housatonic River Project
Pittsfield, Massachusetts

Contract No. DACW33-00-D-0006

Task Order 0003

05P-0602

RESPONSIVENESS SUMMARY TO PUBLIC COMMENTS ON NEW INFORMATION — HUMAN HEALTH RISK ASSESSMENT FOR THE GENERAL ELECTRIC (GE)/HOUSATONIC RIVER SITE, REST OF RIVER

ENVIRONMENTAL REMEDIATION CONTRACT
GENERAL ELECTRIC (GE)/HOUSATONIC RIVER PROJECT
PITTSFIELD, MASSACHUSETTS

Contract No. DACW33-00-D-0006
Task Order No. 0003

DCN: GE-051605-ACRT

Prepared for

U.S. ARMY CORPS OF ENGINEERS
New England District
Concord, Massachusetts

and

U.S. ENVIRONMENTAL PROTECTION AGENCY
New England Region
Boston, Massachusetts

Prepared by

WESTON SOLUTIONS, INC.
West Chester, Pennsylvania

June 2005

Work Order No. 20123.001.096.0728

AUTHORS/CONTRIBUTORS

James Walsh, Florence Sevold, Tim Woods
Avatar Environmental, LLC
West Chester, Pennsylvania

Scott Ferson, Troy Tucker
Applied Biomathematics, Inc.
Setauket, New York

John Cassels
Geodesy, Inc.
Downingtown, Pennsylvania

George F. Fries
Silver Spring, Maryland

Harlee Strauss
H. Strauss Associates, Inc.
Natick, Massachusetts

Donna Vorhees, Cheri L. Butler, and Robin Dodson
Menzie-Cura & Associates, Inc.
Winchester, Massachusetts

Richard McGrath
Sleeman Hanley & DiNitto, Inc.
Boston, Massachusetts

Susan Svirsky, Margaret McDonough
U.S. Environmental Protection Agency
Boston, Massachusetts

Scott Campbell
Weston Solutions, Inc.
West Chester, Pennsylvania; Pittsfield, Massachusetts

TABLE OF CONTENTS

LIST OF ACRONYMS

APR	agricultural preservation restriction
ATSDR	Agency for Toxic Substances and Disease Registry
BCF	bioconcentration factor
CDPHE	Colorado Department of Public Health and the Environment
COPC	contaminant of potential concern
CSF	Cancer Slope Factor
CT	State of Connecticut
CTDEP	State of Connecticut Department of Environmental Protection
CTDPH	State of Connecticut Department of Public Health
CTE	Central Tendency Exposure
DEC	Direct Exposure Criteria
DOE	U.S. Department of Energy
EA	exposure area
EPA	U.S. Environmental Protection Agency
EPC	exposure point concentration
ESC	Environmental Stewardship Concepts
FI	fraction ingested
GE	General Electric Company
HEAL	Housatonic Environmental Action League
HEAST	Health Effects Assessment Summary Tables
HHRA	Human Health Risk Assessment
HRA	Housatonic River Area
HRFUS	Housatonic River Floodplain User Survey
IARC	International Agency for Research on Cancer
IMPG	Interim Media Protection Goal
IRIS	Integrated Risk Information System
MCA	Monte Carlo Analysis
MCLG	maximum contaminant level goal
MDPH	Massachusetts Department of Public Health
MFWP	Montana Fish, Wildlife and Parks
NAS	National Academy of Sciences
NTP	National Toxicology Program
PBA	probability bounds analysis
PCB	polychlorinated biphenyl
PRG	preliminary remediation goal
PSA	Primary Study Area

LIST OF ACRONYMS (Continued)

RAGS	Risk Assessment Guidance for Superfund
RfD	Reference Dose
RME	Reasonable Maximum Exposure
RSR	Remediation Standard Regulation
SAREP	Sportfishing and Aquatic Resources Education Program
SRBC	screening risk-based concentration
TAG	Technical Assistance Grant
TCDD	2,3,7,8-Tetrachlorodibenzo-p-dioxin
TEF	toxic equivalency factor
TEQ	toxic equivalence
TER	Triangle Economic Research
TF	transfer factor
tPCBs	total PCBs

INTRODUCTION

HHRA Responsiveness Summary

Introduction

This document presents the response from the U.S. Environmental Protection Agency (EPA) to comments received from the public pertaining to new information included in the Human Health Risk Assessment for the GE/Housatonic River Site, Rest of River (HHRA). The June 2003 HHRA was revised and reissued in February 2005 in response to comments and questions posed by a Peer Review Panel. The Peer Review was conducted by seven independent experts in the field of human health risk assessment.

Under the terms of the Consent Decree, EPA was required to conduct a human health risk assessment of the area referred to as the "Rest of the River," defined as the area of river and adjacent floodplain downstream from the confluence of the East and West Branches of the Housatonic River in Pittsfield, MA, and to conduct an independent Peer Review of the HHRA. The conclusions of the human health risk assessment, along with the conclusions from the ecological risk assessment that was also conducted by EPA and underwent Peer Review, will be taken into account by GE when developing an Interim Media Protection Goals (IMPG) Proposal that will be submitted to EPA for review.

Following the Peer Review of the June 2003 draft of the HHRA, EPA chose to exercise its option to revise and reissue the document in response to Peer Review comments. The revised HHRA was issued on February 16, 2005, and EPA announced a 30-day public comment period, subsequently extended to 45 days, that began on February 18, 2005, during which members of the public were invited to submit written comments restricted to the new information contained within the document which addressed the Peer Review Comments and questions. The public comment period closed on April 5, 2005. EPA received four sets of comments on the revised HHRA. This document provides EPA's response to those comments.

Approach and Organization of this Document

The full text of each of the four sets of comments received is reproduced in this Responsiveness Summary. The comments are presented alphabetically by commenter or group, as follows (the abbreviation used for each commenter throughout the document is enclosed in parentheses):

- State of Connecticut, Department of Environmental Protection (CT).
- General Electric Company (GE).
- Housatonic Environmental Action League (HEAL).
- Technical Assistance Grant recipient – Housatonic River Initiative/Environmental Stewardship Concepts (TAG).

EPA carefully reviewed the comments from each of the above entities and identified appropriate locations within each set of comments to insert responses. Each response

is identified by the abbreviation for the commenter followed by a sequential number. For example, the first response to comments from the State of Connecticut is identified as RESPONSE CT-1; the seventh response to comments from the General Electric Company is identified as RESPONSE GE-7. Each response is intended to address the comment or related series of comments immediately preceding it. In identifying appropriate locations for comments, EPA attempted to provide more comprehensive responses to related comments, as opposed to responding to individual sentences.

References used by EPA in responding to comments immediately follow the response. Although this in some cases requires citations to be repeated, it will allow readers interested in referring to the references to quickly identify those references that support a particular response.

Relationship of the Responsiveness Summary to the Human Health Risk Assessment for Rest of River

The Peer Review Panel reviewed the June 2003 draft Human Health Risk Assessment and comments provided to the Panel on that document by members of the public. A public Peer Review meeting was held in November 2003, after which the Panel submitted their final written comments on the document. At the beginning of the Peer Review process in 2003, EPA provided the opportunity for any party to submit written comments on the draft Human Health Risk Assessment to the Peer Review Panel for their consideration during the public comment period. EPA also provided the opportunity for any party to comment orally (and in writing) to the Panel at the November 2003 Peer Review Panel meeting. In March 2004, EPA produced a Responsiveness Summary to the Peer Review Panel comments.

EPA chose to revise the HHRA, including new information as necessary, to respond to the Peer Review Panel comments; this resulted in the February 2005 revised Human Health Risk Assessment. The February 16, 2005 notice issued by EPA soliciting public comment on the new information stated that EPA was seeking comment on "only the new information contained in the risk assessment regarding risks to adults and children who are exposed to PCBs and other contaminants while living or working near the Housatonic River, or using the river and floodplain for recreation or agricultural purposes." This additional opportunity for input from the public to the process was provided at this site to continue to promote public involvement in the development of documents and the decision-making process for the Rest of River.

EPA provided paper and/or electronic copies of the February 2005 revised Human Health Risk Assessment to the site information repositories and interested Citizens Coordinating Council members, and also provided a detailed list of the new information included in the revised Human Health Risk Assessment, to facilitate identification and review of the new information. In addition, both the document and the list of new information were posted on EPA's website. At the February 2, 2005 Citizens Coordinating Council meeting, EPA provided an overview of the changes to the HHRA based on the Peer Review Panel comments and answered questions from the public regarding the new information and the comment period. At the request of members of

the public, EPA granted an extension of the public comment period to 45 days, which then closed April 15, 2005. If a comment received during the public comment period did not pertain to the new information presented in the HHRA in response to comments from the Peer Review Panel, EPA did not provide a response in this Responsiveness Summary.

Together with this Responsiveness Summary, the February 2005 revised Human Health Risk Assessment now is considered to be the final HHRA for the GE/Housatonic River Rest of River site. In addition to the opportunities described above that were available for the public to provide input to the Human Health Risk Assessment, pursuant to the Consent Decree, and the Reissued RCRA Permit (Appendix G to the Decree), all parties will have an additional opportunity to comment when EPA issues the Statement of Basis proposing a response action for the Rest of River.

COMMENTS OF THE STATE OF CONNECTICUT, DEPARTMENT OF ENVIRONMENTAL PROTECTION (CT)

Comments of the State of Connecticut, Department of Environmental Protection (CT)

Review of Revised Human Health Risk Assessment for the Housatonic River
Traci Iott
Environmental Analyst III
CT DEP
Bureau of Water Management
April 5, 2005

I have reviewed the revised Human Health Risk Assessment for the Rest of River Portion of the Housatonic River, dated February 2005 and prepared under contract for USEPA as part of the on-going remediation efforts relating to the General Electric facility in Pittsfield, Massachusetts. The portion of the Housatonic River within Connecticut that is considered within this report is designated as river reaches 10 through 16. This is equivalent to the area from the Connecticut/Massachusetts border down to the Derby/Shelton Dam. The area below this dam down to Long Island Sound was not included in the assessment due to the presence of PCBs within this reach of the river from sources other than GE. Two exposure pathways were evaluated within Connecticut: direct exposure to river sediments and fish consumption. Additional scenarios were evaluated for the Massachusetts portion of the river such as exposure to flood plain soils, agricultural products grown in affected soils and consumption of waterfowl. These were not evaluated within Connecticut. Additional chemical constituents were also included in the risk assessments for the Massachusetts portion of the river that were not evaluated within Connecticut.

Direct Exposure to Sediments

The risk assessment concludes that there are no unacceptable risks due to direct contact with river sediments within Connecticut. This is based on 28 data points collected from surficial sediments (0-0.5 ft), with a maximum PCB of 0.47 ppm. Sediment concentrations were compared within the risk assessment with the high-contact sediment screening concentration of 3 mg/kg PCBs as well as the high-contact residential screening criterion of 2 mg/kg. These benchmarks are based on cancer endpoints and assume exposure of children and adults to sediments.

This analysis is slightly different than that which might be conducted under the Connecticut Remediation Standard Regulations (RSRs). These regulations provide two types of criteria for direct contact - residential and industrial/commercial- with the provision made for calculating site-specific criteria as warranted. For a screening evaluation of the data under the RSRs, the use of the residential Direct Exposure Criteria (DEC) for total PCBs of 1 mg/kg is most appropriate in absence of a criterion based on site-specific exposure patterns. The difference between the screening value used in the report and the DEC value are a result of different target cancer risk levels, different assumptions regarding exposure frequency and the consideration of soil adherence factors and exposure surface contact areas within the risk assessment. The maximum PCB concentration in the dataset used in the report is below the Connecticut Residential DEC.

RESPONSE CT-1:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

The dataset used in the risk assessment represents a fraction of the sediment data available for the river. The advantage of this dataset is that it is relatively recent data. The historical dataset spans many years. A brief review of data collected within the past 10 years indicates that most PCB sediment concentrations are below 1 mg/kg in both surficial and deeper sediments but that there are data points greater than 1 mg/kg PCB.

There are, however, deficiencies associated with the data set used in the risk assessment. The portion of the river within Connecticut evaluated within the risk assessment is 72 miles in length. The level of sampling data available is equivalent to one sample for every 2.5 miles of river. This is inadequate to accurately define the nature and extent of contamination within Connecticut and identify localized areas of elevated PCB concentrations.

RESPONSE CT-2:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

Additionally, Section 3.1.2.7 (Connecticut Sediment Sampling) of Volume 1 indicates that the number of samples analyzed for grain size had to be reduced since it was difficult to obtain samples of sufficient size due to large grain size (cobbles/boulders). From this description of the sampling locations, it is unlikely that the sediment data used to evaluate risks were collected from depositional areas that would most likely have retained PCBs. Therefore, use of the current dataset for surficial PCB concentration may underestimate surficial sediment concentrations within Connecticut.

RESPONSE CT-3:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

Sediment samples collected in Connecticut were analyzed for total PCBs. However, a variety of other compounds were included in the risk assessment for the Massachusetts portion of the river. These compounds were evaluated in addition to total PCBs, using a Toxic Equivalence (TEQ) approach. The report concludes that risks from TEQ under direct contact scenarios are similar to risks from total PCBs and that overall risk is a sum of these two categories. Given the lack of

data for TEQ within Connecticut, the potential risks associated with these substances are a source of uncertainty when evaluating the overall risk implications for exposure to contaminated sediments within the Connecticut portion of the Housatonic River.

RESPONSE CT-4:

The commenter is incorrect in stating that a variety of compounds other than PCBs were included in the quantitative direct contact risk assessment in the Massachusetts portion of the River. Although other compounds were analyzed for and detected in the sediment samples collected in Massachusetts, only total PCBs (tPCBs), dioxins, and furans were retained for quantitative assessment after a risk-based screening analysis.

Risks from tPCBs in soil and sediment were evaluated in the risk characterization. The risk from TEQ was addressed in the uncertainty analysis, based on the general recreation scenario (soil), and two risk estimates (tPCB and TEQ) were presented for several concentrations of tPCB. This approach did NOT assume additivity of the two risks, as this comment suggests, for this or any other pathway.

The TEQ concentrations included in the calculations were based on the regression analysis described in Volume I, Attachment 2 because only 10% of the soil samples were analyzed for PCB congeners, dioxins, and furans. It is uncertain if the same regression equations are appropriate for sediment, and no analysis of the impact of including TEQ in direct contact sediment risk was presented in the HHRA. The results of the uncertainty analysis for the general recreation exposure scenario indicate that, if cancer risk were evaluated by the TEQ approach (using the CSF from Health Effects Assessment Summary Tables [HEAST] of 1.5E+05) rather than the tPCB approach, the risks would be 4.6 times higher at tPCB concentrations of 1 mg/kg, 1.9 times higher at 10 mg/kg, 1.3 times higher at tPCB of 50 mg/kg, and slightly lower than the tPCB calculated risk at 100 mg/kg.

Finally, the evaluation of risks from direct contact focused only on surficial sediments. While exposure to surficial sediments will address current potential exposure concentrations, it will not address potential future exposure concentrations. This issue was to be addressed within the revised risk assessment with the consideration of potential future uses/exposures within the Housatonic River Basin. The revised risk assessment, however, did not address this issue for sediments.

RESPONSE CT-5:

EPA agreed to revisit the reasonably foreseeable future use scenarios for floodplain properties in response to comments from the Peer Review Panel. However, none of the Panel commented on the need to revisit future uses for sediment exposures.

The issue of potential future exposures is tied directly to the level of PCBs in deeper sediment layers in depositional areas and behind dams on the river. Future uses of the river in Connecticut may include a variety of activities that could mobilize the deeper sediments; potentially reintroducing elevated PCB levels into surficial sediment horizons. These concentrations could then affect exposures from both direct contact as well as from fish and waterfowl consumption. In order to adequately characterize risks associated with PCBs within the Connecticut portion of the river, potential future risks from the mobilization of PCBs in bedded sediments must be evaluated. This will require the collection and evaluation of a more robust data set for sediment PCB concentrations.

RESPONSE CT-6:

For the purposes of the risk assessment, EPA believes that sufficient data were obtained to determine that there is unacceptable risk associated with consumption of fish from the Housatonic River in CT. How various remedial alternatives would address unacceptable risks, and/or releases that may cause contaminated sediment to become available for human or ecological exposure from possible future activities (such as dam maintenance and/or removal), will be considered in the Corrective Measures Study (CMS) process.

The risk assessment does indicate that nine samples were collected in association with the dams and that PCBs were evaluated to a depth of three feet. However, this data is not presented or used within the risk assessment. Additionally, the report indicates that sufficient sample mass could not be gathered from several locations, indicating that the samples were not likely collected from depositional areas and may not be representative of bedded sediment PCB concentrations.

RESPONSE CT-7:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

As evaluation of risks and remedial needs are considered for the Housatonic River, I recommend the application of the Connecticut Residential Direct Exposure Criteria (DEC) as the goal for PCBs in Connecticut for soils or sediments that people are or could be exposed to or that could migrate to areas where exposure could occur. This recommendation is based on the large area included in the study area for Connecticut and the acknowledgement that a wide range of land uses occurs within this area from remote portions that are not easily accessed to portions of the river that flow through towns and residential areas. CT DEP must approve any modifications to the residential DEC prior to establishing an alternative acceptable level for PCBs in sediments. Application of the residential DEC to the river requires that the 95^{th} upper confidence level of the mean of sediment PCB concentrations for the river must equal 1 mg/kg PCB or less. Connecticut uses an acceptable risk level of 1 in 1,000,000 for cancer endpoints and a hazard

index of 1 for non-cancer endpoints. The risk level used by Connecticut is more restrictive than that used by EPA and should be followed within Connecticut.

RESPONSE CT-8:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

Fish Consumption

A traditional risk assessment approach to evaluating risks associated with consumption of fish is presented in the risk assessment. Both cancer and non-cancer endpoints are evaluated. This approach is acceptable for evaluating current risks at the site. The report indicates that unacceptable risks do exist regarding fish consumption, supporting the current fish consumption advisory. However, as we move through the process of evaluating what actions need to be taken to restore the Housatonic River to a condition where Water Quality Standards and Designated Uses are achieved, it is important to understand the differences between the risk assessment process used in the current document and those employed by the Connecticut Department of Public Health (CTDPH) to evaluate the need for fish consumption advisories due to PCBs. Remedial goals established to insure restoration of the fishery should be based on the health guidance that is at least as stringent as that established by CTDPH.

RESPONSE CT-9:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

The current fish consumption risk assessment includes a discussion of current and traditional fish cooking and consumption practices of the Schaghticoke Tribal Nation within Connecticut. This discussion is presented within the evaluation of uncertainties presented in Section 7 of Appendix C of the revised risk assessment. The presentation in the report filled a major gap in the previous risk assessment documents and identifies greater risk associated with traditional tribal practices. These practices must be considered as remedial goals and activities are identified for the river.

However, the revised risk assessment is deficient in its consideration of subsistence fishing exposures within Connecticut. Section 8.6.3.1 of Volume 1 of the risk assessment indicates that EPA searched for and did not find evidence of any subsistence fishing populations within either Massachusetts or Connecticut. The report indicates that risks to subsistence populations would be higher than those predicted within the current report.

The CTDEP is concerned about subsistence fishing within Housatonic Basin. There is sufficient concern for these populations that CTDEP has translated fishing advisories, signs and informational videos on fish consumption advisories into several different languages to reach

populations that may consume greater amounts of native caught fish. This concern is supported by the findings of a creel survey conducted by the CTDEP during the late 1980s as well as a Connecticut fish consumption survey published in 1999 by Ms. Nancy C. Balcom *et al* and referenced within the revised risk assessment. The study entitled Quantification of Fish and Seafood Consumption Rates for Connecticut was funded in part by the CTDEP. The fish consumption survey evaluated subsistence fishing within the low-income community. Additionally, the cultural practices of several different ethnic groups should be considered within the subsistence fishing group. The Balcom report identified mean consumption rates for these groups ranging from 43.1 g/day for limited income populations to 59.2 g/day for Southeast Asian populations. These consumption rates are greater than the central tendency and high-end consumption rates of 8.7 and 31 g/d used in the revised risk assessment. The data presented in the Balcom report must be used to evaluate risks to subsistence populations such as the limited income and Southeast Asian populations identified in the CT fish consumption rate study.

RESPONSE CT-10:

The data from the study conducted by Balcom et al. (1999) were reviewed during the preparation of the HHRA to determine whether they were appropriate to use in developing a site-specific fish consumption rate; they were judged inadequate for this purpose. The primary objective of the Balcom study was to survey consumption of saltwater fish from both recreational and commercial sources, and thus much of the focused effort was directed toward saltwater anglers. Collection of data on freshwater fishing was a secondary goal.

The mean fish consumption rates of 43.1 g/d for limited income populations and 59.2 g/d for Southeast (SE) Asian populations identified in the Balcom et al. report are based on all sources of seafood, including not only non-commercially obtained saltwater and freshwater species, but also purchased fish and processed fish products such as fish sticks. For example, Table 10 in Balcom et al. (1999) indicates that, on average, the limited income population purchased 30.1 meals/year and caught 9.8 meals/year. Similarly, the SE Asian population purchased 32.9 meals/year and caught 8.8 meals/year. The central tendency fish consumption rate in the HHRA, 8.7 g/d, translates into fourteen 8-oz meals/year, which is higher than the mean number of caught meals/year reported by Balcom et al. (1999) for the limited income and SE Asian populations. Therefore, for the central tendency receptor, the fish ingestion rate used in the HHRA is protective of both the limited income and SE Asian populations.

The highest number of caught meals/year reported by Balcom et al. (1999) was 156 for the limited income population and 78 for the SE Asian population. Again, these catch rates include both saltwater and freshwater fish. The Maine Angler Survey data that provide the basis of the fish consumption rates in the HHRA indicated a maximum consumption rate of 182 g/d, or approximately 300 meals/year (ChemRisk, 1992; Ebert et al., 1993). This is nearly two times the maximum meals/year reported for the limited income population, and nearly four times the maximum meals/year reported for the SE Asian population in the Balcom et al. (1999) study.

In the HHRA, the reasonable maximum exposure (RME) fish ingestion rate is fifty 8-oz meals/year of freshwater fish caught in the Housatonic River, which would be equivalent to fifty-seven 7-oz meals/year, the portion size reported by Balcom et al. (1999) for the SE Asian population. Because Balcom et al. do not report freshwater fish consumption separately, it is not possible to derive a comparable RME ingestion rate for freshwater fish from their data. However, they do present data on fish species consumed, which suggest that a substantial fraction, if not a majority of meals, consist of saltwater species. The maximum number of meals of non-commercially caught freshwater fish and saltwater fish **combined** (78 meals/year) for the SE Asian population is only 25% higher than the RME for freshwater consumption, and the consumption pattern appears to be dominated by saltwater species; therefore, the ingestion rate used in the HHRA will be protective of the SE Asian population. The fish species consumption pattern for the 937 limited-income individuals surveyed by Balcom et al. (1999), a group that included children and may overlap with the SE Asian population, also appears to be dominated by saltwater species; therefore, the assumptions used in the HHRA will similarly be protective of this group.

Fishing Frequency (days/year)

	median	90th percentile	maximum
Maine Angler	17	52	330
CT limited income	9	104	312
CT SE Asian	3	18.5	104

As shown in the table above, the fishing frequency data reported by Balcom et al. (1999) are consistent with the meal consumption data, indicating that the SE Asian population fishes and consumes fish at a frequency less than the Maine anglers that form the basis of the consumption rate in the HHRA. For the limited income population, the central tendency of fish meals and fishing frequency and the maximum number of meals and fishing frequency are similar to or lower than those obtained in the Maine Angler Survey. The 90th percentile of the fishing frequency for limited income anglers, which is subject to the most uncertainty, may or may not be consistent with the Maine angler data. However, the limited income angler data include both saltwater and freshwater angling, and the species data indicate that the fishing frequency is dominated by saltwater angling. This strongly suggests that the ingestion rate used in the HHRA will be protective of the limited income anglers in CT identified in the Balcom et al. (1999) study.

In addition to consumption rates, the Balcom et al. study (Table 16) indicates that the SE Asian population consumes parts of the fish other than fillets, the basis of the main HHRA assessment. However, the traditional food preparation method evaluated for the Schaghticoke (whole fish minus the head), combined with the

central tendency exposure (CTE) and RME fish consumption rates, provides a health-protective prediction of the risks associated with fish consumption for the SE Asian population (see HHRA Volume IV Section 7.2.4.2).

References:

Balcom, Nancy C., Constance M. Capacchione, and Diane Wright Hirsch. 1999. *Quantification of Fish and Seafood Consumption Rates for Connecticut.* Prepared for the Connecticut Department of Environmental Protection, Office of Long Island Sound Programs.

ChemRisk. 1992. *Consumption of Freshwater Fish by Maine Anglers*. 24 July 1992.

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. *North American Journal of Fisheries Management* 13:737-745.

Waterfowl Consumption

The revised risk assessment evaluates potential risks to people who harvest and consume waterfowl from the Housatonic River within Connecticut within the evaluation of uncertainties presented in Section 7 of Appendix C. This risk assessment, however, is based on modeled concentrations of potential PCB concentrations in duck tissues based on sediment concentrations. CTDEP has only one data point for PCBs in ducks collected from the Housatonic River. This data point had PCB concentrations greater than those modeled within the risk assessment. However, it was excluded from the revised risk assessment.

Samples of tissues from waterfowl must be obtained from the Housatonic River watershed within CT and evaluated in order to provide a more accurate estimate of potential risks associated with waterfowl consumption. CTDEP Wildlife Division has submitted a proposal for funding of a study of pesticide and PCB concentrations in waterfowl from the Housatonic and Quinnipiac Watersheds. If this study is funded and conducted, we will share the information with EPA.

RESPONSE CT-11:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

Summary

The revised Human Health Risk Assessment GE/Housatonic River Site Rest of River represents a substantial level of effort from both EPA and their contractors. However, several issues must be addressed to provide an accurate assessment of risks to people within the Housatonic River

watershed within Connecticut. First, the datasets for sediment PCB concentrations, both in surficial and deeper sediment horizons must be expanded to include an adequate amount and quality of data from depositional areas. Additionally, the more conservative CT Direct Exposure Criteria (Residential Exposures) should be used to screen the data, in recognition of the wide variety of land uses that occur within the watershed and not explicitly evaluated within the revised risk assessment.

RESPONSE CT-12:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

Finally, the evaluation of risks associated with fish consumption must be expanded to include subsistence fishers. The fishing habits of both subsistence fishing populations and the traditional practices of the Schaghticoke Tribal Nation must be considered as the remedial process for the restoration of the river proceeds.

RESPONSE CT-13:

Please see Response CT-10.

COMMENTS OF THE GENERAL ELECTRIC COMPANY (GE)

Comments of the General Electric Company (GE)

Comments of General Electric Company on the Human Health Risk Assessment for the General Electric/Housatonic River Site, Rest of River (February 2005 Draft)

Prepared by
AMEC Earth and Environmental Inc.
15 Franklin Street
Portland, ME 04101
(207) 879-4222
and
BBL Sciences, Inc.
Houston, TX
On Behalf of
General Electric Company
Pittsfield, MA
April 2005

1.0 INTRODUCTION

The General Electric Company (GE) is providing these Comments to the U.S. Environmental Protection Agency (EPA) on the February 2005 draft of EPA's *Human Health Risk Assessment for the General Electric/Housatonic River Site, Rest of River* (HHRA) (EPA, 2005). These Comments were prepared on GE's behalf by AMEC Earth & Environmental and BBL Sciences.

The revised HHRA contains a substantial amount of new information and analyses that were not presented in, or have been changed from, the June 2003 draft of the HHRA (EPA, 2003). Some of these additions and changes were intended to address comments made by the peer reviewers on the prior draft, while others were made by EPA on its own initiative.

RESPONSE GE-1:

EPA does not agree that changes were made to the HHRA "on its own initiative." All revisions to the June 2003 HHRA were made either directly or indirectly in response to comments received from the Peer Review Panel, and EPA carefully ensured that all changes were related to either general or specific comments from the Panel.

These Comments focus only on such new or changed information and analyses. Moreover, these Comments address only some of the new or changed material in this revised draft. However, GE adheres to and preserves its positions on all points set forth in GE's prior comments (AMEC and BBL, 2003; GE, 2003) on the June 2003 draft HHRA, and reserves the right to raise those points in any future proceeding. In addition, lack of comment herein on other new material or analyses in the HHRA does not necessarily indicate GE's agreement with such material and analyses; GE reserves the right to present any arguments relating to such material and analyses in an appropriate future proceeding.

These Comments contain the following sections:

- Section 2 discusses certain general points in the revised HHRA – namely: (a) its estimation of population sizes for potentially exposed populations; (b) its discussion of the cancer incidence study conducted by the Agency for Toxic Substances and Disease Registry and the Massachusetts Department of Public Health (ATSDR/MDPH); and (c) its application of the Toxicity Equivalency (TEQ) approach to PCBs.

RESPONSE GE-2:

The EPA responses to the summary GE comments in the three topic areas delineated above are provided below in the detailed responses to Section 2 of the GE comments.

- Section 3 addresses the direct contact assessment. It shows that a number of the changes that EPA has made to the exposure frequencies in the deterministic analyses are not consistent with site characteristics or the available data. It also shows that the new probabilistic analyses added by EPA contain an unwarranted degree of conservatism in some of the distributions used and that its probabilistic model may contain an error in the calculation of a surface area-weighted adherence factor.

RESPONSE GE-3:

The EPA responses to the summary GE comments concerning the direct contact risk assessment are provided below in the detailed responses to Section 3 of the GE comments.

- Section 4 addresses the fish and waterfowl consumption assessment, focusing on problems with the new adult and child fish consumption rates used in the deterministic analysis and the child consumption rates used in the probabilistic analysis.

RESPONSE GE-4:

The EPA responses to the summary GE comments concerning the fish and waterfowl consumption risk assessment are provided below in the detailed responses to Section 4 of the GE comments.

- Section 5 addresses the agricultural products consumption assessment. It notes the speculative nature of some of the HHRA's assertions regarding future agricultural use of the floodplain. It also shows that several of the revised factors used in the deterministic analyses are not justified, and that the probabilistic analyses fail to adequately account for the variability and uncertainties in the risk estimates.

RESPONSE GE-5:

The EPA responses to the summary GE comments concerning the agricultural products consumption risk assessment are provided below in the detailed responses to Section 5 of the GE comments.

- Finally, Section 6 discusses two points in EPA's new integrated risk characterization chapter – the perspective on TEQ exposures and the evaluation of the breast milk pathway. It shows that the revised HHRA does not adequately address the uncertainties in these evaluations.

RESPONSE GE-6:

The EPA responses to the summary GE comments concerning TEQ exposures and the breast milk pathway are provided below in the detailed response to Section 6 of the GE comments.

In each of these sections, GE presents recommendations for further changes to the final HHRA to make it more scientifically supportable.

2.0 GENERAL

This section addresses three general points relating to the revised HHRA. First, it shows that, in estimating population sizes for potentially exposed populations, the HHRA presents some estimates that are not representative of the size of the user population being evaluated in the risk assessment. Second, it shows that the HHRA needs to clarify its discussion of the cancer incidence study that was conducted by the ATSDR/MDPH (2002). Third, it shows that the revised HHRA has not fully or adequately addressed the problems with the application of the TEQ approach to PCBs.

RESPONSE GE-7:

The EPA responses to the three general GE comments summarized above are provided below in the detailed responses to Sections 2.1 through 2.3 of the GE comments, Responses GE-8 through GE-10, respectively.

2.1 Population Estimates

In response to comments received from the peer reviewers, EPA has added an estimation of population sizes for each potentially exposed population. In some cases, however, the general population estimates presented in the revised HHRA do not reflect the size of the user populations being evaluated in the risk assessment. For example, the HHRA estimates that the population of recreational anglers using the Housatonic River is 11,371 individuals (Vol. I, p. 133). While that may be a reasonable estimate of the size of the total user population, it is not the size of the subpopulation of anglers (non-sharing consumers) for whom exposure and risk have been estimated, and in particular is not the size of the subpopulation represented by EPA's Reasonable Maximum Exposure (RME) fish consumption scenario. Thus, it is misleading to present that estimate as the size of the population that may be subject to the levels of potential exposure and risk that are calculated in the HHRA.

The HHRA bases its assumed fish consumption rates for both the RME and central tendency (CTE) risk estimates on data from the Maine angler survey (Ebert et al., 1993) for the fraction of anglers who consume sport-caught fish but do not share their fish with anyone (Vol. IV, p. 4-48). However, the data from that survey indicate a number of differences in fishing and fish

consumption behavior between that subpopulation and the total population of Maine anglers. For example, the average amount of fish harvested per fishing trip was 266 grams/day for non-sharing consumers and 413 grams/day for all other fish consumers. Moreover, the 95th percentile "all waters" consumption rate derived by EPA for non-sharing consumers (31 g/day) is higher than that for all fish consumers (26 g/day). Consequently, the assumptions used to estimate potential risks due to fish consumption can only be considered representative of the behaviors of a small subpopulation of anglers who use the river (i.e., non-sharing consumers).

According to the Maine angler survey, 138 individuals who consumed fish during the one-year survey period did not share their fish with anyone else. These represented 8.56 percent of the total survey population of 1,612 individuals. Applying this percentage to EPA's total estimated user population of 11,371 anglers results in a total population of non-sharing consumers of 973 individuals. Since the CTE risk estimates can be considered representative of the average for the total population, the population size for the CTE risk analysis is estimated to be 973 individuals. However, the RME analysis uses the 95th percentile consumption rate for this subpopulation (Vol. IV, p. 4-48) and thus is representative of approximately 0.43 percent of the total user population for the river (8.56 percent times 5 percent). Applying this percentage to the total of population of 11,371 anglers estimated by EPA results in an estimated RME population size of 49 individuals.

The size of the potentially affected population would be larger, but still considerably smaller than EPA's estimate, even if EPA were correct that the non-sharing consumers were representative of the entire population of fish consumers in the Ebert et al. (1993) survey. In that survey, 1,053 of the 1,612 individuals who responded to the survey indicated that they consumed sport-caught fish from any source during the one-year survey period. Thus, 65 percent of the licensed anglers surveyed actually consumed fish during the year. Applying this fraction to EPA's estimate of 11,371 individuals in the entire Berkshire County angler population results in an estimated population size of 7,391 individuals. It can be considered that the CTE estimate predicted by EPA might be representative of this population. The RME analysis, however, selects the 95th percentile consumption rate and thus is only representative of 5 percent of the consumer population. This results in an estimated population size for the RME analysis of 370 individuals.

Similarly, EPA estimates that there is a population of 3,600 individuals who live in Berkshire County and hunt waterfowl from the primary study area (PSA) (Vol. I, p. 1-35). While GE previously recommended that EPA include a factor to adjust for the fraction of ducks harvested that are non-resident birds (AMEC and BBL, 2003), EPA is instead basing its waterfowl risk estimates only on consumption of resident birds and the PCB concentrations measured in them. To estimate potential exposures to waterfowl consumers, the HHRA assumes that the CTE waterfowl hunter consumes 5.4 meals of resident waterfowl from the PSA each year (Vol. IV, p. 4-84) and that the RME waterfowl consumer ingests 11 waterfowl meals annually from the PSA. EPA assumes that each waterfowl meal is composed of one duck (Vol. IV, p. 4-84). At the same time, however, the HHRA estimates that the size of the resident duck population in the PSA (upon which the exposure point concentration is based) is 120 ducks (Vol. IV, p. 7-14). If there are only 120 resident ducks present in the area, 3,600 waterfowl hunters cannot average even one meal of such ducks during the season. Thus, the ingestion rates used for waterfowl cannot be considered representative of the total waterfowl hunter population estimated by EPA.

Assuming that there are 120 resident ducks in the PSA each year, and using reasonable but conservative assumptions about nesting success, clutch size, and fledging success, one can estimate that there could be as many as 150 resident ducks available for harvest each year without adversely affecting the resident duck population.[1] Based on that total, the CTE consumption rate of 5.4 ducks/year would only allow for an estimated exposed population of 28 consumers of such waterfowl in the PSA. Using the RME consumption rate of 11 ducks/year, it would only be possible for 14 waterfowl hunters to be exposed at the dose rate estimated for the RME. These are likely to be more realistic estimates of the sizes of the potentially exposed waterfowl hunter populations that are being evaluated using the exposure assumptions that have been developed for the HHRA.

Estimation of population size is an important exercise to provide perspective for a risk assessment. Risk management decisions need to consider the size of the population that may potentially be exposed at the levels modeled in the risk assessment. EPA's population size estimates are misleading because they imply that all 11,371 anglers and 3,600 waterfowl hunters might have exposures that result in the risk levels estimated in the HHRA, when in fact there are likely to be substantially smaller populations that would experience that potential level of exposure. For this HHRA, it is important to make risk managers aware that the RME exposures estimated for the fish and waterfowl consumption pathways can only be considered representative of between 49 and 370 recreational anglers and approximately 14 waterfowl hunters, respectively.

RESPONSE GE-8:

As noted in the first section of this comment concerning population sizes, the estimates of the fishing and hunting populations given in the HHRA are for the total population in Berkshire County estimated to engage in these activities. The risks associated with these activities include direct contact with sediment and soil during fishing and waterfowl hunting, in addition to consumption of fish and waterfowl.

The HHRA characterizes cancer risks and noncancer health hazards among individuals within the potentially exposed population. In addition to the RME risks, the CTE risks are included to characterize risks associated with average exposure. Further, a probabilistic assessment was conducted that characterizes the risks for a range of exposures. The information on population size provides some perspective, but has no direct bearing on, the calculation of risks for the RME and CTE individual, as required by EPA guidance and the NCP.

[1] If there are 120 resident ducks, there could be as many as 60 breeding pairs. If it is conservatively assumed that half of the nests are successful (MFWP, 2005; Drilling et al., 2002; Evrard, 2000; Greenwood et al., 1995; Hepp and Bellrose, 1995), each breeding pair averages 10 eggs (EPA, 1993), and 50% of the eggs successfully fledge (Drilling et al., 2002), one can estimate that there are a total of 150 new resident ducks available for harvest each year [30 nests * 10 eggs/nest * 5 fledglings/10 eggs = 150 fledglings].

2.2 ATSDR/MDPH Study

In its discussion of the ATSDR/MDPH (2002) study, the HHRA states that, while the residents of the Housatonic River Area (HRA) did not have excessive cancer incidence for the majority of six cancer types evaluated between 1982 and 1994, the occurrence of bladder cancer among males in the city of Pittsfield was elevated during that 13-year time period and was elevated in females in one census tract between 1987 and 1994 (Vol. I, p. 1-43). While that statement is consistent with the findings of the ATSDR/MDPH study, it could be incorrectly interpreted to mean that the increased bladder cancer observed in Pittsfield was due to PCB exposure. This perception would not be accurate.

It is important to expand the discussion to clarify that bladder cancer has not been an endpoint of concern for PCB exposure (ATSDR, 2000). While a number of investigators have evaluated standard mortality ratios for bladder and/or urinary tract cancers in humans exposed to PCBs (Kimbrough et al., 1999; Gustavsson and Hogstedt, 1997; Loomis et al., 1997), the standard mortality ratios calculated in these studies have not been reported to be significant. Thus, while ATSDR's regional study may have observed an elevation in bladder cancer in Pittsfield, it cannot be concluded that this elevation is related to PCB exposure. In fact, the ATSDR/MDPH (2002) report concluded that the "[r]eview of the available risk factor information related to cancers that were elevated in the city of Pittsfield suggests that cigarette smoking played a role in the increased rates of male bladder cancer" (p. 30). EPA should clarify this issue to avoid misunderstanding by the public.

RESPONSE GE-9:

EPA agrees that the elevated occurrence of bladder cancer in Pittsfield noted in the ATSDR/MDPH study should not be interpreted to suggest that PCBs are the cause of this elevation. EPA's synopsis of the results of this study, as presented in Section 1.7.1.1 of the revised HHRA, is accurate, and neither states nor implies that the reported bladder cancers are related to PCB exposure. Reported risk factors for bladder cancer include cigarette smoking, arsenic ingestion (via drinking water), and certain occupational exposures. Individual chemicals associated with cigarette and/or occupational exposures include aromatic amines such as benzidine and beta-napthylamine, benzidine-based dyes, polycyclic aromatic hydrocarbons (coal tar pitch volatiles), and nitrosamines (NTP, 2004). Benzidine and beta-napthylamine are considered known human carcinogens that cause bladder cancer (IARC, 1987; NTP, 2004). PCBs have not been associated with bladder cancer in occupational studies or animal bioassays (ATSDR, 2000).

In June 1989, the Massachusetts Department of Public Health (MDPH) and the Massachusetts Department of Labor and Industries reported the results of a study of bladder cancer and employment in the Pittsfield area. The study was conducted to evaluate the excess incidence of bladder cancer among male residents of Pittsfield identified in a review of 1982-85 data from the Massachusetts Cancer Registry. Interviews were conducted with male bladder cancer cases or their next-of-kin to obtain detailed work histories, smoking and

residential information. The study design was intended to generate hypotheses about plausible occupation risk factors for bladder cancer, not to identify causal associations. The results suggested that occupational exposure, in addition to smoking, could play a role in the excess bladder cancer cases. Although more than half the bladder cancer cases listed General Electric as their regular employer, contact with PCBs did not emerge as one of the plausible hypotheses to explain the elevated bladder cancer rates among males.

References:

ATSDR (Agency for Toxic Substances and Disease Registry). 2000. Toxicological Profile for Polychlorinated Biphenyls.

ATSDR/MDPH (Agency for Toxic Substances and Disease Registry/ Massachusetts Department of Public Health). 2002. *Health Consultation, Assessment of Cancer Incidence Housatonic River Area, 1982-1994*. Massachusetts Department of Public Health, Bureau of Environmental Health Assessment, Community Assessment Unit. Under a Cooperative Agreement with the Agency for Toxic Substances and Disease Registry.

IARC (International Agency for Research on Cancer). 1987. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs, Volumes 1 to 42. Supplement 7. Lyon, FR.

Massachusetts Department of Public Health and Massachusetts Department of Labor and Industries. 1989. Bladder Cancer and Employment in the Pittsfield Massachusetts Area II: A Follow-up Survey of Bladder Cancer Cases. June 1989.

NTP (National Toxicology Program) Public Health Service, U.S. Department of Health and Human Services. 2004. *Report on Carcinogens*, Eleventh Edition.

2.3 Use of TEQ Approach in Toxicity Assessment

In response to comments received during the peer review process, EPA has revised its application of the TEQ approach to correct for the double-counting of potential risks due to PCBs, which was involved in EPA's previous approach. At the same time, however, EPA has retained its application of the TEQ approach to PCBs (in the main fish and waterfowl consumption assessment and in the sensitivity analyses for the direct contact and agricultural products consumption assessments) despite the substantial comments that were provided both by GE and by peer reviewers showing the flaws in application of the TEQ approach to PCBs. In addition, since the time of the peer review, there have been a number of new developments that have further undermined the appropriateness of the application of the TEQ approach to PCBs. These developments are summarized in a March 15, 2005 letter from GE to Dr. David Eaton, Chair of the National Academy of Sciences (NAS) Committee reviewing EPA's draft Dioxin Reassessment, a copy of which is attached (without its attachments) as Attachment A.

This more recent information confirms that the TEQ approach for PCBs is not appropriate and substantially overestimates potential risks due to PCBs. Consequently, GE again recommends that EPA altogether eliminate the TEQ analysis for "dioxin-like" PCBs from the final HHRA.

RESPONSE GE-10:

The approach to evaluating the toxicity of PCBs in the February 2005 HHRA is responsive to the comments of the Peer Reviewer Panel on the June 2003 draft. One reviewer commented "Calculation of TEQs is an acceptable method of integrating risks associated with exposure to a mixture of PCDDs, PCDFs, and PCBs that have dioxin-like properties." Another reviewer suggested that EPA "re-evaluate the necessity of using the TEQ approach in this HHRA." Four of the seven peer reviewers commented that the approach used to subtract expected TEQ from an Aroclor mixture from the measured or predicted TEQ at the site was confusing or incorrect. This methodology was eliminated in the revised HHRA. EPA revised the TEQ approach used in the risk assessment by eliminating the subtraction of expected TEQ toxicity and instead avoided "double counting" TEQ by not summing the risks from the potency-adjusted dose addition method (TEQ) and sufficiently similar mixtures (PCB) approaches (described below).

One peer reviewer recommended "the TEQ method and the associated use of the cancer slope factor for dioxin not be used in the Housatonic River Human Risk Assessment until the National Academy of Sciences/National Research Council issues its report and recommendations." This comment did not question the applicability of the TEQ approach, but rather reflected the controversy over the slope factor for TCDD. The revised HHRA bases the risk assessment for TEQ on the TCDD slope factor published in HEAST (EPA, 1997), rather than the slope factor in the Dioxin Reassessment (EPA, 2000), and includes a discussion of the Dioxin Reassessment in the uncertainty sections of the document.

In the revised HHRA, cancer risks from PCBs were evaluated using two complementary approaches, one based on the cancer slope factor (CSF) of commercial PCB mixtures and the other based on the sum of the TEQ from PCBs measured in the environmental samples. Cancer risks from the tPCB and TEQ approaches were presented separately, and were not summed. As discussed in the HHRA, this approach has the advantage of fully presenting cancer risks from two complementary toxicological evaluations, and also avoids potential "double-counting" that may result from summing the two risk values, although either individual risk estimate alone may not fully quantify the carcinogenic risk of the PCB, dioxin, and furan mixture at the site. The approach used in the HHRA is consistent with EPA guidance for chemical mixtures (EPA, 1986; 2000).

The 1986 *Guidelines for the Health Risk Assessment of Chemical Mixtures* (EPA, 1986) recommends three approaches to quantitative health risk assessment of a chemical mixture, depending upon the type of data available. In the first approach, when data on the toxicity of the mixture of concern are available, quantitative risk assessment is performed with these data. In the second

approach, when toxicity data are not available for the mixture of concern, the Guidelines recommend using toxicity data for a "sufficiently similar" mixture. If the mixture of concern and the proposed surrogate mixture are judged by the risk assessor to be similar, then the quantitative risk assessment for the mixture of concern may be derived from health effects data on the similar mixture. The third approach is to evaluate the mixture through an analysis of its components, e.g., using dose addition for similarly acting chemicals and response addition for independently acting chemicals. These procedures include a general assumption that interactions at low dose levels either do not occur or are small enough to be insignificant to the risk estimate. The Guidelines recommend the incorporation of data on interactions when available, if not as part of the quantitative process, then as a qualitative evaluation of the risk.

In August 2000, EPA published *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures* (EPA, 2000) that builds on the principles and approaches presented in the 1986 Guidelines. The Supplementary Guidance contains the following recommendations regarding evaluation of the cancer risk posed by PCBs (page 65):

> "Because PCBs can cause cancer through both dioxin-like and non-dioxin-like modes of action, it is important to consider the contribution from both dioxin-like and non-dioxin-like modes of action to the total risk. Risks for the dioxin-like and non-dioxin-like portions of the mixture are calculated separately. For the dioxin-like portion, a relative potency approach is used. The dose of each dioxin-like congener is multiplied by its toxic equivalency factor, then these products are summed to obtain the total dioxin toxic equivalents present in the PCB mixture. This, in turn, is multiplied by the dioxin slope factor to estimate the risk from dioxin-like modes of action. For the non-dioxin-like portion, a similar-standard-mixture approach is used. The total dose of PCBs, less the dose comprising the 13 dioxin-like congeners already considered, is multiplied by the appropriate PCB slope factor as determined in the previous section."

The two complementary approaches used in the revised HHRA – evaluating the PCB mixture and the potency-adjusted dose addition method – are consistent with this guidance.

The GE comment suggests that recent information confirms that the TEQ approach for PCBs is not appropriate and substantially overestimates potential risks due to PCBs. However, data indicating that PCBs have dioxin-like activities, as well as data confirming the TEFs continue to accumulate. For example, as noted in Section 4.2.2.2 of the HHRA, a series of 2-year bioassays conducted by the NTP (2004a,b,c,d) to evaluate the chronic toxicity and carcinogenicity of dioxin-like compounds and structurally related PCBs and

mixtures of these compound has been published. The conclusions of these studies were:

- 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) – clear evidence of carcinogenicity.
- 3,3',4,4',5-Pentachlorobiphenyl (PCB-126) – clear evidence of carcinogenicity.
- 2,3,4,7,8-Pentachlorodibenzo-furan (PeCDF) – some evidence of carcinogenicity.
- Mixture (PCB-126, 2,3,7,8-TCDD, 2,3,4,7,8-PeCDF) – clear evidence of carcinogenicity.

Tissues from the NTP toxicity tests were provided to researchers to study effects of carcinogenic doses of TCDD, 2,3,4,7,8-PeCDF, and PCB-126 on gene expression in the liver. Similar tests were also conducted on PCB-153, a non-dioxin-like PCB congener (Vezina et al., 2004). The tissues were from female Harlan Sprague-Dawley rats that had been exposed for 13 weeks to toxicologically equivalent doses, based on their toxic equivalency factors, of TCDD, PeCDF, and PCB-126 and a mixture where each congener contributed one-third of the total toxicological dose. PCB-153 was dosed at the same rate as PCB-126. The three dioxin-like compounds produced very similar global gene expression profiles that differed from the profile for PCB-153. These same doses produced, at 13 weeks, similar liver hypertrophy for TCDD and PCB-126; PeCDF produced less severe liver hypertrophy. Two-year exposures to these same doses produced liver tumors in animals exposed to TCDD, PeCDF, and PCB-126. Again, the tumor incidence was higher for TCDD and PCB-126 than PeCDF.

Walker et al. (2005) modeled the dose-response curves for the four tumor types produced in these NTP toxicity tests (NTP, 2004a,b,c,d). They found that the dose-response curve for each tumor type had the same shape for TCDD, PCB-126, and PeCDF and for the mixture of the three, an important consideration when predicting the dose-response of mixtures. They also observed that the number of tumors in the mixture was consistent with a potency-adjusted dose additive effect. For all four tumor types, the potency for PCB-126 relative to TCDD was 0.1, consistent with the WHO TEF value used in the HHRA. For PeCDF, the potency relative to TCDD was 0.5, consistent with the WHO TEF for two tumor types, while for two other tumor types, the relative potency of the PeCDF congener was lower (0.2 to 0.3). These dose-response modeling results are consistent with the gene expression and pathology results described by Vezina et al. (2004).

The GE comment cites new data that are summarized in a March 15, 2005 letter from GE to Dr. David Eaton, Chair of the National Academy of Sciences (NAS) Committee reviewing EPA's draft Dioxin Reassessment. These data have not been peer reviewed or published, and were not available during the preparation of the HHRA, and thus were not considered in its development. However,

because PCB and TEQ cancer risk estimates are presented separately, the HHRA provides the information needed for risk managers to make decisions on the basis of the PCB approach, the TEQ approach, or a combination of these approaches.

References:

EPA (U.S. Environmental Protection Agency). 1986. *Guidelines for the Health Risk Assessment of Chemical Mixtures.* Risk Assessment Forum. EPA/630/R-98/002.

EPA (U.S. Environmental Protection Agency). 1997. Health Effects Assessment Summary Tables. Office of Research and Development, Washington, DC. EPA 540/R-97-036.

EPA (U.S. Environmental Protection Agency). 2000. *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures*. EPA/630/R-00/002. August 2000.
http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=20533

NTP (National Toxicology Program). 2004. TR-520 Toxicology and Carcinogenesis Studies of 3,3',4,4',5-Pentachlorobiphenyl (PCB-126) (CAS No. 57465-28-8) in Female Harlan Sprague-Dawley Rats (Gavage Studies).

NTP (National Toxicology Program). 2004. TR-521 Toxicology and Carcinogenesis Studies of 2,3,7,8 Tetrachlorodibenzo-p-dioxin (TCDD) (CAS No.1746-01-06) in Female Harlan Sprague-Dawley Rats (Gavage Studies).

NTP (National Toxicology Program). 2004. TR-525 Toxicology and Carcinogenesis Studies of 2,3,4,7,8 Pentachlorodibenzo-furan (PeCDF) (CAS No. 57117-31-4) in Female Harlan Sprague-Dawley Rats (Gavage Studies).

NTP (National Toxicology Program). 2004. TR-526 Toxicology and Carcinogenesis Studies of a Mixture of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) (CAS No. 1746-01-06), 2,3,4,7,8-Pentachlorodibenzofuran (PeCDF) (CAS No. 57117-31-4), and 3,3',4,4',5-Pentachlorobiphenyl (PCB-126) (CAS No. 57465-28-8) in Female Harlan Sprague-Dawley Rats (Gavage Studies).

Vezina, C.M., N.J. Walker, and J.R. Olson. 2004. Subchronic Exposure to TCDD, PeCDF, PCB126 and PCB 153: Effect on Hepatic Gene Expression. *Environ. Health Perspect*. 112 (16)1636-1644.

Walker, N.J. P.W. Crockett et al. 2005. Dose-Additive Carcinogenicity of a Defined Mixture of "Dioxin-like Compounds". *Environ. Health Perspect.* 113(1):43-48.

3.0 DIRECT CONTACT ASSESSMENT

EPA has made some changes to its deterministic risk assessment for the direct contact exposure pathways and has added probabilistic analyses of direct exposures using a surrogate soil exposure point concentration (EPC) of 1 ppm. GE believes that a number of the changes made to the exposure frequencies in the deterministic assessment are not consistent with site conditions or the available data. In addition, while GE supports the use of probabilistic analyses to provide additional information about potential risks to individuals involved in direct contact activities, the probabilistic analyses presented in the revised HHRA involve distributions that are unjustifiably skewed high, and its probabilistic model appears to contain errors in either calculation or reporting. GE's recommended changes to the direct contact assessment are discussed below.

RESPONSE GE-11:

The EPA responses to the GE comments summarized above are provided below in Responses GE-12 through GE-18.

3.1 Deterministic Analysis – Exposure Frequencies

There have been changes in the exposure frequencies used in the HHRA for many of the exposure areas (EAs) evaluated. In several cases, however, either the magnitude of the change in the frequency or the basis for the change is not supportable. In this connection, while EPA has considered the Housatonic River Floodplain User Survey (HRFUS) data collected by Triangle Economic Research (TER, 2003), it appears that EPA has been somewhat selective in doing so. In particular, it appears that EPA has used the HRFUS data when they support more conservative assumptions about the frequency of use of individual EAs, but has not reduced the exposure frequencies for some EAs for which the HRFUS data clearly indicate little to no use. GE believes that EPA should revisit the exposure frequency assumptions for the following EAs.

RESPONSE GE-12:

The Housatonic River Floodplain User Survey (HRFUS) was only one of multiple lines of evidence/information that EPA and MDEP used in revisiting the exposure frequencies on a parcel-by-parcel basis in response to Reviewers' comments. The other criteria included the presence of trails or other evidence of use patterns (e.g., campfire ring), observations of use by individuals associated with the project other than those conducting the HRFUS, relative size of the parcel, and proximity to and accessibility from nearby current or future residential properties and/or established recreational areas (e.g., Canoe Meadows Audubon Sanctuary). These lines of evidence and criteria were established and applied with care in response to the Reviewers' comments, thus resulting in the exposure frequencies increasing for some parcels and decreasing for others.

The information from the HRFUS was not used in a biased manner in the revised HHRA. It should be recognized that in a survey of the type and duration of the HRFUS, while observation of use is definitive, the lack of observation of use is not; therefore, information from such a survey should not be used while ignoring

other information. In addition, such a survey cannot reflect the use that would occur in the absence of PCB contamination. That is why EPA and MDEP used other information and criteria, along with the information from the HRFUS, to assign exposure frequencies for individual parcels.

EA 18

EA 18 is a portion of a farm property, which the HHRA considers a potential future residential property (Vol. IIIA, p. 5-36). The revised draft HHRA has increased the assumed exposure frequency for this EA from an RME exposure frequency of 90 days/year and a CTE exposure frequency of 30 days/year to the default residential frequency of 150 day/year for both the CTE and RME analyses (Vol. IIIA, p. 5-37).

EA 18 is subject to an agricultural preservation restriction (APR) and, as such, is not likely to be converted for residential use. Moreover, even if this EA were converted to residential use, the default residential frequency of 150 days/year is not reasonable for portions of this area, which are not accessible. Specifically, only seven of the 14 sampling locations used to derive the EPC for this EA can be accessed without crossing open water (see Vol. IIIB, Figure 5-19). Despite the inability to access seven of the sampling locations without crossing open water, the HHRA designates most of them as "walkable" areas and thus gives them a full weight in the use-area weighting approach used to develop the EPC. Because these inaccessible sampling locations have higher PCB concentrations than the accessible areas, their inclusion in the development of the EPC with a weight of 100 percent inflates the estimate of the EPC for the area that would be likely to be accessed on a regular basis even if the EA were used for residential purposes. This, combined with an unreasonable exposure frequency that includes these areas, results in substantially overestimated exposures and risks for this EA.

To solve this problem, GE recommends that EPA either: (a) reduce the exposure frequency for the overall EA to the frequencies used in the prior draft; or (b) subdivide the EA into two different exposure areas with different EPCs – one that includes all of the land area that could be accessed without crossing open water and the other that includes only those areas that would have to be accessed by boat – and assign a lower exposure frequency to the latter.

RESPONSE GE-13:

EPA was not aware of the agricultural preservation restriction (APR) that exists for EA-18. EPA conducted a significant effort to interview local land use planners and review land use plans, and examined specific zoning restrictions. However, review of the individual deeds for the properties being evaluated was beyond the scope of an HHRA; such a situation would typically be dealt with at the time of the remedial decision and/or implementation. Given the existence of the APR for EA 18, EPA agrees that future residential use is not reasonably foreseeable for this parcel and should not be considered in making remedial decisions affecting this parcel.

EA 42

EPA has reduced the RME exposure frequency for EA 42 from 90 days/year to 60 days/year and has retained the exposure frequency of 30 days/year for the CTE analysis (Vol. IIIA, p. 583). While GE agrees that it is appropriate to reduce the RME exposure frequency for this EA, it believes that the exposure the frequencies for the RME and CTE analyses should be further reduced to 30 and 15 days, respectively, to reflect likely low usage of the entire EA.

This parcel has only a very small walkable section that can be accessed without crossing water. The majority of the EA is wadable/difficult with very thick vegetation. In fact, only two of the 15 sampling points used to derive the EPC were obtained from walkable areas that can be accessed without crossing open water (see Vol. IIIB, Figure 5-42). While EPA has assigned a lower use-weighting factor to those areas, the reality is that it is likely that they will receive no usage except if accessed from the river itself.

Very limited use of the floodplain portion of this EA is also supported by the data collected during the HRFUS. No observations of floodplain use in this area were made from the river during the 60 canoe-based survey days, and during the car-based survey, only six cars were observed parked along October Mountain Road, which is at a substantial distance from the floodplain portion of the EA. Consequently, the recreation activities recorded by TER, which were associated with these parked cars, may or may not have occurred within the floodplain itself.

GE believes that EPA should revise its handling of EA 42 in one of two ways. The first way would be for EPA to subdivide EA 42 so that the area being evaluated as EA 42 only includes the walkable area that can be accessed from land without crossing open water. If this approach is taken, it would be reasonable to use the exposure frequencies of 60 and 30 days/year that EPA is currently using, given the change in the area included in the EPC. Alternatively, if EPA does not further subdivide the parcel, EPA should reduce the exposure frequencies to 30 and 15 days to reflect the limited usage that is likely to occur in the majority of the EA.

RESPONSE GE-14:

See Response GE-12 on the use of the HRFUS.

EA 42 is considered a medium-use area because it is accessible from a trail leading from Roaring Brook Road and via walkable areas from the EA that borders to the north; it is located within ½ mile of about 10 residences; and it is bordered on the south by a residential property. EPA disagrees with the statement that only two of the 15 sample points used to derive the exposure point concentration (EPC) were obtained from walkable areas that can be accessed without crossing open water, and that only a small area is walkable without crossing water. Furthermore, all of the samples collected on the parcel were used and weighted according to the appropriate use-weighting factor (reflecting the accessibility in the area of the sample) when calculating the EPC; therefore, the concentrations for samples located on difficult-to-access areas were discounted by the use-weighting factor. EPA believes that the reduction in

exposure frequency at this area reflected in the revised HHRA is appropriate for the current and future uses.

EA 43

EPA also reduced the RME exposure frequency for EA 43 from 90 days/year to 60 days/year and has retained the former CTE frequency of 30 days/year (Vol. IIIA, p. 5-84). GE supports a reduction in the RME exposure frequency for EA 43 but believes that both the RME and CTE frequency should be reduced further to 30 and 15 days/year, respectively. This is because only a tiny sliver of land is actually walkable and one needs to go down a steep slope to get to it. It is unlikely that this area receives any regular usage.

During 60 days of the canoe-based survey in this area, there were no individuals observed using it. Map 5 of Attachment E of the HRFUS indicates that only one car was observed parked along October Mountain Road during the car survey. The observation point for this automobile was located at a substantial distance from the floodplain portion of the EA. The walking activity recorded by TER associated with the parked car was likely limited to October Mountain Road with no contact with floodplain soils.

The physical characteristics of this EA are very similar to EAs 9 and 29, to which EPA has assigned exposure frequencies of 30 and 15 days for the RME and CTE analyses, respectively. In addition, the HRFUS indicated very little to no use of the area. Thus GE recommends that the exposure frequencies for EA 43 be further reduced to 30 days/year for the RME analysis and 15 days/year for the CTE analysis.

RESPONSE GE-15:

See Response GE-12 on the use of the HRFUS.

EA 43 is considered a medium-use area because it is composed of two residential properties and there is a trail down the hill to the river; it is accessible from Roaring Brook Road; it is located within ½ mile of a number of residences; and it includes not only riverbank but floodplain habitat.

The use-weighting factor explicitly included in the calculation of the EPC accounts for areas of an EA that may receive less use than others. EPA does not believe that it is appropriate to include both a use-weighting factor to effectively "discount" concentrations due to accessibility and to also reduce the exposure frequency beyond what would be assigned for a similar parcel based on the criteria that were used in making the determination of the EF; such an approach would result in a double "discount" of the risk associated with an exposure area.

In addition, EPA does not believe that it is appropriate to compare this exposure area to EA 9 and EA 29 as a means of establishing exposure frequencies. EA 43 is 1.53 acres, whereas EA 9 is 0.04 acres and EA 29 is 0.34 acres. EA 9 represents only the riverbank on a residential parcel, and EA 29 is a portion of a parcel owned by MassWildlife, much of which is also riverbank. EPA believes

that the reduction in exposure frequency at this area is appropriate for the current and future uses.

EAs 45, 46, 48, and 54

EPA has added the general recreation scenario to EAs 45, 46, 48 and 54 and assigned exposure frequencies of 90 and 30 days to them for the RME and CTE analyses, respectively (Vol. IIIA, pp. 5-88, 5-90, 5-94, and 5-105). GE believes that these assigned exposure frequencies are too high, given the physical characteristics of the EAs and the use levels observed during the HRFUS.

Access to a substantial portion of EA 45 is blocked by water or by wadable/difficult terrain. During the 60 days of canoe-based counts in the HRFUS, no individuals were observed using the floodplain portion of this EA. The car-based counts reported a total of 11 cars parked along October Mountain Road in this area. These cars were parked a marked distance from any of the floodplain area and close to residences and a garden, which are located outside of the floodplain along October Mountain Road. Thus, at least a substantial number of these parked cars may have been associated with activities other than recreational activity in this EA.

Much of EA 46 is blocked by open water or is wadable/difficult. During 60 days of canoe-based counts, TER did not observe any individuals engaged in activities within the floodplain. The car-based counts included 5 observations of parked cars and indicated that there were two individuals engaged in waterfowl hunting (which is already being evaluated as a separate exposure scenario for this EA) and three individuals engaged in general recreation. Thus, it appears that the general recreational use of this area is very limited, likely due to the physical characteristics of the EA.

Most of EA 48 is wadable/difficult. During the 60 days of canoe-based counts conducted by TER, no individuals were observed using the floodplain in this EA. The car-based counts indicated that 9 vehicles were observed parked adjacent to the EA along October Mountain Road and that those individuals were engaged in walking, hunting and other recreational activities. Hunters are already being evaluated for this EA. In addition, because of the distance between the parking area along October Mountain Road and the floodplain, as well as the physical characteristics of the EA itself, it is likely that these other recreational activities occurred along October Mountain Road or within October Mountain State Park and resulted in no exposure to floodplain soils.

Except for the boat launch area, EA 54 is wadable/difficult and is not likely to be regularly used except as a boat launch. During 60 days of canoe-based counts, no individuals were observed engaged in floodplain based activities there. A total of nine cars were observed parked in the area during the 60 days of car-based counts. One of these cars was reported to be associated with fishing activity, one with walking, and the other 7 with unknown general recreational activities. It is likely that these 7 cars were associated with the boat launch activity or activities along October Mountain Road or in the adjacent State Forest.

Due to the physical characteristics of these EAs, GE believes that all of these areas should be evaluated as "low use" areas, using exposure frequencies of 30 and 15 days for the RME and CTE analyses, respectively.

RESPONSE GE-16:

See Response GE-12 on the use of the HRFUS.

EAs 45, 46, 48, and 54 are considered high-use areas because they are accessible from October Mountain Road, which is a popular and frequently used area by walkers, runners, dog walkers, hikers, and other recreational users. EPA agrees that open water or wadable/difficult terrain could possibly limit access to EA 45 if access was gained directly from October Mountain Road to the east. However, access is not hampered by open water or wadable/difficult terrain if access is gained from the residential properties to the north or from the southern portion of the EA, the majority of which is walkable. The same situation applies to EA 46. The open water could restrict access to the middle portion of the area, but there are no impediments to access in the walkable southern portion of the area, which is readily accessible from October Mountain Road. In addition, lessening the exposure based upon the same criteria used for establishing the use-weighting factor would doubly discount the risk on the EA based upon the same information. EPA believes that the exposure frequency used for these areas is appropriate for the current and future uses.

EA 55

In the previous draft of the HHRA, EPA evaluated EA 55 using frequencies of 30 and 15 days (for older children and adults, respectively) due to the fact that the area was remote, densely vegetated, and wet. The revised HHRA increases the exposure frequencies to 90 and 30 days (for older children and adults) and adds potential exposure to young children with a frequency of 15 days/year for both the RME and CTE analyses (Vol. IIIA, p. 5-107). The addition of the young child receptor is appropriate given that young children were observed in the EA during the HRFUS. GE believes, however that the revised frequencies assigned to older children and adults are too high, given the conditions reported by EPA for the EA, which have not changed since the 2003 draft.

While EPA has justified this change in frequency based on the fact that it is possible to access this EA from October Mountain Road and the canoe/boat launch to the north (Vol. IIIA, p. 5107), the EA is still not that likely to be heavily accessed for other recreational purposes due to physical conditions of the EA. During 60 days of canoe-based observations in the HRFUS, a total 5 individuals were observed using the floodplain. A total of 23 cars were reported parked along October Mountain Road toward the southern end of EA 55. At this location, however, the river is located at a substantial distance from October Mountain Road across difficult terrain and thus is not likely to be the focus of the recreational activity associated with these cars. This part of October Mountain Road also provides access to trails leading into October Mountain State Forest. Thus it is likely that most of the activities associated with these cars were related to walking/hiking along October Mountain Road or within the State Forest. Neither of these is located within the floodplain of the river.

In light of the physical conditions in this EA and the types of observations made during the HRFUS, GE recommends that revised frequencies of 60 and 30 days be used for the RME and CTE analyses of older children and adults in this EA. GE supports the use of EPA's current frequency of 15 days for young children.

RESPONSE GE-17:

See Response GE-12 on the use of the HRFUS.

The exposure frequency and description referenced by GE for EA 55, which was provided in the draft HHRA, was incorrect; this error was corrected in the revised HHRA. In addition, EPA would like to note that an error remains in the revised HHRA on Figure 5-55 depicting this EA. In this figure, only Waterfowl Hunting and Hunting (non-waterfowl) are listed as the applicable uses; General Recreation should also be listed.

EA 55 is considered a high-use area because it is easily accessible from October Mountain Road paralleling the entire EA to the east, which is a popular and frequently used area by walkers, runners, dog walkers, hikers, and other recreational users; the terrain is not "difficult." In addition, in the HRFUS, people were observed using the EA and a popular canoe launch/parking spot is located immediately to the north. EPA believes that the exposure frequency used for this area, as calculated in the revised HHRA, is appropriate for the current and future uses.

<u>EA 56</u>

EPA changed the recreational frequencies for this EA from 30 and 15 days/year to 60 and 30 days/year, based on the proximity of the EA to a residence and the Woods Pond footbridge (Vol. IIIA, p. 5-109). GE believes that this frequency is too high. In fact, only two older children were observed bicycling through in this EA during 118 observation days of the HRFUS. While there is one residential property nearby, the residential property has its own frontage on Woods Pond and that frontage is likely to be preferentially used by those residents. GE recommends that EPA reassign its former frequencies of 30 and 15 days/year for the RME and CTE analyses, respectively, to EA 56.

RESPONSE GE-18:

See Response GE-12 on the use of the HRFUS.

The exposure frequency for EA 56 was revised to represent a medium-use area because it is a residentially-owned property, and is accessible from an adjacent trail, available parking, and a recreational attraction (Berkshire Scenic Railway). The presence of the railroad track was considered to provide a limited impediment to access. EPA believes that the increase in exposure frequency at this area is appropriate for the current and future uses.

3.2 Probabilistic Assessment

EPA has added probabilistic analyses of the direct contact pathways, including a Monte Carlo Analysis (MCA) and a Probability Bonds Analysis (PBA). In many cases, the upper bound estimates from the MCA predict higher risks than the RME point estimates. This is due largely to the fact that, in that probabilistic analysis, EPA has modified the underlying distributions for certain parameters, adding degrees of conservatism at the outset that are not appropriate. In addition, it appears that EPA may have made an error in its probabilistic model, specifically related to the development of a factor identified as "X," which is a surface area-weighted adherence factor. If an error in calculation was made, the predicted risk results may be incorrect.

RESPONSE GE-19:

The EPA responses to the GE comments summarized above are provided below in Responses GE-20 through GE-23.

3.2.1 Use of Skewed Distributions

For some of the parameters used in the probabilistic analyses, the revised HHRA uses distributions that are unjustifiably truncated at an artificially high minimum value or are otherwise skewed toward higher values, thus producing overestimates of exposure and risk. These parameters are the soil ingestion rates for certain exposure scenarios, the fraction of soil ingested, and the dermal absorption rate for PCBs.

Soil Ingestion Rate

For certain scenarios (young children, hunters, ATV/dirt bikers), the MCA used in the revised HHRA sets the minimum soil ingestion rate at 50 mg/day (Vol. IIIA, p. 6-11). This approach artificially skews the analysis toward higher ingestion rates and does not reflect the underlying data upon which the soil ingestion rates are based. The Stanek and Calabrese (1992) data for soil ingestion, upon which EPA's point estimate soil ingestion rates for young children are based, indicate that, with the exception of the one pica child involved in the study, the rates of ingestion by 1 to 5 year old children ranged from a minimum rate of 5 mg/day to a maximum of 241 g/day, with a median of 37 mg/day and a mean of 54 mg/day. Thus, even for young children, EPA's approach to the soil ingestion input distribution does not reflect the data upon which it is based and will result in substantially biased estimates of exposure and risk. This bias is even more pronounced for older children and adults, who are known to have substantially lower soil ingestion rates than young children.

In addition, for the hunter scenario, the revised HHRA not only sets the minimum soil ingestion rate at 50 mg/day but also sets the maximum soil ingestion rate at 200 mg/day (Vol. IIIA, p. 611), rather than the upper bound estimate of 100 mg/day that is used in the deterministic analysis. The PBA is even more skewed toward high-end soil ingestion because it sets the maximum rate at 300 mg/day (Vol. IIIA, p. 6-11). As support for these maximum values, the HHRA cites the ingestion rate modeled by Hawley (1985) and the results of the Stanek et al. (1992) adult consumption study, which reported one adult with a soil ingestion rate of 331 mg/day (Vol. IIIA, p. 6-11). The Hawley soil ingestion rate has no empirical basis, was not supported by any direct measurements, and is based on a number of assumptions that have since been determined to be unrepresentative of real-life conditions. EPA's *Exposure Factors*

Handbook (1997) describes it as a "conjectural" value. In addition, the 331 mg/day upper bound ingestion rate reported in the Stanek et al. study was reported by those authors to be an unreliable estimate of daily soil ingestion because it reflected three to four days of accumulation rather than a single day of ingestion. One of these authors has since recommended that the 75th percentile of the soil ingestion rate distribution from that study, 49 mg/day, is a more reliable estimate of upper bound soil ingestion by adults (Calabrese, 2003).

For these reasons, GE believes that EPA should revise its probabilistic analyses to be representative of the available data on soil ingestion. For young children, EPA should either directly use the empirical data provided by Stanek and Calabrese (1992) or use a triangular distribution that reflects those data, with a minimum of 5 mg/day, a maximum of 241 mg/day, and a mode of 37 mg/day. For adults and older children, EPA should revise its distribution to have a minimum of 1 mg/day, a maximum of 100 mg/day, and a mode of 10 mg/day, based on the information provided by Stanek et al. (1992).

RESPONSE GE-20:

EPA reviewed data on soil ingestion rates from the scientific literature to define model inputs for the Monte Carlo analysis (MCA) and probability bounds analysis (PBA). For the MCA, EPA defined the variability in soil ingestion rates with a triangular distribution. In the PBA, EPA defined uncertainty with a p-box specified by a minimum value, maximum value, and mode. The minimum and maximum values of the p-box were changed from the minimum and maximum values used in the triangular distributions to account for uncertainty in long-term average soil ingestion rates. This approach is one of several that could be used to define uncertainty and variability in soil ingestion rates, particularly given that there is no consensus on the best way to apportion variability and uncertainty for this parameter. For this reason, EPA conducted a sensitivity analysis showing the effect on risk conclusions of using a different method for estimating variability (see Section 7.2.2.6 of HHRA Volume IIIA).

There are many uncertainties associated with the magnitude of the soil ingestion rate and its variability. These include:

- Measurement methods, including selection of tracers and measuring mass balance in human subjects.
- Activities being conducted by the study subjects and whether they are representative of soil-contact activities evaluated in the HHRA.
- Extrapolation of long-term soil ingestion from short-term studies.

These uncertainties were considered in the development of the distributions for the soil ingestion rate.

EPA recognizes that the scientific literature reports soil ingestion rate measurements less than the 50 mg/day minimum value used in the MCA input distribution. The literature also includes rates that are higher than the 200 mg/day maximum value used in the MCA input distribution. It is difficult to

measure soil ingestion rate, and different rates can be obtained based on the tracer selected and how data from multiple tracers and individuals are combined. EPA selected a range, to one significant figure, that was observed frequently for the measured tracers and combinations. If EPA had defined the minimum soil ingestion rate for the MCA with one of the lowest values reported in the literature, such as the 5 mg/day recommended in this comment, it would also have needed to increase the maximum beyond the 241 mg/day recommended.

The MCA input distribution does not include all reported soil ingestion rates because the distribution is intended to represent potential variability in a long-term average soil ingestion rate. There is uncertainty associated with this approach because there is no consensus among scientists on the degree of this variability. EPA accounts for the fact that long-term rates could be higher or lower than values in the MCA input range by defining probability boxes (p-boxes) with a minimum soil ingestion rate of 0 mg/day and a maximum soil ingestion rate of 300 mg/day. To avoid biasing the triangular distribution upward, EPA used 300 mg/day as the upper bound on its p-box, despite the fact that higher soil ingestion rates have been reported among young children. EPA did not use modeled soil ingestion rates from Hawley (1985) as the empirical basis for this upper bound as this comment indicates. EPA clearly stated that the Hawley soil ingestion rate was based on a model and further explained why it was too high to be the basis of the upper-bound value as follows:

> However, Kissel et al. (1998) questioned the likelihood of such a high consumption rate among nonsmoking, non-geophagic adults based on adult volunteers reporting that the presence of roughly 10 mg of soil in the mouth is readily detected and unpleasant. Kissel et al. (1998) concluded that “high-end estimates of daily soil ingestion rates in the range of 500 mg/day would appear to be implausible, at least for non-smoking, non-geophagic adults” (Kissel et al., 1998). (HHRA Volume IIIA, Section 6.5.1.5)

In addition to the uncertainties described above, there also is uncertainty in extrapolating results from exposure patterns that are the subject of studies in the scientific literature to individuals engaged in site-specific activities, such as dirt-biking and hunting in a floodplain. This is particularly a problem for adults, but also is relevant for young children.

Because of the uncertainty in extrapolating data to site-specific conditions, EPA elected to define soil ingestion rates with triangular distributions rather than a more refined probability distribution based on any single study or combination of studies from the scientific literature. EPA could have defined a more refined distribution, assuming uncertainties were known and acknowledged appropriately.

To explore how risk results might change by defining a more refined distribution, EPA conducted a sensitivity analysis of the young child exposure scenario to

determine how MCA results would be affected if the soil ingestion rate were described by a lognormal probability distribution based on a study of soil exposure among young children (see Section 7.2.2.6 of HHRA Volume IIIA). EPA, Colorado Department of Public Health and the Environment (CDPHE), and the Department of Energy (DOE) (2002) developed this distribution using data for young children living in and around Anaconda, Montana (Calabrese et al., 1997). They recommended use of a truncated lognormal distribution, with an arithmetic mean of 47.5, standard deviation of 112, and a maximum of 1,000 mg/day. Results using this distribution differed somewhat from the MCA results (based on triangular distributions): at the 25th, 50th, and 75th percentiles, the HHRA distributions were a factor of 2.4 to 3 higher. The differences were smaller at the higher percentiles of the distribution: about a factor of 1.9 higher at the 90th percentile, 1.4 higher at the 95th percentile, and almost no difference at the 99th percentile.

Yet a different distribution would be obtained using data from the Amherst, MA cohort of young children (Calabrese et al., 1989; Stanek and Calabrese, 1995), but a comparison of results from the Anaconda and Amherst cohorts (see Figures A-7 and A-8 and Table A-10 in EPA, CDPHE, DOE, 2002) suggests that associated risk estimates are not likely to be lower than those obtained with the distribution based on the Anaconda cohort, and could be higher.

Therefore, EPA believes that use of distributions that are more refined than the triangular distributions used in the MCA to evaluate young children would not greatly influence risk conclusions. Insufficient data are available to define more refined MCA distributions for older receptors.

References:

Calabrese, E.J., H. Pastides, R. Barnes, C. Edwards, P.T. Kostecki, E.J. Stanek, P. Veneman, and C.E. Gilbert. 1989. How much soil do young children ingest: An epidemiologic study. *Regulatory Toxicology and Pharmacology* 10: 1-15.

Calabrese, E.J., E.J. Stanek, P. Pekow, R.M. Barnes. 1997. Soil ingestion estimates for children residing on a Superfund site. *Ecotoxicology and Environmental Safety* 36: 258-268.

EPA (U.S. Environmental Protection Agency), CDPHE (Colorado Department of Public Health and the Environment), and DOE (U.S. Department of Energy). 2002. *Task 3 Report and Appendices: Calculation of Surface Radionuclide Soil Action Levels for Plutonium, Americium, and Uranium*. (One of five reports prepared by U.S. Environmental Protection Agency, Colorado Department of Public Health and Environment, and U.S. Department of Energy). September 30, 2002. 401 pp.

Hawley, J.K. 1985. Assessment of health risk from exposure to contaminated soil. *Risk Analysis* 5:289-302.

Kissel, J., J.H. Shirai, K.Y. Richter, and R.A. Fenske. 1998. Investigation of Dermal Contact with Soil in Controlled Trials. *J. Soil Contamination* 7:737-752.

Stanek, E.J. and E.J. Calabrese. 1995. Daily Estimates of Soil Ingestion in Children. *Environ. Health Perspect*. 103:276-285.

Fraction Ingested

The HHRA also truncates the input distribution for the fraction of soil ingested from the site and uses a uniform distribution ranging from 50 to 100 percent (Vol. IIIA, p. 6-13). This distribution is also biased and does not reflect the variability that is likely to occur for individuals involved in direct contact activities. For example, there may be many days when individuals are present in exposure areas for only a very brief period of time and do not have any hand contact with soils. Since hand-to-mouth activity is generally considered to be the source for soil ingestion, there would be no soil ingested from the exposure areas on those days. Similarly, there may be many days during which hunters or other recreators will wear gloves, due to cold temperatures, so that there will be no direct contact between hands and soils. Thus, it is appropriate to allow the input distribution to range between zero and 100 percent to reflect the natural variation that may occur within the exposed population and during different days of activity. Accordingly, EPA should replace its current distribution for fraction ingested with a uniform distribution that ranges from zero to 100 percent.

RESPONSE GE-21:

Soil ingestion rates represent the total daily intake of soil. Fraction ingested (FI) is a unitless term that represents the fraction of the soil or sediment ingested daily from the contaminated source. Even if an individual spends only part of a day at the site, the intensity of recreational soil exposure could far exceed other soil ingestion exposures that may occur during the rest of the day. In addition, this exposure could also contribute to ingestion exposure during the rest of the day and beyond if soils are tracked into a house or remain on clothing.

A FI of 1.0 was used in the point estimate RME evaluation for all of the scenarios to represent a high-end exposure in which all soil or sediment ingested by a person was assumed to be from the contaminated area. A factor of 0.5 was used in the point estimate CTE evaluation for all recreational scenarios to reflect the assumption that exposure time at a recreational scenario may be only a fraction of the day and that other scenarios (such as activities at home) also account for daily soil ingestion. This range was used in the MCA analog analysis assuming a uniform distribution to represent variability in the amount of soil ingested from the contaminated area. The same range was used in the PBA, but defined as an interval rather than a precise uniform distribution to address uncertainty about selection of this distribution type.

In defining FI for the probabilistic risk characterization, EPA began by defining who was to be protected. Specifically, these are people who spend time at the site. Because the 1-ppm tPCB isopleth usually represents the portion of the exposure areas closest to the river, an attractive resource, it is reasonable to

assume that one might spend all of one's time in this area closest to the river. Therefore, the FI upper bound was set to 1. Because the whole point of the assessment is to evaluate someone who spends time at the exposure area, zero is not a logical choice for the lower bound of the FI distribution, although the lower bound for FI cannot be defined with certainty. Because it is reasonable to assume that, in general, people are more likely to be near the river (i.e., within the 1-ppm tPCB isopleth) than far from the river, given that the river is an attractive resource, the distribution of 0.5 to 1 reflects this tendency. In addition, the use of 0.5 and 1 as the limits of the FI term is an acknowledgment of the fact that the literature on soil ingestion does not provide sufficient information to apportion ingested soil quantities among different places and activities. Rather, soil ingestion rates represent exposure during an event such that a receptor could get all of his or her exposure from the contaminated source. Given the limited data available to further refine the FI distribution and the likelihood that receptors would favor locations near the river, the FI used in the probabilistic risk characterization was not refined beyond what was used in the point estimate risk characterization. Also, if a fraction of an individual's daily soil ingestion is attributed to the portion of a day spent in a recreational area, it is reasonable to assume that the remaining fraction of the daily soil ingestion may occur at another contaminated site.

The comment that the FI is biased high because clothing worn by some receptors during cold winter months would limit soil ingestion has no merit because EPA assumes that all receptors except the waterfowl hunter have soil and sediment ingestion rates of zero during the colder months of the year. The waterfowl hunter is assumed to be exposed only during hunting season from early September through December, when they might wear gloves as noted by the commenter. However, FI is not used to evaluate dermal exposures. Therefore, wearing gloves is relevant to the soil ingestion pathway only to the extent that soil ingestion is correlated with hand-to-mouth activity. The commenter notes that "there will be no direct contact between hands and soils" when hunters wear gloves, but soil can adhere to gloves with subsequent soil transfer to the mouth.

PCB Dermal Absorption Rate

For its probability bounds analysis, EPA has developed a highly skewed input distribution for the PCB dermal absorption rate, with an interval that ranges from 6 percent to 41 percent. The upper end of the range was derived through MDEP's (Harnois and Smith, 2001) manipulation of the data from the Huntingdon Life Sciences study, subsequently reported by Mayes et al. (2002), and is based on an assumption that is not supported in the published literature. The lower end of the range is based on the upper-bound estimate of absorption derived by EPA (1992a) based on the Roy et al. (1990) study. This value was previously used by EPA (EPA, 1992a) as its upper-bound estimate of the dermal absorption factor for PCBs. Thus, the distribution fails to consider the other data provided by Roy et al. (1990), which indicated that dermal absorption could be as low as 0.6 percent (EPA, 1992a). Consequently, EPA's input distribution does not provide an

accurate representation of the range of variability and uncertainty associated with this important exposure parameter.

The Mayes et al. (2002) study evaluated absorption of Aroclor 1260 through the skin of rhesus monkeys, demonstrating absorption factors of approximately 4 percent. During that study, the researchers could account for roughly 59 percent of the Aroclor 1260 dose in the dosing apparatus or on the skin at the end of the dosing, leaving approximately 41 percent of the original dose unrecovered. In their discussion of these data, Harnois and Smith (2001) assumed that the remaining 41 percent of the nominal dose was retained in the skin for later absorption. This is the basis for the upper end of the dermal absorption distribution used in the probabilistic analysis of the HHRA.

Harnois and Smith's (2001) assumption that 41 percent of the dose was retained in the skin is not borne out by experiments conducted by Wester et al. (1993), the study upon which the HHRA bases its point estimate dermal absorption factor of 14 percent. In that study, Wester et al. also evaluated the percutaneous absorption of Aroclors 1242 and 1254 into human skin *in vitro*. These Aroclors were applied to the skin in soil, mineral oil, or water. The material was left on the skin for 24 hours. The skin surface was then washed once with liquid soap and twice with distilled water. Wash solutions and cells were then analyzed for PCB content. Wester et al. (1993) reported that only 2.6 percent of the Aroclor 1242 applied in soil and 1.6 percent of the Aroclor 1254 applied in soil remained on the skin after the soil was removed and the skin was washed. While Wester et al. did not evaluate Aroclor 1260, it is likely that the amount remaining on the skin would be similar or lower, given the very high binding affinity of Aroclor 1260 to soil particles. These findings appear to indicate that MDEP's assumption that 41 percent of the applied dose would be retained in the skin after washing is unlikely to be true and results in overestimated estimates of absorption.

In addition, that upper-bound absorption factor is not supported by dermal absorption studies of other chlorinated organic compounds (EPA, 2001). As shown in the following table, EPA's (2001) dermal guidance (Exhibit 3-4) recommends the following absorption factors for specific chlorinated organic compounds.

Chlorinated Organic Compound	Dermal Absorption Fraction
PCBs	0.14
Chlordane	0.04
2,4-D	0.05
DDT	0.03
TCDD	0.03
Lindane	0.04
Benzo(a)pyrene	0.13

Clearly, there is uncertainty associated with the dermal absorption factor for PCBs, and GE believes that it is appropriate for EPA to consider the range of possible values in its probabilistic

analysis. However, as with the soil ingestion and fraction ingested parameters, the HHRA biases the dermal absorption distribution by using only upper-bound values, rather than evaluating the full range of uncertainty that is evident. At a minimum, EPA should revise the lower bound of the PBA range from 6 percent to 0.6 percent to capture the full range of uncertainty associated with this factor.

RESPONSE GE-22:

There is uncertainty associated with the value of the dermal absorption factor for PCBs. As discussed in Section 6.5.1.7 of HHRA Volume IIIA, dermal absorption is a function of skin type, duration of exposure, congener composition of the PCB mixture in the floodplain, and organic content of soil. Insufficient data are available to define with confidence minimum and maximum dermal absorption values for the contaminant mixture at this site. For this reason, the uncertainty was evaluated using a p-box defined only by an interval.

Because PCBs in the Housatonic River Area (HRA) are a mixture of many congeners, the dermal absorption data for PCB mixtures, based on the studies conducted by Wester et al. (1993) and Mayes et al. (2002), were preferred over data from studies on individual congener data to define the PBA input. The Mayes study used soil from the Housatonic River floodplain that was spiked with Aroclor 1260 and aged for two different periods. The use of site-specific soil, in principle, would make the Mayes study the preferred basis of the dermal absorption value. However, a review by EPA and MDEP (2001) (referred to as Harnois and Smith 2001 in the comment) of the protocols and procedures used in this study questioned the reliability of its results, and suggested that the absorption fraction reported by Mayes et al. (2002) is biased low. The potential low bias of results from Mayes et al. (2002) is discussed in Volume IIIA, Section 4.5.1.4, as well as in EPA (2001) and MDEP (2001).

The upper bound of the p-box was defined based on the MDEP (2001) assessment of the Mayes study and thus appropriately includes the extent of uncertainty based on the experiment. Although the comment includes the opinion that this value is too high, there is no further explanation or justification for criticism of EPA's interpretation of results from Mayes et al. (2002). EPA chose the higher upper bound from Mayes et al. (2002) because this study involves a PCB mixture specific to the HRA and Wester et al. (1993) does not.

Because limited data for PCB mixtures were available, EPA used data from an experiment with a single congener, 3,3',4,4'–tetrachlorobiphenyl, to define the lower bound for absorption in the PBA of 6% (EPA, 1992). This minimum value is similar to the dermal absorption value presented in Mayes et al. (2002), 4%, which may be biased low, as already discussed.

The claim that the upper-bound absorption fraction is not supported by dermal absorption studies of other chlorinated organic compounds is without merit for two reasons. First, PCBs are a complex mixture of congeners and some congeners in the mixture will be absorbed to a greater extent than the

compounds listed in this comment (not all of which are chlorinated although they were designated as such). Second, the dermal absorption fractions listed in the table are average absorption values recommended for use by EPA (2001) in a point estimate approach to dermal exposure. They are not intended to encompass the range of uncertainty associated with the dermal absorption fraction; an earlier document titled *Dermal Exposure Assessment: Principles and Applications* (EPA, 1992) provides additional relevant data. For example, Table 6-2 of EPA (1992) indicates that for TCDD adhered to soil, the fraction of applied dose absorbed ranges from 0.01 to 0.16. For soil-adhered tetrachlorobiphenyl, the listed range is 0.074 to 0.5.

References:

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

EPA (U.S. Environmental Protection Agency). 2001. Superfund Dermal Workgroup Review of PCB Dermal Absorption for Soils Study. Memorandum from Daniel Stralka, EPA Regional Toxicologist to Margaret McDonough, Region I EPA. November 12, 2001.

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. Toxicol Pharmacol*. 35(3):289-95.

MDEP (Massachusetts Department of Environmental Protection). 2001. Huntingdon Life Sciences Study 00-3431. Memorandum from Marion Harnois, MDEP Office of Research and Standards to Bryan Olson, Region I EPA. November 16, 2001.

Wester, R.C., H.I. Maibach, L. Sedik, J. Melendres, and M. Wade. 1993. Percutaneous absorption of PCBs from soil: in-vivo rhesus monkey, in-vitro human skin, and binding to powered human stratum corneum. *Journal of Environmental Toxicology and Environmental Health* 39:375-382.

3.2.2 Potential Errors in Development of the "X" Factor

The revised HHRA uses a subprogram in its probabilistic analyses that is intended to combine a number of factors, including body surface areas based on body weight and height (adults only), body part-specific adherence factors, and seasonal variations in exposure to produce an input distribution to assist in evaluating potential risks due to dermal exposure for each age and receptor group (Vol. IIIA, pp. 6-15 - 6-16). This subprogram combines distributions for body weights, heights, surface areas for each body part exposed (accounting for seasonal changes), and adherence factors for each body part to derive an input distribution for the single factor, X,

that goes into the probabilistic risk calculation. The values estimated by EPA for X are provided in Table 6-3 (Vol. IIIA) of the HHRA.

GE has not been able to duplicate the "X" values derived by EPA. This is likely due, at least in part, to the lack of information provided about the distributions used for soil adherence factors. It may also be due to errors in the approach.

Table 6-4 of the HHRA indicates that EPA has developed "empirical" distributions for the soil adherence factors and it provides minimum and maximum values for these distributions. It does not, however, provide any information about the shape of the distribution itself. Thus, it is not possible to ensure that the inputs for adherence factors used by GE in an effort to duplicate the calculation are or are not correct.

Table 6-4 also states that the equation used to calculate the surface area (SA*) for each body part is $SA^* = a\ BW^b\ H^c$, in which a, b, and c are constants listed in that table. That table does not agree, however, with Table 6-1 in EPA's *Exposure Factors Handbook* (1997), which describes the same methodology. According to Table 6-1 of *Exposure Factors Handbook*, SA is calculated as follows:

$$SA = a_o\ W^{a1}\ H^{a2}$$

Using the female head as an example, EPA (1997) reports that the values for these components of the equation are the following:

$a_o = 0.0256$

$W^{a1} = 0.124$ where W is the body weight (equivalent to BW in HHRA Table 6-4)

$H^{a2} = 0.189$ where H is the height (equivalent to H in HHRA Table 6-4)

The values for a_o agree between these two documents but there is not agreement in the values for "b" and "c." According to Table 6-4 of the HHRA, the value of "b," which is the exponent for the body weight in that equation for the female head, is 0.124. However, EPA (1997) indicates that 0.124 is actually the value of the entire factor W^{a1}, not just the exponent. Similarly, the HHRA reports that the value of "c", which is the exponent for H in Table 6-4, is 0.189. Again, however, EPA (1997) reports that 0.189 is, in fact, the value of the entire factor H^{a2}. In addition, EPA (1997) reports that this approach calculates surface areas in square meters while the HHRA reports them to be in square centimeters. When the units are converted to be consistent, the calculated values do not match.

It is not clear whether EPA has made errors in its calculations. It does not appear that the equation presented in the *Exposure Factors Handbook* is correct because all values in the equation would be constant so that all surface areas would be calculated to be the same, regardless of weight or height. At the same time, however, when AMEC and BBL attempted to use the equation presented in the HHRA, we were not able to derive the numbers that are reported. EPA needs to check its approach to make sure that it has been conducted correctly; and if it has, needs to fix any errors that may be in the equation and provide a more transparent

explanation in the text of how these surface area estimates, and the resulting X factor, are derived.

RESPONSE GE-23:

GE was unable to reproduce the "X" factors used in the probabilistic analyses and questioned whether errors were made in developing these factors. GE suggested three possible reasons for their inability to reproduce the "X" factors:

- The equation used to calculate skin surface area from body weight and height in the HHRA appears to be inconsistent with the equation presented in the *Exposure Factors Handbook*;
- The surface area units appear to be inconsistent with those in the *Exposure Factors Handbook*; and
- The shapes of the empirical distributions used for soil adherence factors are unclear.

There are no mathematical errors inherent in "X" factor estimates, and the remainder of this response addresses the three specific issues raised by GE.

Surface Area Equations

The equation used to calculate surface area is correct and consistent with the *Exposure Factors Handbook*. Also, the surface area equation calculates surface area in square meters. In the HHRA, surface areas were converted to square centimeters before multiplying by the soil adherence factors (mg/cm^2), but the conversion factor of 10,000 cm^2/m^2 was not shown in the equation for "X" reported in Section 6.5.1.9 of HHRA Volume IIIA.

In Table 6.1 in EPA's *Exposure Factors Handbook* (1997), the a1 values are mislabeled as Wa1, and the a2 values are mislabeled as Ha2. The surface area equations used in the HHRA are consistent with the correct equations, which appear in the source document for Table 6.1 (EPA, 1985; Table 3-5 on page 21). EPA derived the regression equations from individual body weight, height, and body part surface area measurements. The derivations can be reproduced following the methodology reported in the EPA (1985) document and using the data presented in the Appendix B-1 of that document.

Soil Adherence Factor Distributions

The shapes of the empirical distributions used for soil adherence factors are simply a function of the data because these distributions were defined entirely by the soil adherence data. The data used for each distribution are indicated in Tables 6-4 to 6-15 of HHRA Volume IIIA, and all data are provided in EPA (2001). Empirical distributions were created by importing soil adherence factor data into Excel and defining custom distributions using Crystal Ball®. Rather than

fitting a standard distribution type to the data, the distribution is assumed to be defined completely by the data.

References:

EPA (U.S. Environmental Protection Agency). 1985. *Development of Statistical Distributions or Ranges of Standard Factors Used in Exposure Assessment. Final Report*. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-85/010. August 1985.

EPA (U.S. Environmental Protection Agency). 1997. *Exposure Factors Handbook,* Volumes I-III. Office of Research and Development. EPA/600/P-95/002Fa, b, and c. August 1997.

EPA (U.S. Environmental Protection Agency). 2001. *Risk Assessment Guidance (RAGS) for Superfund,* Volume I: *Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment), Interim.* Office of Emergency and Remedial Response. September 2001.

4.0 FISH AND WATERFOWL CONSUMPTION ASSESSMENT

In response to the peer reviewers' comments on the previous draft HHRA, EPA has revised the fish consumption rates for both the deterministic and probabilistic analyses. The HHRA now bases its fish consumption estimates on a subpopulation of anglers from the Maine angler survey (Ebert et al., 1993) – namely, those who did not share any of the fish they caught or obtained with any other individuals. The HHRA then assumes that young children (aged 1 to 6) have fish consumption rates that are one-half the adult rates. The HHRA needs to recognize, however, that the subpopulation of non-sharing consumers is not representative of the total population of fish consumers who may use the study area. In addition, there is evidence that the consumption rate estimates used to evaluate young children do not represent consumption of sport-caught fish by this age group.

RESPONSE GE-24:

The EPA responses to the three general GE comments summarized above are provided below in the detailed responses to Sections 4.1 through 4.3 of the GE comments, Responses GE-25 through GE-27, respectively.

4.1 Adult Fish Consumption Rate in Deterministic Assessment

The revised HHRA bases its fish consumption rates on a small subpopulation of individuals who participated in the Maine angler survey (Ebert et al., 1993). The consumption rates are now based only on adults in that survey who consumed 100 percent of the fish that they caught or obtained from other sources (i.e., those who did not share any of that fish with any other individual throughout the one-year survey period) (Vol. IV, p. 4-48). This subpopulation was selected due to EPA's concern that the sharing assumptions that had been used in deriving the consumption rates in the Maine angler survey might be underestimating exposures to adult male

consumers who might consume more fish than would women or children in the survey (Vol. IV, p. 4-41). In making this selection, the revised HHRA assumes that this subpopulation of non-sharing consumers is representative of all sport-caught fish consumers.

As discussed in Section 2.1 above, this assumption does not appear to be correct; and EPA should make clear that this subpopulation is likely to be very small and cannot be considered representative of the total population of recreational anglers that use the Housatonic River.

Moreover, in an effort to support its adult fish consumption rates, the HHRA cites the results of the MDPH exposure prevalence study (MDPH, 1997) and the CT Housatonic River creel survey (Ebert et al., 1996) (Vol. IV, pp. 4-50 - 4-53). These comparisons, however, contain errors that need to be corrected before the HHRA is finalized.

The HHRA indicates that the data collected in the MDPH exposure prevalence study support the RME consumption rate of 31 g/day used in the HHRA (assumed to be equivalent to 50 8ounce meals/year). The rationale for this conclusion is that the 95th percentile frequency of fish meals reported in the MDPH survey was 104 meals/year and that 75 percent of those meals were sport-caught meals, resulting in an estimate of 78 sport-caught fish meals/year (Vol. IV, p. 4-51). This conclusion cannot be drawn from the MDPH survey data due to the way in which the data were collected. The questionnaire used in the MDPH (1997) study does not allow EPA to calculate the fraction of sport-caught fish meals consumed by those individuals. The participants in that study were asked to estimate the frequency of freshwater fish meals they consumed. In the following question, they were asked to indicate how they "usually" obtained those fish. They were not given an option of assigning a fraction of meals as sport-caught or supermarket/grocery store fish. Hence, the respondents were forced to designate all of their fish meals as either sport-caught or supermarket/grocery store fish even though they may have consumed a mixture of both. Because of the way that the questionnaire was designed, each participant would have had a frequency of either 100 percent sport-caught or zero percent sport-caught. Thus, the HHRA's assumption that 75 percent of the meals were sport-caught actually reflects the fraction of study participants who reported that they "usually" obtained those fish from sport-fishing, not the actual fraction of fish meals consumed that were sport-caught. Consequently, the HHRA's comparison is misleading and should be dropped from the discussion.

In addition, the HHRA incorrectly cites the results of the CT Housatonic River creel survey (Ebert et al., 1996). The HHRA reports that the 95th percentile consumption rate estimates ranged from 21.3 g/day to 32 g/day, depending upon the assumptions made about the number of individuals with whom the fish were shared (Vol. IV, p. 4-52). In fact, the range of 95th percentile rates reported by the authors for different sharing assumptions ranged from 12 g/day (assuming that fish was shared equally among household members) to 32 g/day (assuming that all of the fish harvested were consumed by a single individual). EPA needs to correct this error.

The HHRA goes on, on page 4-52, to make a separate calculation, based on the Maine angler survey consumption rate data and the information on relative gender-specific sizes of all types of fish meals (not just sport-caught fish meals) provided in the EPA's *Exposure Factors Handbook* (1997; Table 10-37). The calculation is intended to demonstrate that men consume more fish than women and that when the total mass of consumable fish reported by each Maine angler (not just the non-sharing individuals) is assumed to be consumed by one male and one female

consumer per household and adjusted for relative gender-specific portion size, the same estimate of 31 g/day results.

This comparison is completely artificial because it manipulates the data from the Maine angler survey, making separate assumptions about sharing that are not supported by the data provided by the participants themselves, and then applying a ratio of gender-specific consumption rates that are also not necessarily reflective of long-term consumption behaviors. The data upon which EPA's gender-specific fish consumption rate ratio is based are short-term (3-day diary study) data, and are more representative of portion size for individual meals than they are of long-term consumption rates. These data represent a three-day average consumption rate for only those individuals who consumed fish during the 3-day study period. They did not capture the long-term behavior of those individuals nor did they include individuals who consume fish but did not do so during that 3-day study period. While the ratio of portion sizes may be appropriate, it does not mean that women have lower long-term consumption rates than men, because long-term consumption is a combination of portion size and meal frequency, and the EPA data that are being used to make this comparison do not provide any information about long-term frequency of fish meals.

A better measure of the relative consumption by males and females is provided in the data for the subpopulation of non-sharing consumers in the Maine angler survey (Ebert et al., 1993) upon which the HHRA bases its consumption rates. These data indicate that the HHRA's unequal sharing assumption may not be representative when sport-caught fish are consumed. In fact, as shown below, the information available from the Ebert et al. (1993) survey for those non-sharing individuals indicates that long-term rates of consumption of sport-caught fish by men and women are very similar.

Fish Consumption Rates (g/day) for Non-Sharing Consumers

	Male	Female
Minimum	0.1	0.2
Maximum	182	52.4
Median	2.8	3.7
Mean	8.9	9
75th percentile	8.9	11.5
90th percentile	21	17
95th percentile	31	31

These data indicate that, over a one-year survey period, the non-sharing female fish consumers ate comparable amounts of fish to the amounts eaten by men. In fact, the 95th percentile for non-sharing females was identical to the 95th percentile value for non-sharing males. These data indicate that males and females eat approximately the same amounts of sport-caught fish, so that the assumption used in the Maine angler survey may have been a very reasonable assumption. These data also undermine EPA's rationale for selecting fish consumption rates for this non-sharing subpopulation, and instead support GE's previously recommended 95th percentile consumption rates derived from the Ebert et al. (1993) survey (12 g/day for rivers/streams and 16

g/day for lakes/ponds; see AMEC and BBL, 2003) as likely more representative of the total angler population.

RESPONSE GE-25:

EPA disagrees that the consumption rate is based on a small and unrepresentative subpopulation from the Maine Angler Survey. The consumption rate used in the HHRA was based on the subset of the Maine angling population that does not share fish with other household members to avoid the bias introduced by the assumption of equal sharing.

The structure of the MDPH questionnaire is described correctly in this comment, and EPA agrees that the assumption used in the HHRA that 75% of the meals were sport-caught represents the fraction of study participants who reported that they "usually" obtained those fish from sportfishing. Although the MDPH survey should not have been used as an example of the consistency of the fish consumption rate used in the HHRA with other sources of data, EPA notes that the consumption rate used in the HHRA was not based on the data from the MDPH questionnaire and this inappropriate comparison does not affect the risk assessment results in any way.

EPA does not agree that the HHRA incorrectly cites the results of the CT Housatonic River Creel Survey, although the language in the HHRA could have been clearer. As stated in the HHRA: "Estimates of the 95th percentile of fish consumption ranged from 21.3 g/day to 32 g/day, depending upon assumptions of sharing within a family (and using specific family size for recreational anglers, rather than the statewide average)." The consumption rate described in this statement was calculated by EPA, and is not the estimate published by Ebert et al. (1996) in which the consumption rate was calculated assuming all members of a family shared the catch equally, based on an average family size of 2.5. This family size is the average for the State of Connecticut. However, subsequent to the publication by Ebert et al. (1996), Balcom et al. (1999) published data indicating the average household size of sport fishing families is 1.5. The 21.3 g/day value reported in the HHRA is based on the Ebert data, but modified by a household size of 1.5. This was explained in HHRA Volume IV, Section 4.5.2.2.3.

EPA disagrees with the comment that the difference in consumption rates between males and females presented in the HHRA is a reflection of meal sizes rather than long-term consumption rates. The consumption rates derived for the HHRA were based on a large-scale national survey of 15,000 individuals (the Continuing Survey of Food Intake by Individuals, CSFII, for the years 1989-91) that used a stratified sampling technique and obtained data on three consecutive days of food consumption by interview and diary (EPA, 1997). The discussion of this issue in the *Exposure Factors Handbook* (EPA, 1997) states that "Such short-term data [three days of food information] are suitable for estimating mean average daily intake rates representative of both short-term and long-term consumption. However, the *distribution* of average daily intake rates generated

using short term data do not necessarily reflect the long term *distribution* of average daily intake rates." The analysis used in the HHRA is based on the mean average daily rate, not the distribution, and thus required only that the three-day food information be reflective of average long-term consumption rates.

Although EPA believes that the Maine Angler Survey provides the most appropriate data for long-term fish consumption rates for the Housatonic River, EPA believes the larger national dataset provides a stronger statistical basis for establishing the *differences* in the long-term fish consumption rates between males and females. However, even if the data described in the comment regarding the lack of difference between males and females consumption rates of non-commercially caught freshwater fish (based on a 1-year recall study of 87 or 138 anglers, depending upon the source of the fish) were accurate, it would make little or no difference to the consumption rate used in the HHRA. The consumption rate used in the HHRA was based on the subpopulation of Maine anglers who did not share their fish, and included both the males and females in this population. The fish consumption rates for this subpopulation are 11% and 16% higher than the 95th percentile and mean consumption rates calculated by Ebert el al. (1993) based on the assumption that only adults in a household consume fish, and that they share equally.

References:

Balcom, Nancy C., Constance M. Capacchione, and Diane Wright Hirsch. 1999. *Quantification of Fish and Seafood Consumption Rates for Connecticut.* Prepared for the Connecticut Department of Environmental Protection, Office of Long Island Sound Programs.

ChemRisk. 1992. *Consumption of Freshwater Fish by Maine Anglers*. 24 July 1992.

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

EPA (U.S. Environmental Protection Agency). 1997. *Exposure Factors Handbook*, Volumes I-III. Office of Research and Development. EPA/600/P-95/002Fa, b, and c. August 1997.

4.2 Child Fish Consumption Rate in Deterministic Assessment

The revised HHRA uses data from EPA's *Estimated Per Capita Fish Consumption in the United States* (2002) to set children's fish consumption rates at 50 percent of the adult fish consumption rates (Vol. IV, pp. 4-53 - 4-54). The rates provided in that document, however, are not representative of long-term consumption rates, but instead are representative of meal sizes since they are short-term measures of consumption from 2-day diaries. As discussed in Section 4.1,

such comparisons do not necessarily comport with frequency or long-term consumption behavior.

Researchers at Cornell University (Knuth et al., 1998) evaluated consumption of sport-caught fish by children aged 8 to 14 years. This diary study collected information about children from sport-fishing families who were involved in the New York Sportfishing and Aquatic Resources Education Program (SAREP). These researchers asked the children to record meal-by-meal information for all fish meals eaten between July 1 and October 15, 1996. The source of each fish meal (store, restaurant, or sport-fishing) and a relative portion size for each were also recorded.

According to the raw data provided by the study's authors, these children ate between 0 and 12 total fish meals and 0 to 11 sport-caught fish meals during the survey period of 3.5 months, with average rates of 4 total fish meals and 1 sport-caught fish meal during that time period (Knuth et al., unpublished data). Using the portion size information provided by each individual for each meal consumed, summing all meals consumed to derive a total amount of fish consumed during the survey period, and dividing by the number of days included in the survey period (107 days), AMEC derived a distribution of fish consumption rates for the survey period that ranged from 1 to 22 g/day with a mean of 1.7 g/day and a 95th percentile of 4.3 g/day. These rates were based on data collected for children aged 8 to 14 years and thus are likely to substantially overestimate consumption by children under the age of six, who are likely to have smaller portion sizes than the older children studied by Knuth et al. (1998). In addition, these rates likely overestimate consumption over a one-year period because sport-fishing activities and consumption are likely to be greatest during open water season (the period in which the survey was conducted) so that consumption for many of these children may have been substantially lower during the winter months.

The following is a comparison of the rates derived based on the Knuth et al. (1998) data with the point estimates used in EPA's deterministic analysis. (While the HHRA does not specifically identify consumption rates for older children, it essentially uses the same consumption rates for anyone over the age of 6 years.)

Sport-Caught Consumption Rates (g/day)

	Knuth et al. data	EPA point estimates (bass)	
	8-14 year olds during the Diary Period	1-6 Year Old Children	Older Children/ Adults
Mean	1.7	4.3	8.7
95th %ile	4.3	16	31

It appears that the 95th percentile rate used by EPA to evaluate children aged 1 to 6 years overestimates consumption by this age group by at least a factor of 4, since this age group would be expected to consume smaller amounts of fish than the 8 to 14 year old children included in the Knuth et al. (1998) survey. In addition, the RME adult consumption rate, which is assumed to be applicable to anyone over the age of 6 years, overestimates consumption by 8 to 14 year old children by a factor of 8. In fact, because the Knuth et al. data covered the period between July and October, which is likely to be a much more active fishing period for most anglers than the

winter months, even this analysis of the Knuth et al. data is likely to overestimate consumption for this age group.

Although the Cornell study did not collect diary information on children under the age of 8, another phase of the study provided information on the estimated 12-month meal frequencies for younger children in these same families (Knuth et al., unpublished data). Using a very conservative meal size of 4 oz. (the most common meal size reported by the 8 to 14 year old children, who would be expected to eat larger portions than 1 to 6 year old children), the data available for these young children indicated that their mean consumption rate is 2 g/day and their 95th percentile consumption rate is approximately 3.0 g/day. Thus, based on this comparison of long-term rates, it appears that EPA's point estimate consumption rates for young children overestimate mean (CTE) consumption by a roughly a factor of 2 and RME consumption by more than a factor of 5.

Given the availability of specific sport-caught fish consumption information for children aged 8 to 14 years in the Knuth et al. (1998) study, GE recommends that EPA revise its deterministic fish consumption rates for young children to include a mean of 2 g/day and a 95th percentile of 3 to 4 g/day. While the HHRA does not separately evaluate fish consumption by older children, it should discuss the fact that older children are inherently included in the adult fish consumption rates and that these consumption rates overestimate potential exposures for the older children.

RESPONSE GE-26:

For reasons similar to those presented in Response GE-25 (where additional discussion of this topic may be found), EPA disagrees with the assertion that the child fish consumption rates published in Estimated Per Capita Fish Consumption in the United States (EPA, 2002) are not reflective of mean long-term consumption rates. EPA considers the mean consumption rate to be reflective of both short-term and long-term consumption rates.

The Knuth et al. report (1998) on children participating in the Sportfishing and Aquatic Resources Education Program (SAREP) in upstate New York was carefully evaluated by EPA during its development of a child fish consumption rate for the HHRA. The unpublished data mentioned in the comment were not available for review (attempts to contact Dr. Knuth for the data were not answered). The specific objectives of this study were to:

1) Identify fish consumption health advisory awareness, understanding, and related behaviors (fishing, fish preparation, fish consumption), among families whose youth participate in SAREP.

2) Evaluate the extent to which youth adhere to fish consumption advisory recommendations including analyzing what types of fish are caught from which locations, the types of fish preparation methods used, and the extent of fish consumption, focusing on fishing and fish consumption during the summer months.

EPA agrees that this type of study can provide useful information about fishing and consumption behaviors for children who fish. However, since the focus is on the fishing patterns of youth, it is not clear whether this population of 53 individuals aged 8-14 is representative of the consumption patterns of younger children whose parents are more likely to be the anglers catching the fish. For example, the youth fished smaller waterbodies than adults and most commonly caught panfish.

After consideration of the limitations of the Knuth et al. (1998) study, and the results of studies of children of anglers such as that by Beehler et al. (2002) and Balcom et al. (1999), EPA concluded that the approach taken in the HHRA provides a more reasonable and likely representation of child consumption rates for children of anglers, the young child population of concern at the Housatonic River.

References:

Balcom, Nancy C., Constance M. Capacchione, and Diane Wright Hirsch. 1999. Quantification of Fish and Seafood Consumption Rates for Connecticut. Prepared for the Connecticut Department of Environmental Protection, Office of Long Island Sound Programs.

Beehler, G.P., J.M. Weiner, S.E. McCann, J.E. Vena, D.E. Sandberg. 2002. Identification of sport fish consumption patterns in families of recreational anglers through factor analysis. *Environ Res* 89(1):19-28.

EPA (U.S. Environmental Protection Agency). 2002. *Estimated Per Capita Fish Consumption in the United States.* EPA-821-C-02-003. August 2002.

Knuth, B.A., N.A. Connelly, and B.E. Matthews. 1998. Children's Fishing and Fish Consumption Patterns. Cornell University Human Dimensions Research Unit. HDRU Series No. 98-3. May 1998.

4.3 Child Fish Consumption Rate in Probabilistic Assessment

In its probabilistic analyses, the revised HHRA evaluates exposures to young children due to the consumption of fish by developing input distributions for the numbers of meals consumed during the year and the sizes of those meals (Vol. IV, p. 6-28). As discussed above, the Knuth et al. (1998) survey provides information on the frequency of sport-caught meals consumed by 8 to 14 year old diary participants during the survey period. A separate survey instrument used by these authors to screen families and select children for the diary survey asked families to estimate the number of sport-caught and total fish meals consumed by themselves and ALL children in their families (all ages) on an annual basis. The following table provides a comparison of the estimated meal frequencies (meals/year) provided in the family survey for diary participants (aged 8 to 14 years) and for all children aged 1 to 14 years, with the input distributions used in the HHRA's probabilistic analyses (MCA and PBA) for bass consumption in all reaches:

Comparison of Meal Frequencies in Cornell Data and EPA Probabilistic Inputs						
	Meals/Year Based on Family Survey Data Collected by Knuth et al. (1998)				EPA Probabilistic Analyses for Bass	
Summary statistic	Diary (8-14 yr-old) participants including those who ate no sport-caught meals	Diary (8-14 yr-old) participants who ate at least one sport-caught meal	All children (all ages) including those who ate no sport-caught meals	All children (all ages) who ate at least one sport-caught meal	MCA Input Distribution for Bass	PBA Input Distribution for Bass
Minimum	0	1	0	1	0.25	0.03
Maximum	20	20	100	100	145	490
Mean	3	6	4	9	13.1	8.3 - 24.3
50th %ile	1	4	0	4		
95th %ile	17	20	20	20		
n	48	27	113	54		

As demonstrated in the above table, there were many children of sport-fishing families who did not consume any sport-caught fish despite the fact that their families did. To be representative of children who do eat sport-caught fish meals, it is most appropriate to base meal frequency estimates on the population of children who were reported to consume at least one sport-caught fish meal. For such 8 to 14 year old children in the diary study, the meal frequencies ranged from 1 to 20 meals/year, with a mean of 6 meals/year and a 95th percentile of 20 meals/year. For all children in these families, including those who did not participate in the diary survey, the meal frequencies for those who ate at least one sport-caught meal ranged from 1 to 100 meals/year, with a mean of 9 meals/year and a 95th percentile of 20 meals/year.

The HHRA's range for the MCA is quite conservative in that it ranges up to 145 meals/year and has a central estimate of 13.1 meals/year, which is 50 percent higher than the arithmetic mean based on the Knuth et al. (1998) data. Moreover, the range for the PBA analysis is highly over-conservative in comparison with the Knuth et al. (1998) data. The range of potential meal frequencies used in the PBA is 0.03 to 490 meals/year and the central estimate is an interval bounded by 8.3 and 24.3 meals/year. The maximum value, which is equivalent to 1.3 fish meals per day, is not supported by available fish consumption data (including adult consumption rates from the Maine angler survey upon which it is purportedly based) and appears to overestimate the maximum meal frequency reported in the Knuth et al. study by a factor of 5. The lower bound of the central estimate interval in the PBA is similar to the arithmetic mean meal frequency observed in the Knuth et al. family survey data, but the upper bound of the interval is higher by nearly a factor of 3. It appears that this approach will yield highly inflated estimates of exposure to young children.

GE recommends that EPA base its children's fish consumption rate distribution on the data provided in the Knuth et al. (1998) study. A distribution based on these data would include a minimum of 1 meal/year, a maximum of 100 meals/year, and a central estimate of 9 meals/year.

Use of such a distribution in the probabilistic analyses would yield more representative estimates of exposure and risk.

RESPONSE GE-27:

For the reasons discussed above (Response GE-26), EPA disagrees that the Knuth et al. (1998) dataset is an appropriate basis for establishing the child consumption rate for the Housatonic River HHRA.

References:

Knuth, B.A., N.A. Connelly, and B.E. Matthews. 1998. Children's Fishing and Fish Consumption Patterns. Cornell University Human Dimensions Research Unit. HDRU Series No. 98-3. May 1998.

5.0 AGRICULTURAL PRODUCTS CONSUMPTION ASSESSMENT

EPA has made a number of changes to the agricultural products consumption assessment. To begin with, EPA has added text attempting to support the assumption that the agricultural products consumption pathways evaluated in the HHRA represent reasonably anticipated future uses (Vol. V, Section 2.1.2). This discussion is based in part on interviews with local farmers and staff at the Pittsfield office of the USDA Farm Services Agency, local agricultural groups as well as personal observations by EPA personnel, contractors, and risk assessors. Much of this information, however, consists of personal opinions, not verified by independent sources, and as such is speculative. GE believes that the HHRA should not rely on such speculations, but should base its discussion on actual data.

In addition, EPA has made changes to its deterministic analysis of the agricultural products consumption pathways and has added probabilistic analyses of these pathways in an attempt to evaluate the variability and potential uncertainties associated with them. As discussed in the following subsections, a number of the revised factors used in the deterministic analysis are not justified, and the probabilistic analyses fail to adequately account for the variability and uncertainties in the risk estimates by using conservative point estimates rather than distributions.

RESPONSE GE-28:

The EPA responses to the GE comments summarized above are provided below in Responses GE-29 through GE-36.

5.1 Deterministic Assessment

At least two of the factors used in the revised HHRA's deterministic assessment of the agricultural consumption pathways are unjustified. First, the revised soil-to-plant transfer factor that is used to evaluate transfer to both exposed plants and fruits fails to take into account the removal of soil as vegetables and fruits are prepared for consumption, and does not even reflect the underlying data upon which it is based. Second, while the point estimate used for the mammalian bioconcentration factor (BCF) may have been reasonable for the range of soil concentrations modeled in the previous draft of the HHRA, it is not appropriate for the higher

soil concentrations that are being modeled in the revised HHRA. These points are explained below.

Soil-to-Plant Transfer Factor Used To Evaluate Exposures Via Vegetable and Fruit Consumption

Like the 2003 draft of the HHRA, the 2005 draft explains that "the maximum wet weight soil-to-plant [transfer factor] for corn" was used to estimate total PCBs (tPCBs) in "exposed" surface vegetables and fruits (Vol. V, pp. 4-12, 4-14). Although the basis for this transfer factor (TF) did not change (i.e., use of the maximum value for corn), the number used in the deterministic assessment was increased from 6.4E-04 in the 2003 draft to 1.8E-03 in the current version.

Using the maximum value overestimates the amount of tPCBs on ingested vegetables because it assumes that none of the food items consumed are ever washed. For a daily lifetime exposure, this is an erroneous assumption. This assumption is particularly critical since the TF of 1.8E-03 accounts for the only mechanism for PCBs to be ingested via this exposure pathway. If it is assumed that an individual washes the fruit and/or vegetable before consumption, thereby removing the PCBs and rendering the TF irrelevant, there would be no exposure via this pathway. While the deterministic assessment incorporates a "Produce Loss Factor" for fruit and vegetables to take into account removal during the processing of the food for consumption (e.g., peeling), this loss factor was only included in the CTE analysis (see Vol. V, Table 4-10). Thus, for the RME, the HHRA assumes that 100 percent of the PCBs deposited on the surface of the fruit or vegetable, as estimated by the TF of 0.0018, is consumed. This is an unreasonable assumption, resulting in an overestimation of exposure and risk from this pathway. A reasonable high-end estimate would be that, over time, at least 50 percent of the transported PCBs would be removed as a result of washing before eating, preparation before cooking, or a combination of the two.

In addition, the data presented in Table 4-4, upon which this TF is based, only include the samples with detectable concentrations of tPCBs in unwashed corn stalks. In fact, half of the corn samples analyzed as part of that sampling event had no detectable levels of PCBs (see Vol. V, Table 2-5). Thus, transfer factors based on these samples, if included, would have been zero. The fact that some of the corn stalks had no detectable levels of tPCBs underscores the highly and unnecessarily conservative nature of EPA's use of the maximum value as the TF.

In summary, the revised HHRA: (a) uses the maximum soil-to-plant transfer factor calculated for corn to evaluate all exposures to PCBs in surface vegetables and fruit; (b) assumes that there is no washing of any vegetable or fruit before consumption throughout the exposure period; (c) assumes, for the RME scenario, that vegetables and fruits are never peeled before consumption; and (d) bases the transfer factor on only the corn stalk samples that had detectable levels of tPCBs, thereby ignoring 50 percent of the available data. This combination of assumptions results in an unreasonable exposure scenario. It should be modified to reflect more realistic exposure conditions.

RESPONSE GE-29:

The approach to defining point estimate soil-to-plant transfer factors (TFs) for evaluating exposures via vegetable and fruit consumption used in the revised HHRA has not changed from the June 2003 HHRA. It still involves use of the

maximum transfer factor for corn samples from the site, but the second to highest value was erroneously used in the previous assessment. The revised assessment simply corrects this error. Therefore, GE's comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information. In addition, the comment made regarding the point estimate transfer factors for exposed vegetables and fruit was addressed in the revised HHRA by the addition of a probabilistic risk characterization.

Mammalian Bioconcentration Factor

While the mammalian BCF used in the deterministic assessment has not changed from the 2003 version, the modeled PCB soil concentrations have been altered in the revised draft. This fact impacts the choice of the mammalian BCF values, which appear to be dependent on the soil concentrations.

Fries (1996) reported a range of BCFs, 1.5 to 3.6, for the transfer of Aroclor 1254 into milk fat, with an apparent inverse relationship between the PCB concentration in feed and the estimated BCF (i.e. the BCF decreases as the PCB concentration increases). In its 2003 draft HHRA, EPA selected the maximum value reported for Aroclor 1254 (3.6) based on the rationale that the range of BCFs (3 to 3.6) was selected from studies in which dietary concentration for the test animals was in the range of dietary concentrations predicted in the assessment (i.e., <1 ppm PCBs) (EPA, 2003, Vol. V, p. 4-14). This value was retained in the 2005 updated HHRA (Vol. V, p. 4-27). However, unlike the June 2003 version of the HHRA, where the maximum floodplain soil concentration modeled was 2.0 ppm, the revised report attempts to model milk and beef concentrations in animals exposed to soil concentrations as high as 25 ppm. Therefore, EPA's justification for using the higher BCF does not apply to the analyses in the revised deterministic approach. Rather, since the revised HHRA is modeling PCB concentrations that range over an order of magnitude, GE believes that the mean of 2.6 of the values from the report by Fries (1996) should be used for the deterministic approach.

RESPONSE GE-30:

Increasing the modeled soil total PCB (tPCB) concentrations up to 25 mg/kg does not appreciably change the weighted average dietary concentration of tPCB for beef and dairy cattle because only a small percentage of the animals' diet consists of soil. Commercial dairy cattle, backyard dairy cattle, and commercial beef cattle were assumed to eat diets with tPCB concentrations less than 1 mg/kg. Backyard beef cattle were assumed to eat diets with tPCB concentrations slightly greater than 1 mg/kg, with the PBA incorporating a range of 1.3 to 1.6 mg/kg. As explained in Table 4-7 of HHRA Volume V, these concentrations are lower than the maximum assumed total PCB concentration in soil of 25 mg/kg because the cattle's diet does not consist entirely of soil. For example, the 1.6 mg/kg dietary concentration is based on an assumed backyard beef cattle diet that consists of 3% soil and 97% grass-based feed.

Fries (1996) reported bioconcentration factors (BCFs) for Aroclor 1254 transfer to milk fat ranging from 1.5 to 3.6. The HHRA explains the reason for selecting 3.6 for the point estimate assessment as follows:

> BCFs on the low end of this range were measured when animals were exposed to dietary PCB concentrations of 5 to 50 ppm. BCFs on the high end of this range were measured when animals were exposed to dietary PCB concentrations below 1 ppm. The grass and corn concentrations anticipated on current and possible future agricultural parcels are closer to 1 ppm than 5 to 50 ppm. Therefore, BCFs on the high end of the range measured in the studies of Aroclor 1254 are more applicable to the GE/Housatonic River Site (see page 4-27 of Volume V).

The maximum weighted average tPCB dietary concentration of 1.6 mg/kg is similar to doses upon which the selected BCF is based (Fries, 1996). Therefore, the rationale for the use of the higher BCF remains applicable and EPA's justification for using a BCF of 3.6 does apply to all of the assumed tPCB soil concentrations in the revised assessment.

Reference:

Fries, G.F. 1996. Ingestion of sludge applied organic chemicals by animals. *Sci. Total Environ*. 185:93-108.

5.2 Probabilistic Assessment

In the revised HHRA, EPA has added probability analyses, including an MCA and a PBA, to the agricultural products consumption assessment, which were not presented in the 2003 draft. The purpose of the MCA and PBA was to characterize the variability and, for the PBA, the uncertainty inherent in the deterministic approach (see Vol. V, p. 6-1). To do so, probability distributions of exposure variables replaced some of the point estimates used in the deterministic analyses. Tables associated with Section 6 (Vol. V) of the revised HHRA summarize the inputs used for the MCA and identify the types of distributions assumed for each in the assessment. Unexpectedly, for many (and for some of the scenarios, the majority) of the input variables, the HHRA continues to use point estimates as inputs to the MCA, rather than replacing them with distributions of values. Since the purpose of developing these alternative approaches was to quantify the effect of variability on the risk estimates, it is puzzling that point estimates were retained for so many of the inputs.

This approach is not necessary or warranted. As described in detail below, while site-specific data were not available on many of the inputs and in some cases only limited information exists in the published scientific literature, input distributions can be developed for some of the critical exposure inputs based on available data. The significance of EPA's choice to use point estimates rather than distributions is clear when evaluated in the context of the Sensitivity Analysis, which is also contained in Section 6. For example, of the three variables that contributed most significantly to the uncertainty and variability in the backyard beef consumption model (soil-to-

grass TFs, soil ingestion by farm animals, and human consumption rates), two are represented in the MCA by point estimates rather than distributions (Vol. V. p. 6-33). The benefits that should have been provided by developing the MCA for these exposure pathways are lost when the critical inputs are not changed from the deterministic approach.

RESPONSE GE-31:

The purpose of including a probabilistic risk characterization was to quantify variability and uncertainty, not just variability as the comment suggests. MCA inputs were defined as point estimates in cases where insufficient data were available to quantitatively distinguish variability from uncertainty, and for which uncertainty likely dominated. However, these inputs were defined in the PBA by using p-boxes (which include all plausible values) that account for both variability and uncertainty. The effect on risk results of the variability and uncertainty of these inputs was examined in the sensitivity analysis.

The rationale for this approach is specific to each parameter, as discussed in Section 6 of HHRA Volume V. EPA quantitatively described variability for all parameters with a sufficient quantity and quality of data relevant to the site (e.g., animal product fat contents, food consumption rates, cooking loss, exposure duration). This approach necessarily results in differences in the type and number of distributions used for the different pathways, and EPA notes that the Agricultural Product Consumption risk characterization (HHRA Volume V) includes more inputs without a quantitative description of variability alone than the Fish and Waterfowl risk characterization (HHRA Volume IV). This difference is simply a function of the amount and quality of data that are available and relevant to site-specific conditions. Because the full range of possible intake values for each parameter was evaluated in the PBA, the full benefit of the quantitative uncertainty analysis has been realized in that assessment.

With regard to the three input parameters specifically noted in the comment (food consumption rates, soil ingestion by farm animals, and grass transfer factors [TFs]), EPA notes that variability and uncertainty about food consumption rates were quantitatively described in the HHRA, as acknowledged in the comment. The PBA for soil ingestion by farm animals included p-boxes that ranged from 1 to 3% for beef cattle, and 8 to 12% for poultry. These ranges are small, so even if sufficient data were available to define a narrower range that quantitatively describes variability, the overall effect on the results would be minor.

The soil-to-grass TF p-box involves a larger range of values. EPA considered two approaches to define soil-to-grass TFs: (1) use site-specific data to define a variability distribution and quantify uncertainty about the parameters of this distribution; or (2) use site-specific data to define variability and relevant literature-based data to define uncertainty. Few site-specific data are available to define a variability distribution. Also, the range of site-specific TFs is similar to applicable data from the literature (see Table 4-5 of HHRA Volume V). Consequently, EPA concluded that there are insufficient data available to quantitatively distinguish variability and uncertainty.

Bioavailability of tPCBs from Soil

The HHRA uses a point estimate bioavailability factor of 100 percent in the MCA (Vol. V, p. 620). The rationale presented for using this approach is that insufficient data were available for tPCBs that would allow EPA to define the variability in this value or to provide a better estimate. The HHRA itself, however, provides substantial information about the variability associated with this important input to modeled exposures. In fact, it incorporates that variability into the PBA for agricultural products.

EPA's choice to assume that tPCBs in soil are 100 percent bioavailable has a substantial impact on the risk estimates for the agricultural product consumption pathways. This is because, as modeled, the soil ingestion pathway accounts for 55 percent of the risk estimated for the commercial beef consumption pathway, 32 percent of the risk estimated for the consumption of dairy products from backyard farms, and 36 percent of the risk estimated for the consumption of beef from backyard farms. In addition, it accounts for 100 percent of the tPCB intake by free-range poultry and, therefore, 100 percent of the risk estimated for the consumption of meat and eggs from those birds.

These pathways account for the highest cancer risks and noncancer hazards in the agricultural products consumption assessment. While data specific to the bioavailability of PCBs in soil ingested by farm animals are limited, there are data that can be used to develop distributions for the purpose of investigating the impact that EPA's arbitrary assumption of 100 percent has on the risk estimates.

For example, Ruby et al. (2002) reported that the bioaccessibility (a surrogate for oral bioavailability) of low concentrations of polychlorinated dioxins and furans ranged from 19% to 34%. Similar results were reported by Hack and Selenka (1996) for PCBs in a "standardized gastro-intestinal model." The HHRA itself cites the long-term feeding study in chickens by Stephens et al. (1995) and concludes that "these findings suggest that aging of contaminants in soil may reduce bioavailability" (Vol. V, p. 4-32). Although that study investigated the behavior of dioxins and furans, the HHRA recognizes that these classes of compounds (i.e., persistent, organic, lipophilic compounds) behave similarly in the environment because they share important physical/chemical properties (Vol. V, p. 6-20). In addition, as shown in Table 4-8b of Volume V of the HHRA, EPA recognizes the reduced bioavailability of dioxin-like PCB congeners. That table reports that "predicted absorption" values for the dioxin-like PCB congeners range from 41 to 71 percent.

For the PBA, the HHRA acknowledges that bioavailability is not 100 percent, and in Section 6.5.3.4 explains its decision to set a range for bioavailability of PCBs from soil relative to feed. In fact, the HHRA provides information on a potential range of bioavailability factors that might be used to develop a distribution in the MCA. It is unclear why EPA did not use the information presented in Section 6.5.3.4, or the information included in Attachment I of GE's comments on the 2003 HHRA (AMEC and BBL, 2003), to develop a probability distribution for this important input factor. Since direct soil ingestion is a significant and, in some cases, the only modeled uptake mechanism for farm animals and subsequent human exposures by these pathways, EPA should revise its approach to include an input distribution for this important variable to

demonstrate the impact of the variability in this parameter on final risk estimates for the agricultural product consumption pathways.

RESPONSE GE-32:

EPA did not assume that PCBs in soil are 100% bioavailable. Instead, in the point estimate risk characterization and MCA, EPA assumed that the bioavailability of PCBs in soil is equal to PCB bioavailability from normal feeds. Although PCBs may be less available in soil than in normal feeds, there is no consistent body of evidence that would provide a basis for assigning a value or distribution to the variability with confidence. Therefore, a value of 1 was used for soil bioavailability in the MCA, and a range of values (i.e., 0.65 to 1) was used for this parameter in the PBA (see Section 6.5.3.4 in HHRA Volume V).

The mammalian BCFs for the congeners and mixtures listed in Table 4-8a of HHRA Volume V were based on studies in which the contaminants were incorporated in normal diets. Thus, the BCFs already account for reduced bioavailability of PCBs from the feed matrix; therefore, only the reduced bioavailability from soil relative to feed must be evaluated in conjunction with such feed-based BCFs.

The Ruby et al. (2002) and Hack and Selenka studies examine bioavailability of PCBs, dioxins, and furans from soil instead of bioavailability from soil relative to feed. In addition, they use in vitro methods that have not been validated against a living animal model. The bioavailability p-box used in the HHRA is based on data from in vivo studies, which were preferred by EPA over in vitro studies.

Ruby et al. (2002) concluded that the bioavailability from soil for a number of dioxin and furan congeners ranged from 19 to 34%, and Hack and Selenka (1996) used a gastrointestinal model to estimate bioavailability from soil of selected PCB congeners (i.e., PCB-28, -52, -101, -138, -153, -180) of 33 to 64%. In developing the PBA lower bound of 0.65, EPA assumed that tPCB bioavailability from soil ranged from 30 to 40%. Neither Ruby et al. (2002) nor Hack and Selenka (1996) report the denominator needed to calculate bioavailability in soil relative to feed (i.e., bioavailability from feed). Nevertheless, if Ruby et al. (2002) results were used instead of EPA's soil bioavailability assumption of 30 to 40%, the lower bound of the p-box for this input would decrease, increasing the uncertainty about this input on cancer risk and noncancer hazard estimates. If EPA used only the PCB congener data from Hack and Selenka (1996) because of their potentially greater relevance to tPCBs, the lower bound of the p-box would not change appreciably.

References:

Hack, A. and F. Selenka. 1996. Mobilization of PAH and PCB from contaminated soil using a digestive tract model. *Toxicol. Lett.* 88:199-210.

Ruby, M.V., K.A. Fehling, D.J. Paustenbach, B.D. Landenberger, and M.P. Holsapple. 2002. Oral bioaccessibility of dioxins/furans at low concentrations (50-350 ppt toxicity equivalent) in soil. *Environ. Sci. Technol.* 36:4905-4911.

Soil-to-Grass Transfer Factor

The soil-to-grass transfer factor is also a critical input parameter in the probabilistic exposure assessment for the agricultural product consumption pathways. As shown in the HHRA, the soil-to-grass exposure pathway accounts for 44 percent of the risk estimated for the commercial beef consumption pathway, 68 percent of the risks in the backyard dairy farm scenario, and 64 percent of the risks in the backyard beef consumption scenario (Vol. V, pp. 5-4, 5-5, 5-6).

In the MCA, the HHRA uses a point estimate of 0.036 for this transfer factor (Vol. V, p. 6-19, Figure 6-13), despite information provided in the previous draft HHRA and the PBA discussion in this draft of the HHRA, which indicates that this transfer factor may span an order of magnitude or more. The PBA specifically describes a range from 0.0098 to 0.094 (Vol. V, Table 6-4). Thus, EPA's approach does not consider any of the variability associated with this important parameter.

As stated in GE's Comments on the 2003 HHRA (AMEC and BBL, 2003), the methodology used to obtain the site-specific soil-to-grass transfer factors likely overestimated the actual transfer that is occurring over time. In fact, in the revised HHRA, EPA acknowledges that the grass data "represent an upper bound on exposure concentrations of PCBs for grazing cattle" (Vol. V, p. 4-7). It goes on to state, in Section 7.2.2.1.1 (p. 7-5), that there is a range of literature-based values that span several orders of magnitude, indicating that there is enormous variability associated with this parameter.

There are substantial amounts of data available on soil-to-grass transfer. These include studies discussed by EPA in the HHRA, as well as other studies identified in GE's comments on the earlier draft (AMEC and BBL, 2003). In the 2003 version of the Uncertainty Analysis, EPA provided transfer factors obtained from ATSDR (Section 6.3.2.1) that ranged over two orders of magnitude. However, rather than incorporating the breadth of information available, EPA selected a soil-to-plant transfer factor at the high end of the range found in the scientific literature. Thus, instead of utilizing the advantages, and stated purpose, of the MCA to assess the effect of variability on risk estimates, EPA relied solely on the "upper bound" transfer factor to estimate PCB intake from grass, thereby minimizing the value of the MCA.

GE believes that EPA should revise its approach to incorporate a distribution of soil-to-grass transfer factors based on site-specific and literature-based values. This will allow the MCA to consider the impact of the enormous variability associated with this important parameter and provide more insight into the range of potential exposures and risks that are potentially associated with the exposure pathways that include this transfer route.

RESPONSE GE-33:

EPA did not ignore variability in the soil-to-grass transfer factor or any other input. However, in some cases, EPA concluded that it was not possible to quantitatively distinguish variability from uncertainty due to the limited information

available from the studies. In these cases, uncertainty was assumed to be greater than variability; therefore, the range of possible values the input might assume was defined with a p-box instead of a precise probability distribution. EPA evaluated the influence of this input on cancer risks and noncancer hazards (see Tables 6-23 to 6-25 of HHRA Volume V).

In addition, EPA conducted a thorough review of the literature and GE's previous comments regarding PCB transfer from soil to grass and other plants; this review is summarized in Table 4-5 of HHRA Volume V. It is incorrect to state that EPA ignored this information; the information was incorporated into the p-box for the soil-to-grass transfer factor.

Soil-to-Plant Transfer Factor

As discussed in Section 5.1, EPA used the maximum wet weight soil-to-plant transfer factor for corn to estimate tPCBs in "exposed" surface vegetables and fruits. The point estimate TF that was used in the deterministic risk calculations was also used in the MCA (Vol. V, p. 6-16). This maximum value overestimates the amount of tPCBs on ingested vegetables and fruits for all the reasons previously discussed in Section 5.1.

While a "Produce Loss Factor" was used in both the MCA and PBA assessments to account for the removal of soil during the processing of fruits and vegetables for consumption, these assessments did not consider the additional impact that washing would have on the exposure estimated using the TF. For example, the maximum loss for "exposed vegetables" was assumed to be 0.64 (Figure 6-51), which assumes that 36 percent of the PCBs transferred from soil to the surface of the vegetable, as quantified by the TF point estimate, were consumed. Likewise, for "exposed fruits," the maximum loss was assumed to be 0.41 (Figure 6-53); thus, it was assumed that the ingested PCBs were 59 percent of the total estimated by the TF. The minimum values of these distributions were both set at 0, indicating the potential for no loss of PCBs before consumption. It would seem appropriate in this assessment to evaluate the effect of both washing and preparation loss on exposure and risk estimates. While there is a potential that some fruits and vegetables will be consumed without washing, assuming that this never occurs is not a reasonably anticipated occurrence. Rather, including the effect that even periodic washing would have on PCB concentrations, and therefore extending the maximum ranges of the distributions, is consistent both with the purpose of the MCA and with EPA guidance for including high-end, but not worst-case, exposure assumptions (EPA, 1992b, 1995). The potential effects that these two activities have on the deposited PCB concentrations would range from very small impacts (very little removed from either washing or peeling and a loss factor approaching 0) to almost the complete elimination of this as an exposure route (and a loss factor approaching 100 percent).

RESPONSE GE-34:

Loss due to washing produce prior to consumption was accounted for in defining p-boxes for these inputs as described in HHRA Volume V, Sections 6.5.6.1.1 and 6.5.6.1.3. Site-specific data were used to define p-boxes, including data from Sawhney and Hankin (1984) for crops that were washed in warm water and

brushed prior to laboratory analysis (see discussion of these data in Section 2.3.7).

Reference:

Sawhney, B.L. and L. Hankin. 1984. Plant contamination by PCBs from amended soils. *Journal of Food Protection* 47(3):232-236.

Mammalian Bioconcentration Factors

For the MCA, the HHRA uses a point estimate of 3.4 to represent the mammalian BCF (Vol. V, p. 6-16), rather than incorporating an input distribution of values based on available data. Tuinstra et al. (1981) orally dosed lactating cows with "lower chlorinated biphenyls and technical grade PCB mixture Aroclor 1260" and reported accumulation factors into milk fat for individual congeners ranging from 0.1 to 5.4, with a mean of 2.22. Fries (1996) reported BCFs into milk fat for Aroclor 1254 ranging from 1.5 to 3.6.

As noted in the HHRA (p. 6-16), BCFs for exposures of less than 1 ppm in the diet exceeded 3.0. However, as discussed previously, unlike the June 2003 draft of the HHRA, where the maximum floodplain soil concentration evaluated was 2.0 ppm, the revised HHRA attempts to model milk and beef concentrations in animals exposed to soil concentrations as high as 25 ppm. Consequently, EPA's rationale for using the higher BCF, based on a dietary concentration range for test animals of <1 ppm PCBs, does not apply to the MCA. Rather, the available information used to derive the point estimate (Fries, 1996) should be considered, along with the data provided by Tuinstra et al. (1981), to develop an appropriate distribution for the mammalian BCF input variable for the MCA.

RESPONSE GE-35:

Please see Response GE-30. In addition, the data were considered and used to define p-boxes because uncertainty about the BCFs as applied to site-specific conditions, and especially the use of BCFs based on dairy cows to evaluate beef cattle, is greater than the understanding of variability. Also, use of data from Tuinstra et al. (1981) to derive a BCF for Aroclor 1260 would be difficult. For example, some of the congeners in Tuinstra et al. (1981) were listed by retention times and were not identified.

Reference:

Tuinstra, L.G.M.Th, K. Vreman, A.H. Roos, and H.J. Keukens. 1981. Excretion of certain chlorobiphenyls into the milk fat after oral administration. *Neth. Milk Dairy J.* 35:147-157.

Steady-State Conditions

In addition to the above instances in which EPA has used point estimates rather than distributions in the MCA, the HHRA's probabilistic analyses fail to take adequate account of variability in animal tissue levels (meat, milk, and eggs) that may result from intermittent PCB

exposures. One of the issues raised in GE's comments on the 2003 draft HHRA (AMEC and BBL, 2003) related to the enormous uncertainty associated with EPA's assumption that animals that had contact with PCBs in floodplain soils were at steady-state. In the revised Uncertainty Analysis, EPA acknowledges, at least qualitatively, that fluctuations in concentrations in the diet will result in fluctuations in milk concentrations (Vol. V, p. 7-9). This fluctuation is a result of the fact that non-steady-state conditions exist for lactating animals. In its discussion, EPA concludes that the alterations in milk concentrations that would result from physiological changes would be less than the changes that would result from the variability in contaminant concentrations in feed (Vol. V, p. 7-9). That is not necessarily true.

In an important study cited by EPA for determining milk PCB concentrations, Thomas et al. (1999) reported that under constant exposure conditions, even while PCB intakes increased (because of increased silage consumption), milk concentrations dropped by an average of 25%. It is likely that removal of lactating animals from access to feed containing PCBs (either grass, silage, or soil) for even short periods of time would result in a significant reduction in milk concentrations. Thus, the changes in milk concentrations due to physiological changes may be less than, equal to, or greater than those attributed to fluctuations in concentrations of PCBs in feed. Because of the variability in exposure conditions, a general conclusion about the quantitative impact of this effect on human exposure cannot be reached.

This does not, however, preclude consideration of this variability in the quantitative estimate of exposure and risk associated with the dairy consumption pathways. GE recommends that EPA use a non-steady-state model (e.g., a pharmacokinetic model) in the MCA to address this important source of uncertainty. Models that evaluate intermittent exposures have been used in risk assessment, including the microexposure event simulation for fish consumption in the Housatonic River HHRA (Vol. IV. Section 6.3). Adopting the principles of the microexposure event analysis, and incorporating a consideration of the pharmacokinetics of PCBs in agricultural animals, which can be gleaned from the Thomas et al. (1999) report, would provide a quantitative method for evaluating the impact that this variability has on animal product PCB concentrations, and consequently risks to human consumers.

RESPONSE GE-36:

This question was addressed on page 4-17 and pages 7-8 to 7-9 of HHRA Volume V, and the modeling approach used in the revised assessment has not changed from the June 2003 assessment. Therefore, the comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

6.0 INTEGRATED RISK CHARACTERIZATION

EPA has added an integrated risk characterization in Volume 1, Chapter 10 of the HHRA, in response to comments raised by the peer reviewers. This section includes a perspective on the TEQ exposures that are estimated for several of the exposure scenarios and also discusses

potential concentrations of PCBs in breast milk. GE believes that these discussions need to be revised to discuss additional uncertainties that potentially affect the conclusions drawn.

RESPONSE GE-37:

The EPA responses to the GE comments summarized above are provided below in the detailed responses to Sections 6.1 and 6.2 of the GE comments, Responses GE-38 through GE-39, respectively.

6.1 Perspective on TEQ Exposures

In Section 10.2 of Volume I, EPA presents a "Perspective on TEQ Exposure," which compares the exposure levels modeled in the HHRA with background intake levels in the current American food supply that are provided in the published literature. The purpose of this section is to advise the public whether the substitution of agricultural products or fish and waterfowl obtained from the study area would increase intake of TEQ by Housatonic River Area (HRA) residents over TEQ intake that would occur from similar products obtained from national chain grocery stores.

The HHRA compares the TEQ concentrations predicted for milk, beef, and poultry products, based on a 2 ppm soil concentration, with measurements of TEQ in these products as found in the national food supply. It concludes that TEQ concentrations in milk obtained from commercial dairies in the HRA are similar to the national supply, but that milk from backyard farms and beef and poultry from both commercial and backyard farms in the HRA have greater TEQ concentrations than does the national food supply.

In discussing these comparisons, it is important that EPA express the degree of uncertainty associated with the comparisons. For example, Table 10-8 of the HHRA indicates that the PCB-related TEQ concentration predicted to be present in the fat of backyard-raised beef in the HRA is 171 times higher than the PCB TEQ concentration in beef fat in the national food supply. However, as discussed in these comments, the analyses of backyard beef are likely to substantially overestimate concentrations in beef fat due to a combination of highly conservative transfer and bioconcentration factors. In addition, even without the use of input distributions for many of the parameters in the MCA, the predicted risks, and hence exposures, in the MCA range over nearly two orders of magnitude at the 2 ppm soil concentration (see Vol. V, Table 615), so that the background level reported by EPA might very well fall within the range of predicted beef fat concentrations. Furthermore, if the MCA had used input distributions for many of the parameters for which it used point estimate values, the range of potential risks would likely have been even greater. Thus, the HHRA's comparison of its hypothetical and highly conservative exposure estimates for the agricultural pathways to national TEQ data should be qualified to account for the enormous uncertainties associated with the HHRA's predictions.

Finally, the HHRA discusses these agricultural exposures as if they are actually occurring. There are enormous uncertainties, however, associated both with the likelihood of occurrence of the modeled scenarios and with the exposure estimates derived using EPA's approach to the agricultural risk assessment. EPA should revise this section to provide more discussion of the potential uncertainties associated with the predictions, and should note that exposures to agricultural products grown or raised in the HRA may or may not have higher levels of TEQ than are found in the background food supply.

RESPONSE GE-38:

The calculation of TEQ from dioxins, furans and dioxin-like PCBs predicted for milk, beef, and poultry products, and the associated uncertainty were discussed in HHRA Volume I, Section 9, with complete details provided in Volume V, Appendix D. Section 9 and Appendix D were referenced at the beginning of the discussion of agricultural products in Section 10.2.

EPA highlighted the uncertainty associated with predicted TEQ concentrations in agricultural products by limiting discussion of TEQ risks to the uncertainty analysis section of HHRA Volume V, rather than including it in the main risk characterization. In addition, a quantitative uncertainty analysis of PCB-126 exposure on commercial dairy farms was performed to further illustrate the uncertainty associated with this exposure pathway (see Addendum 6.1 of HHRA Volume V).

It is correct that the backyard beef cattle comparison shows the greatest difference between predicted concentrations for the Housatonic River Area (HRA) and measured concentrations for the U.S. food supply. As explained in Section 4.5.2 of HHRA Volume V, predictions for backyard animals often exceed U.S. food supply concentrations because backyard animals are assumed to have greater soil and grass-based feed exposures than commercial animals. Section 4.5.2 also clearly describes other potential reasons for the difference between HRA predictions and the U.S. food supply that apply to both commercial and backyard beef farms. In addition, tPCB concentrations on U.S. farms with beef cattle could differ from the 2 mg/kg assumption. In Section 4.5.3, EPA explains that the predictions for poultry also differ from the U.S. food supply because the predictions are based on free-range poultry with diets consisting of as much as 10% soil, whereas commercial poultry operations typically involve little to no soil exposure.

EPA disagrees that uncertainties were ignored as a result of using point estimates for some MCA inputs. EPA did not ignore uncertainty and, in fact, added a PBA analysis for the purpose of quantifying uncertainty. The “range of likely risks” from the MCA is provided in the PBA. Additional information regarding this point may be found in Response GE-31.

Commercial dairy farms and at least one backyard beef operation exist in the HRA. As a result, some agricultural exposures are occurring in the HRA. However, farm-specific exposures were not evaluated directly because management practices and animal types on any given farm may change over time, and such farm-specific assessments would become obsolete when these changes occur. The hypothetical nature of the predictions is explained in the Executive Summary and Section 4.1 of HHRA Volume V and in Section 9 of HHRA Volume I, which is the summary of the agricultural product consumption risk assessment.

6.2 Breast Milk Pathway

In response to the comments of the peer reviewers, EPA has added an evaluation of the breast milk pathway by predicting estimated PCB and congener concentrations in breast milk and then comparing those estimated concentrations with available data about background concentrations of these compounds in breast milk (Vol. I, Sec. 10.3). EPA has calculated these concentrations using simplistic models, based on the dose levels estimated in the HHRA for adults in the fish consumption, waterfowl consumption, and backyard dairy farm milk ingestion scenarios. The HHRA acknowledges that there are uncertainties associated with the calculation of the breast milk concentrations, particularly as related to the range of half-life estimates available in the published literature (Vol. I, Section 10.3.1.2). It does not, however, adequately address either: (a) the additional uncertainties associated with the use of the simplistic model, which may or may not represent the actual mechanism of the concentration of PCBs into breast milk; or (b) the substantial uncertainties associated with deriving the estimated dose levels for the fish consumption, waterfowl consumption, and backyard dairy milk ingestion scenarios, which are discussed at some length in these comments and in GE's previous comments (AMEC and BBL, 2003; GE, 2003). GE recommends that EPA add a subsection to Section 10.3 of the HHRA that specifically discusses all of the potential sources of uncertainty associated with the breast milk concentration estimates that have been derived.

RESPONSE GE-39:

The breast milk pathway and associated uncertainties were evaluated in response to comments received from the Peer Review Panel. An equilibrium partitioning model was used to estimate contaminant concentrations in breast milk fat. While this is a simple model, that does not necessarily mean it is incorrect or inappropriate. EPA acknowledges that there is model uncertainty associated with every model, and the use of a different model may give a different estimate of contaminant concentrations in breast milk fat. Alternate model estimates could be higher or lower than the estimate using the model incorporated into the HHRA. It should be pointed out that the limits of the model were discussed in Section 10.3.1, including its applicability only under steady-state conditions and that the parameters used in the calculation are consistent with achieving steady-state conditions.

EPA recognizes that the dose levels incorporated into the calculation for contaminant concentrations in breast milk fat were based on the equations and parameters described for the fish consumption, waterfowl consumption, and backyard dairy milk ingestion scenarios. The uncertainties associated with the calculation of these doses were described both qualitatively and quantitatively in Sections 8 and 9 of Volume I, and in more detail in Volume IV (Appendix C) and Volume V (Appendix D). Section 10.3.1.1, Maternal Intake, describes the average daily dose (ADD) equation and makes explicit reference to these sections and volumes.

REFERENCES

AMEC Earth & Environmental and BBL Sciences. 2003. *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.* July 28.

ATSDR/MDPH. 2002. *Health Consultation, Assessment of Cancer Incidence Housatonic River Area, 1982-1994*. Massachusetts Department of Public Health, Bureau of Environmental Health Assessment, Community Assessment Unit. Under a Cooperative Agreement with the Agency for Toxic Substances and Disease Registry.

ATSDR. 2000. *Toxicological Profile for Polychlorinated Biphenyls (PCBs)*. U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Diseases Registry. November.

Calabrese, E.J. 2003. Letter from Edward Calabrese to K. Holtzclaw re: Soil Ingestion Rates. July 23. Exhibit E.1 to AMEC and BBL (2003).

Drilling, N, R. Titaman, and F. McKinney. 2002. Mallard (*Anas platyrhynchosl*). In: The Birds of North America, No. 658 (A. Poole and F. Gills, eds.). The Birds of North America, Inc., Philadelphia, PA.

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

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. 2005. *Human Health Risk Assessment GE/Housatonic River Site Rest of River.* Prepared by Weston Solutions Inc. for U.S. Environmental Protection Agency and U.S. Army Corps of Engineers. February.

EPA. 2003. *Human Health Risk Assessment GE/Housatonic River Site Rest of River*. Prepared by Weston Solutions Inc. for U.S. Environmental Protection Agency and U.S. Army Corps of Engineers. June

EPA. 2002. *Estimated Per Capita Fish Consumption in the United States*. U.S. Environmental Protection Agency. EPA-821-C-02-003. August.

EPA. 2001. Risk Assessment Guidance for Superfund, Volume I - Human Health Evaluation Manual Supplemental Guidance, Dermal Risk Assessment Interim Guidance. Public Review Draft. U.S. Environmental Protection Agency, Office of Emergency and Remedial Response, Washington, D.C. EPA/540/R/99/005. September.

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

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

EPA. 1993. *Wildlife Exposure Factors Handbook.* U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C. EPA/600/R-93/187. December.

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

EPA. 1992b. Final Guidelines for Exposure Assessment. U.S. Environmental Protection Agency, Washington, D.C. *Federal Register* 57(104):22888-22938. May 29.

Evrard, J.O. 2000. The conservation reserve program and duck and pheasant production in St. Croix County, Wisconsin. *Research Report 183.* Wisconsin Department of Natural Resources, October.

Fries, G.F. 1996. Ingestion of sludge applied organic chemicals by animals. *Sci. Total Environ.* 185:93-108.

GE. 2003. *Comments of the General Electric Company on EPA's Human Health Risk Assessment for the GE-Housatonic River Site – Rest of River.* Presentation to the Peer Review Panel. General Electric Company, Pittsfield, MA. November 18.

Greenwood, R.J., A.B. Sargeant, D.H. Johnson, L.M. Cowardin, T.L. Shaffer. 1995. Factors associated with duck nest success in the Prairie Pothole Region of Canada. *Wildlife Monographs* 128:1-57. Jamestown, ND: Northern Prairie Wildlife Research Center Home Page. http://www.npwrc.usgs.gov/resource/othrdata/nestsuc/nestsucc.htm (Version 02JUN99).

Gustavsson, P. and C. Hogstedt. 1997. A cohort study of Swedish capacitor manufacturing workers exposed to polychlorinated biphenyls (PCBs). *Am. J. Ind. Med.* 32(3):234-239.

Hack, A. and F. Selenka. 1996. Mobilization of PAH and PCB from contaminated soil using a digestive tract model. *Toxicol. Lett.* 88:199-210.

Harnois, M. and C.M. Smith. 2001. Memorandum from M. Harnois and C. M. Smith, Massachusetts Department of Environmental Protection, to Bryan Olson, U.S. EPA re: Huntingdon Life Sciences Study 00-3431. November 16, 2001.

Hawley, J.K. 1985. Assessment of health risk from exposure to contaminated soil. *Risk Analysis* 5:289-302.

Hepp, G.R. and F.C. Bellrose. 1995. Wood Duck (*Aix sponsa*). In: The Birds of North America, No. 169 (A. Poole and F. Gill, eds.). The Academy of Natural Sciences, Philadelphia, and the American Ornithologists' Union, Washington, D.C.

Kimbrough, R.D., M.L. Doemland, M.E. LeVois. 1999. Mortality in male and female capacitor workers exposed to polychlorinated biphenyls. *J Occup. Environ. Med*. 41(3):161-171

Knuth, B.A., N.A. Connelly, and B.E. Matthews. 1998. *Children's Fishing and Fish Consumption Patterns.* Cornell University Human Dimensions Research Unit. HDRU Series No. 98-3. May.

Loomis D, S.R. Browning, A.P. Schenck et al. 1997. Cancer mortality among electric utility workers exposed to polychlorinated biphenyls. *Occup. Environ. Med.* 54(10):720-728.

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.

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

MFWP. 2004. Animal Field Guide:Gadwall. Montana Fish, Wildlife and Parks (MFWP). http://fwp.state.mt.us/fieldguide/detail_ABNJB10160.aspx.

Roy, T.A., J.J. Yang, A.J. Krueger, and C.R. Mackerer. 1990. Percutaneous absorption of neat 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and TCDD sorbed on soils. *Toxicology* 10(1):308.

Ruby, M.V., K.A. Fehling, D.J. Paustenbach, B.D. Landenberger, and M.P. Holsapple. 2002. Oral bioaccessibility of dioxins/furans at low concentrations (50-350 ppt toxicity equivalent) in soil. *Environ. Sci. Technol.* 36:4905-4911.

Stanek, E.J. and E.J. Calabrese. 1992. Soil ingestion in children: Outdoor soil or indoor dust? *J. Soil Contam.* 1(1):1-28.

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.

Stephens, R.D., M.X. Petreas and D.G. Hayward. 1995. Biotransfer and bioaccumulation of dioxins and furans from soil: Chickens as a model for foraging animals. *Sci. Total Environ.* 175:253-273.

TER. 2003. *Housatonic River Floodplain User Survey Summary Report.* Triangle Economic Research (TER), Research Triangle, NC. January.

Thomas, G.O., A.J. Sweetman, and K.C. Jones. 1999. Input-output balance of polychlorinated biphenyls is a long term study of lactating dairy cows. *Environ. Sci. Technol.* 33:104-112.

Tuinstra, L.G.M.Th., K. Vreman, A.H. Roos, and H.J. Keukens. 1981. Excretion of certain chlorobiphenyls into the milk fat after oral administration. *Neth. Milk Dairy J.* 35:147-157.

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

ATTACHMENT A
LETTER FROM KEVIN W. HOLTZCLAW TO DR. DAVID L. EATON RE: COMMENTS OF THE GENERAL ELECTRIC COMPANY ON EPA'S DRAFT EXPOSURE AND HUMAN HEALTH REASSESSMENT OF 2,3,7,8-TETRACHLORODIBENZO-P-DIOXIN (TCDD) AND RELATED COMPOUNDS
MARCH 15, 2005

Kevin W. Holtzclaw
Manager - PCB Issues

General Electric Company
Corporate Environmental Programs
3135 Easton Turnpike,W1B, Fairfield, CT 06431
*203 373-2610,Fax:-2650, DC:8*229-2610*
email:kevin.holtzclaw@corporate.ge.com

March 15, 2005

Dr. David L. Eaton, Chair

Committee on EPA's Exposure and Human Health Reassessment of TCDD and Related Compounds
The National Academy of Sciences
Board on Environmental Studies and Toxicology
500 Fifth Street, N.W.
Washington, DC 20001

RE: Comments of The General Electric Company on EPA's Draft Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds

Dear Dr. Eaton:

The General Electric Company ("GE") appreciates the opportunity to present to the National Academy of Science's Committee ("NAS Panel") on EPA's Exposure and Human Health Reassessment of TCDD and Related Compounds ("draft Reassessment") its comments and research concerning a fundamental flaw in the draft Reassessment: the belief that certain PCB congeners are the toxic equivalents of dioxin. This belief is contradicted by a substantial body of evidence demonstrating that the internationally recognized criteria for application of the TEQ approach are not met for any of the PCBs. Remarkably, the draft Reassessment does not even mention the vast majority of this evidence. If EPA had considered this evidence, it would have had no choice but to conclude that PCBs are not the toxic equivalents of dioxin and should not be included in the TEQ risk assessment methodology.

This fundamental flaw in the draft Reassessment is important because the application of the TEQ approach to so-called "dioxin-like" PCBs results in a significant overestimate of the risks to human health from exposure to the compounds in question. In the draft Reassessment, EPA calculates that the "dioxinlike" PCBs contribute up to one-third of a typical person's Total Daily Intake of TEQ. Using the TEQ approach, EPA treats this intake as if it is dioxin itself. The net result is that EPA concludes that humans are exposed to more "dioxin", in the form of TEQ, than they are exposed to dioxin per se. Moreover, EPA concludes that the risks arising

from PCB exposure, calculated as TEQ, are substantially greater than they would be if the risks from exposure to PCBs were calculated using the available empirical, PCB-specific data.

As many of the members of the NAS panel are undoubtedly aware, there is a vast literature on the toxicity of PCBs and this class of compounds has been closely regulated by EPA using risk-based standards. Both Reference Doses ("RfDs") and Cancer Slope Factors ("CSFs") have been established by EPA for PCBs and are available on EPA's Integrated Risk Information System ("IRIS") database. The current CSFs for PCBs were established by EPA in 1996. In that year, EPA performed a comprehensive reassessment of the carcinogenicity of PCBs and, based on animal data, established an upper bound CSF for PCB exposure to the higher chlorinated PCB congeners of 2.0 $(mg/kg\text{-}day)^{-1}$ EPA, 1996 (PCBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures) ("1996 PCB Reassessment"). Risks of exposures to PCBs likely to involve lesser chlorinated PCBs were to be assessed using lower CSFs. Id. Applying the TEQ methodology to PCBs, as urged by the draft Reassessment, would have the effect of increasing the estimated PCB cancer risk by up to 30 times over the upper bound CSF of 2.0 $(mg/kg\text{-}day)^{-1}$. This increase in estimated risk does not result from any error in, or updating of, the 1996 PCB Reassessment -- indeed, the draft Dioxin Reassessment does not even mention the 1996 PCB Reassessment. Instead, the increase in estimated risk is based on a surrogate risk metric, i.e., the CSF for 2,3,7,8-tetrachlorodibenzo-p-dioxin ("TCDD") of 1.4×10^{6} $(mg\ TCDD\ TEQ/kg/day)^{-1}$.

The remainder of this letter summarizes the evidence that the NAS panel should consider when determining whether PCBs should be evaluated using the TEQ approach and included in the dioxin reassessment. Some of that evidence is contained in or referenced by Attachments to this letter. Additional evidence has been submitted for publication, and we appreciate the opportunity to provide a summary of this evidence in a presentation to the NAS Panel at the Panel's March 21, 2005 meeting.

Evidence that the TEQ Approach Substantially Over-Predicts the Carcinogenicity of PCBs

EPA's error in applying the TEQ approach to PCBs results largely from the Agency's failure to validate the risk predictions of the TEQ approach for PCBs by comparing them with empirical data on the effects of PCB exposures in animal studies. A basic premise of the TEQ approach 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). There are at least two ways to test the validity of this premise in the case of PCBs. The first is to calculate CSFs for PCB-derived TEQ in rodents, and to compare those CSFs to the CSFs derived from rodents exposed to dioxin. The second is to calculate the cancer potency of PCB mixtures using TEQs, and to compare the calculated CSFs to CSFs derived in the 1996 PCB Reassessment for use in assessing human health risks.

Comparison of rodent CSFs

To evaluate the validity of the TEQ methodology in estimating the cancer potency of PCB mixtures, Dr. Russell Keenan and co-workers used the results of two-year cancer bioassays involving four PCB mixtures of known composition that were fed to Sprague-Dawley rats. Those tests are described and their results presented in a journal article that has been submitted

for publication and in a short paper that appears in the Dioxin 2003 conference proceedings (Attachment 1a).[1]

In one test, CSFs in rats were determined for the "dioxin-like" components of the four PCB mixtures and compared to that of TCDD (based on a similar two-year cancer bioassay of TCDD in rats). This was done by analyzing the four PCB mixtures to determine their concentrations of dioxin-like congeners and then, for each mixture, determining the total TEQs of the mixture by summing the products of the concentrations of dioxin-like congeners and the TEFs for those congeners, as set forth in the draft Reassessment. The empirically-determined CSF for each mixture was then divided by the TEQ of the mixture to derive the CSF per unit TEQ for the mixture. If the TEQ approach is valid, the CSF per unit of TEQ for each of the PCB mixtures should equal the TEQ of TCDD, i.e., the tests should give equivalent results.

The results of this "litmus test" do not support the TEQ approach. The CSFs for the TEQ in PCB mixtures were not equal to the CSF for TCDD; in fact, the experimentally-determined CSFs for the PCB mixtures based on TEQ varied over a 24-fold range. This discordance demonstrates that the TEQ approach for evaluating cancer risks associated with exposure to PCB mixtures is seriously flawed.

Comparison of human CSFs

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 the 1996 PCB Reassessment. If the TEQ method were 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 empirically derived CSFs. In fact, the comparisons showed that the TEQ-based CSFs were considerably greater than the empirically-derived CSFs, indicating that the TEQ approach substantially over-predicts the carcinogenic potency of PCB mixtures.

In each case, Dr. Keenan's findings are in sharp contrast to the results that one would expect if the fundamental premise of the TEQ method were true. Each of these analyses indicates that there is a fundamental fallacy associated with the use of the TEQ approach for estimating the carcinogenic potential of PCB mixtures.

Additional evidence from the National Toxicology Program study

The inability of the TEQ method to predict the carcinogenicity of PCB mixtures is confirmed by the results of the recent 2-year bioassays of the National Toxicology Program ("NTP") on TCDD, PCB 126, 2,3,4,7,8-pentachlorodibenzofuran, and a mixture of these three compounds (NTP, 2003), followed by bioassays of PCB 153, a mixture of PCB 153 and PCB 126, and a mixture of PCB 118 and PCB 126 (NTP, 2004). NTP conducted this series of bioassays in

[1] Note that similar papers, which are contained Attachments 1b, were presented to the EPA Science Advisory Board in connection with that group's review of the Dioxin Reassessment in 2000 and 2001. The current version of the draft Reassessment does not reference this work.

female Harlan Sprague-Dawley rats to evaluate the chronic toxicity and carcinogenicity of dioxin, "dioxin-like" compounds, structurally-similar PCBs, and mixtures of these compounds. NTP conducted its evaluation to address "the lack of data on the adequacy of the TEQ methodology for predicting relative potency for cancer risk" (NTP, 2003).

The initial NTP (2003) bioassay results provide evidence of non-additive interactions among "dioxin-like" compounds, inconsistencies in dose-response depending on the dose metric analyzed, and different relative potencies depending on endpoint observed. These results undermine the assumptions essential to the application of the TEQ approach to PCBs. We are preparing two papers for publication, which explore these issues in greater depth and would be pleased to submit these manuscripts to the panel upon their acceptance. A brief summary of our findings will be presented to the Panel on March 21.

Evidence from human studies

Although it is clear that the TEQ approach substantially over-predicts the animal carcinogenicity of PCBs, it should also be noted that the predictions of the TEQ approach for human carcinogenicity are wholly unsupported by available human data. The TEQ approach as set forth in the draft Reassessment ignores the vast body of PCB human epidemiological studies indicating that PCBs are very likely not human carcinogens at all. More than 50 peer-reviewed, epidemiological cancer studies specific to PCBs have been published over the past 30 years. Many of those studies involved thousands of workers with occupational exposures far greater than those that would result from environmental exposures. None of those studies support a finding that PCBs are human carcinogens. One study, Kimbrough et al. (1999), as updated by Kimbrough et al. (2003), is particularly noteworthy.

Kimbrough et al. (1999) (Attachment 2) represents one of the largest occupational studies ever conducted of a population of workers that was heavily exposed to PCBs. The cohort consisted of 4,062 men and 3,013 women who worked between 1946 and 1977 at two General Electric capacitor manufacturing facilities. Jobs at the two facilities were classified as high or low exposure. The average follow-up time for the workers was 31 years, providing the longest latency period of any PCB-exposure occupational study. The cohort was followed through 1993, providing 120,811 person-years of observation for men and 92,032 person-years observation for women. There were 763 (19%) deceased males and 432 (14%) deceased females. Kimbrough et al. (1999) found that, compared to the general U.S. population, among all workers, including those classified as having the highest PCB exposure, there was no statistically significant increase in deaths due to cancer or any other disease. Moreover, the death rate due to all types of cancer combined was at or below the expected level.

The Kimbrough et al. (2003) (Attachment 3) study followed the cohort through 1998, providing 133,845 person-years of observation for men and 102,139 person-years observation for women. There were 1022 (25%) deceased males and 632 (20%) deceased females. The Kimbrough et al. (2003) update similarly found that, among all workers, including those classified as having the highest PCB exposure, there were no statistically significant increases in deaths due to cancer. There were also no statistically significant increases in cancer or other mortality associated with length of employment or latency.

Golden et al. (2003), a summary paper that discusses the findings of Kimbrough et al., as well as all of the other human evidence relating to the potential carcinogenicity of PCBs, is included in Attachment 4.[2] Golden et al. (2003) concluded that "[a]pplying a weight-of-evidence evaluation to the PCB epidemiological studies can only lead to the conclusion that there is no causal relationship between PCB exposure and any form of cancer . . ." A more detailed review of all the relevant human cancer studies involving exposure to PCBs is included on a compact disk contained in Attachment 6. That review also concluded that the weight of the human evidence does not support an association, much less a causal relation, between PCB exposure and any type of cancer. All of this information leads inexorably to the conclusion that the TEQ approach, rather than providing a method for more accurate assessment of cancer risk posed by dioxin and so-called dioxin-like PCB congeners, instead would lead to human health risk assessments that unjustifiably exaggerate risk and lead to misallocation of societal resources.

Reasons that the TEQ Approach Does Not Accurately Predict the Human Carcinogenicity of PCBs

Recent studies have investigated why the TEQ approach, as well as animal bioassays, do not accurately predict the human carcinogenicity of PCBs. These studies have thrown considerable light on the differing sensitivities of rodents and humans to PCB exposure. At the March 21st meeting, Dr. Jay Silkworth will present to the Panel data from new studies that show that human liver cells respond differently to both PCB and TCDD than do rat cells. Human cells require higher doses to elicit a response, and the potency of the most potent "dioxin-like" PCB congener (PCB 126) relative to dioxin in human cells is much less than the currently assigned TEF value of 0.1, which is heavily based on data from rodent liver cells. In addition, Dr. Silkworth will present data, based on genomic studies, showing that dioxin elicits responses distinct from PCBs, contrary to the concept of toxic equivalency.

There is No Need to Apply the TEQ Approach to PCBs

It is clear that the TEQ approach is less accurate in predicting the human health risks of PCBs than EPA's traditional methods (RfDs and CSFs) based on empirical, PCB-specific data. We also believe that the justification that EPA has offered for application of the TEQ approach to PCBs is faulty. EPA has suggested that application of the TEQ approach to PCBs is justified as a means of ensuring that risks resulting from PCB congeners that preferentially bioaccumulate in fish tissue are not underestimated. The theory behind this suggestion is the idea that perhaps certain more toxic congeners might accumulate to a greater degree than other less toxic congeners found in the original mixtures, thus enriching the toxicity of the mixture beyond that of the original test material. Hence, according to this theory, the PCB CSF that is based on the original test mixtures might not be protective of potential risks posed by the altered mixture of congeners. This theory would have no validity, however, if the TEQs of environmental mixtures are no greater than the TEQ of the PCB test mixtures upon which EPA's PCB CSF of 2 $(mg/kg\text{-}day)^{-1}$ is based.

[2] Attachment 4 also includes a letter to the editor in reference to the Golden et al. paper and the authors' response to that letter.

This, in fact, is the situation for fish collected from a number of data sets that we have examined. This analysis is described and the results are presented in a journal article that is being submitted for publication. A copy will be provided to the panel upon acceptance by the journal and is summarized below.

EPA's CSFs for PCBs are based on bioassay data from studies of Aroclors 1254, 1242, 1260 and 1016 (EPA, 1996; Cogliano, 1998). According to Cogliano (1998), the TEQ concentration from coplanar PCBs in the Aroclor 1254 mixture used in the bioassays was 46.4 mg TEQ/kg PCB. Similarly, the PCB TEQ in the tested Aroclor 1242, 1260, and 1016 mixtures were lower at 8.1, 7.1, and 0.14 mg TEQ/kg PCB, respectively (Cogliano, 1998). The PCB CSF of 2 $(mg/kg\text{-}day)^{-1}$ is protective for the Aroclor with the greatest TEQ (i.e., Aroclor 1254) and, therefore, is protective for all PCB mixtures of equal or lesser TEQ. It follows, therefore, that the PCB CSF is protective of any exposure to an environmental PCB mixture that has a total TEQ of 46.4 mg TEQ/kg PCB or less.

For numerous fish and soil samples taken from fourteen water bodies noted for their PCB contamination,[3] total PCB concentrations were determined as the sum of all PCB congeners, using one-half the detection limit for non-detected congeners. The total TEQ for each sample was determined as the sum of the data for each coplanar congener (using one-half the detection limit for non-detected congeners) times its respective WHO TEF. Our analysis reveals that these samples from waterbodies noted for their PCB contamination have mean TEQ levels that are statistically significantly lower than 46.4 mgTEQ/kg PCB – the level of TEQ found in the test material upon which EPA's CSF for PCBs is based. Consequently, the use of the PCB CSF developed by EPA in 1996 to evaluate potential cancer risks is more than adequately protective of the carcinogenic potential of the PCB mixtures found in these fish tissues. There is no need to use the TEQ approach to ensure that risks are not underestimated.

* * *

Finally, we would like to call the Panel's attention to two additional issues.

- The Panel should be aware that the use of the TEQ approach for evaluating PCB cancer risks will lead to the conclusion that unacceptable risks exist when, in fact, the PCB congeners of concern are not even present in a sample. Under the TEQ approach, PCB 126 (3,3',4,4',5-pentachlorobiphenyl) is assigned a TEF of 0.1 relative to that of TCDD, designating it as the most potent of the so-called "dioxin-like" PCB congeners. Due to its elevated TEF, PCB-126 may contribute substantially to projected risk estimates, despite the fact that it is usually a minor constituent of the "dioxin-like" PCBs found in environmental residues. Because EPA risk assessment practice calls for the assumption that an undetected chemical is carried through the risk assessment as if it were present at a concentration equal to one-half of its analytical method detection limit, the probability is that undetectable residues of PCB-126 and other PCB congeners will result in elevated risk estimates, even though they are not detected in actual samples. In particular, it appears that

[3] The Delaware, Hudson, Housatonic, Fox, Kalamazoo, Sheboygan, Spokane, and Christiana Rivers; San Francisco Bay, Newark Bay, Green Bay, and Saginaw Bay; the South California Bight, the Great Lakes, Long Lake (WA) and Dick's Creek (OH).

the use of the TEQ approach will result in the conclusion that any fish sample, collected at any location, will pose human health cancer risks that exceed EPA's risk benchmark of 1x10-4, due to the assumed presence of PCB-126, regardless of whether this congener is actually present in the sample. A paper discussing this matter is being prepared for publication and will be submitted to the Panel if possible.

- The NTP (2003; 2004) bioassays also provide data to illustrate that EPA's (2003) draft Dioxin Reassessment has inappropriately commingled the use of a TEQ approach for PCBs based on administered dose with a CSF for dioxin based on a body burden dose metric. In EPA's draft Dioxin Reassessment, the proposed CSF for TCDD is based on a body burden dose metric. EPA then proceeds to relate the other dioxin-like congeners, including the "dioxin-like" PCBs, to TCDD using TEFs based on administered dose studies. However, as shown in Attachment 5,[4] the combined use of a CSF for dioxin based on body burden with a TEQ approach based on administered dose is incorrect and will serve to artificially magnify the estimated risks. In our analysis based on body burden, the relative cancer potencies of PCB 126, 4-PeCDF, and the TEQ mixture were much lower than predicted using the current WHO TEF scheme. This is not surprising because the TEF scheme was developed based on administered dose comparisons (Van den Berg et al., 1998), and the pharmacokinetics and distribution patterns for other TEQ-contributing compounds are substantially different from those of TCDD (DeVito et al., 1998). This analysis suggests that for carcinogenesis, the WHO TEF values substantially overpredict the cancer potency of 4-PeCDF and PCB 126 on a body burden basis. The current TEF values are based on intake-based assessments and should not be relied upon for assessments of cancer risk on a body burden basis.

In conclusion, it is of the utmost importance for the Panel to take the time needed to ensure that the risks of PCBs are accurately calculated. Otherwise, we are likely to see a significant misallocation of limited societal resources to address unfounded concerns regarding the safety of the food supply and perceived risks arising from contaminated sites. Indeed, we believe that prevention of misallocation of resources was one of the principal reasons that Congress asked for this NAS review.

We appreciate your consideration of these lines of evidence that show that the dioxin TEQ approach should not be used to assess the cancer risks of PCB mixtures, and that PCBs should not be included in the dioxin reassessment. We look forward to the opportunity to present our research and to address your questions.

Sincerely,

Kevin W. Holtzclaw
Manager, PCB Issues
The General Electric Company

[4] Attachment 5 has been extracted from a paper that has been submitted for publication.

Attachments

Attachment 1a: Keenan, R., J.Hamblen, J.Silkworth, M.Gray, P.Gwinn, S. Hamilton. 2003. An Empirical Evaluation of the Potency of Dioxin Toxic Equivalents (TEQs) in Several PCB Mixtures. Organohalogen Compounds, 65: 312-315. Proceedings Dioxin 2003 – the 23rd International Symposium on Halogenated Environmental Organic Pollutants and Persistent Organic Pollutants, Boston, Massachusetts, USA. August 24-29.

Attachment 1b: Papers presented to the EPA Science advisory Board in connection with their review of the draft Dioxin Reassessment in 2000-2001.

Attachment 2: Kimbrough, R., M.Doemland, M.LeVois. 1999. Mortality in Male and Female Capacitor Workers Exposed to Polychlorinated Biphenyls. J. Occup. Environ. Med. 41:161-171

Attachment 3: Kimbrough, R., M.Doemland, J.Mandel. 2003. A Mortality Update of Male and Female Capacitor Workers Exposed to Polychlorinated Biphenyls. J. Occup. Environ. Med. 45:271-282.

Attachment 4: Golden,R., J.Doull, W. Waddell, J.Mandel. 2003. Potential Human Cancer Risks from Exposure to PCBs: A Tale of Two Evaluations. Critical Reviews in Toxicology, 33(5):543–580.

Attachment 5: Body Burden Based on Cancer Potencies of Selected Dioxin-like Compounds Are Lower Than Predicted by the Toxic Equivalency (TEQ) Approach (summary of the results of a manuscript that is currently in submission (Gray et al., in submission).

Attachment 6: Compact Disk containing a detailed review of all of the human cancer studies.

References

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

Keenan, R.E., J.M. Hamblen, J.B. Silkworth, M.N. Gray, P.O. Gwinn, and S.B. Hamilton. 2003. An empirical evaluation of the potency of dioxin toxic equivalents (TEQs) in several PCB mixtures. *Organohalogen Compounds 65: 312-315. Proceedings Dioxin 2003 – the 23rd International Symposium on Halogenated Environmental Organic Pollutants and Persistent Organic Pollutants,* Boston, Massachusetts, USA. August 24-29.

National Toxicology Program (NTP). 2004a. *DRAFT NTP Technical Report on the Toxicology and Carcinogenesis Studies of 2,3,4,7,8-Pentachlorodibenzofuran (PeCDF) (CAS No. 57117-31-4) in Female Harlan Sprague-Dawley Rats (Gavage Study) (NTP TR 525),* National Toxicology Program.

National Toxicology Program (NTP). 2004b. *DRAFT NTP Technical Report on the Toxicology and Carcinogenesis Studies of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) (CAS No. 1746-01-6) in Female Harlan Sprague-Dawley Rats (Gavage Study) (NTP TR 521),* National Toxicology Program.

National Toxicology Program (NTP). 2004c. *DRAFT NTP Technical Report on the Toxicology and Carcinogenesis Studies of 3,3'4,4',5-Pentachlorobiphenyl (PCB 126) (CAS No. 57465-28-8) in Female Harlan Sprague-Dawley Rats (Gavage Study) (NTP TR 520),* National Toxicology Program.

National Toxicology Program (NTP). 2004d. *DRAFT NTP Technical Report on the Toxicology and Carcinogenesis Studies of a Mixture of 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) (CAS No. 1746.01-6), 2,3,4,7,8-Pentachlorodibenzofuran (PeCDF) (CAS No. 57117-31-4), and 3,3'4,4',5-Pentachlorobiphenyl (PCB 126) (CAS No. 57465-28-8) in Female Harlan Sprague-Dawley Rats (Gavage Studies) (NTP TR 526),* National Toxicology Program.

COMMENTS OF THE HOUSATONIC ENVIRONMENTAL ACTION LEAGUE, INC. (HEAL)

Comments of the Housatonic Environmental Action League, Inc. (HEAL)

Comments on the revised Human Health Risk Assessment
Judy Herkimer
Housatonic Environmental Action League, Inc.

Thank you for the additional opportunity to provide comments on the above document.

HEAL fully endorses and supports the comments by Peter deFur, PhD of Environmental Stewardship Concepts submitted on behalf of the Housatonic River Initiative and other stakeholder groups associated with this site.

RESPONSE HEAL-1:

See responses to TAG Comments below.

COMMENTS OF THE TECHNICAL ASSISTANCE GRANT RECIPIENT – HOUSATONIC RIVER INITIATIVE/ENVIRONMENTAL STEWARDSHIP CONCEPTS (TAG)

Comments of the Technical Assistance Grant Recipient – Housatonic River Initiative/Environmental Stewardship Concepts (TAG)

Comments on "Human Health Risk Assessment: GE/Housatonic River Site, Rest of River"
Prepared by Dr. Peter L. deFur
Environmental Stewardship Concepts
On Behalf of
The Housatonic River Initiative
April 5, 2005

Introduction

EPA requested comment on the changes to the Human Health Risk Assessment for the GE/Housatonic River Site, Rest of the River, originally released in 2003. EPA clearly asked for comments to address only those elements of the risk assessment that changed. The Housatonic River Initiative has contracted Dr. Peter deFur of Environmental Stewardship Concepts (ESC) to comment on the revised document. Notwithstanding EPA's request, ESC will comment on any areas in which this revised version is still deficient. EPA's revisions to the document, though minor, demonstrate that new information can alter the context of the entire report. The changes to the risk assessment are also sufficiently numerous and extensive that it is difficult to precisely determine all the changes.

General Comments

For the most part, the revisions represent an improvement of the risk assessment, and the additions make the document more complete. In particular, the EPA was wise to directly contact the Schaghticoke Tribe in Connecticut. The tribe represents a vital constituency in the cleanup of the Housatonic River, and should be included in all discussions regarding its remediation. Details of the contact with the Tribe are needed.

RESPONSE TAG-1:

The April 29, 2004 meeting between the Schaghticoke Tribal Nation and EPA was cited in the text as a personal communication. The memo to the file associated with this meeting is included as Attachment 1 to this Responsiveness Summary and is part of the public record.

The revision continues to omit any quantitative analysis of the non-cancer effects of dioxin and dioxin-like compounds, and states that there is no RfD for either dioxin or Ah active compounds. EPA can argue that they have abandoned the use of the RfD that was developed for dioxin, but to state that there is no RfD for dioxin is simply factually incorrect. The RfD for reproductive effects is old, but if EPA is going to use the old cancer potency factor and the older cancer classification, then they can certainly use the older RfD. The point is not that there is not an RfD, but that EPA chooses to not use the one that was determined previously. The reason that EPA does not use the RfD is that the population is already over-exposed to dioxin and the RfD would then mean that no additional exposures could be allowed.

RESPONSE TAG-2:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

The revised version still only gives token treatment to the Connecticut portion of the river. ESC has long argued that there is insufficient data to support many of EPA's claims regarding the risks posed by PCBs in the section of the river contained in Connecticut. The increased involvement of Connecticut regulatory agencies in recent months only emphasizes this deficiency. Previously, the EPA has been exemplary in its efforts to involve all parties affected by the contamination in the river. If the EPA continues to limit discussion regarding Connecticut, it could potentially alienate Connecticut stakeholders and leave a vital voice out of the process.

RESPONSE TAG-3:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

Comments on Specific Sections

Volume I

Section 4, Toxicity Assessment:

The Section 4 Toxicity Assessment has been substantially changed, with some additional information and much rearranging. The total effect is to make the specific revisions difficult to sort out. The addition of the section on the Dioxin Reassessment adds a great deal to the reassessment and is a positive addition.

The additional material on dioxin toxicity could and should include current literature. Several important papers (cited below) offer additional support for the conclusions that EPA reached in the 2000 version of the Dioxin Reassessment.

RESPONSE TAG-4:

The summary description of dioxin toxicity in the HHRA was based on review documents prepared by EPA and ATSDR, and provided references to these comprehensive reviews. It is beyond the scope of a site-specific risk assessment to include a full review of the recent literature on dioxin toxicity. As noted in the HHRA, the Dioxin Reassessment has been sent to the National Academy of Sciences for just such a review. Please refer to Response GE-10 for additional comments regarding recent papers on dioxin toxicity.

Page 4-3 lines 15-18, explains the chronic RfD and should include the notation that the dose refers to total dose from all sources, not just the source(s) under investigation. Many people fail to recognize or understand this point and the significance thereof.

RESPONSE TAG-5:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

Section 4, Table 4-1 seems to be the revised version of Table 2-1 from the previous version. There is no obvious reason why EPA removed the other chemicals of potential concern (COPC) from this table of toxicity values. The other compounds may not have been carried forward in the final analysis, but the toxicity values surely came into play in the screening and therefore still inform the reader of the technical input to the process.

The present Table 4-1 has dioxin information from the HEAST database. The problem is that the literature citation for this database is not complete and cannot be used to obtain the toxicity value in the table. The EPA should give a website or full document citation, preferably the former. In addition, the toxicity listing for dioxin paints a rather limited picture. The National Toxicology Program lists dioxin as a known human carcinogen (as does IARC, the agency of the World Health Organization that addresses carcinogens). The only reason EPA has not upgraded the carcinogenic classification of dioxin is political pressure; EPA has taken almost no policy or regulatory action on dioxin, despite the wealth of information from research scientists and the NTP. The human health risk assessment must at least acknowledge the fact that the classification is out of date, due for updating and that the NTP has classified dioxin as a carcinogen. The current draft has gone to the trouble of indicating that the Dioxin Reassessment has been sent to the National Academy of Sciences for review, which was at the request of industrial interests. The dioxin reassessment and all other aspects of the scientific assessment, regulation and policy regarding dioxin have been incredibly political and this assessment needs to at least provide the multiple perspectives on the issue.

RESPONSE TAG-6:

Table 4-1 provides toxicity values for contaminants that were considered Contaminants of Potential Concern (COPCs) after the initial screening process. Table 4-1 is analogous to Table 2-1 of the June 2003 draft of the HHRA in that it has values for all COPCs carried through the quantitative risk assessment. For soils, the screening was based on preliminary remediation goals (PRGs) developed by EPA Region 9 and listed in Table 2-5 of the HHRA Volume IIIA (Appendix B). For fish, the screening was based on screening risk-based concentrations (SRBCs) listed in Table 2-15 of HHRA Volume IV, Appendix C developed by EPA Region 3. The chemical-specific toxicity values are incorporated into the PRGs and SRBCs, along with assumptions regarding exposure.

The full citation for the HEAST document is: EPA (U.S. Environmental Protection Agency). 1997. Health Effects Assessment Summary Tables. Office of Research and Development, Washington, DC. EPA 540/R-97-036. It can be obtained from the NTIS website (www.ntis.gov) using the document number PB97-921199.

The human health assessment revision now adds language that the EPA has sent the dioxin reassessment to the National Academy of Sciences for additional review. It is not clear why this piece of information has been included, but for the sake of honest and completeness, but the report needs to add information that offers the scientific perspective and the public perspective. The scientific perspective is consistent with the findings over the past few decades- dioxin is a complete carcinogen and causes a range of non-cancer effects, including reproductive and developmental abnormalities. Furthermore, as recently reported in Environmental Health Perspectives, experimental results support the use of toxic equivalency factors for mixtures of dioxin-like compounds (Walker, N. J. et al. 2005, Environmental Health Perspectives 113: 43-48). Steenland et al. (Environmental Health Perspectives 112: 1265- 1268, 2004) reviewed the controversy over dioxin carcinogenicity and concluded that the IARC classification is consistent with and supported by the research that has been published since 1997. All of these results need to be reported along with the reference to the National Academy of Sciences text.

RESPONSE TAG-7:

The noncancer effects of TCDD and dioxin-like compounds, including effects on the immune system, reproductive system and developmental effects, are summarized in Section 4.5.2 of the HHRA.

The HHRA used toxic equivalency factors (TEFs) as one of two complementary approaches to evaluating the carcinogenicity of PCBs, and as a method for evaluating the combined effects of dioxin-like PCB congeners, dioxins, and furans. In Response GE-10 of this Responsiveness Summary, EPA addresses recent experimental data and analyses supporting the applicability of the potency-adjusted dose addition based on the TEFs published by WHO and used in the HHRA. The carcinogenicity of PCBs and some non-carcinogenic dioxin-like effects are also discussed in that response.

Section 8, Risks from Fish and Waterfowl Consumption:

Section 8.4.1, page 8-9, lines 14-16 refers to EPA finding no evidence of subsistence fishing on the Housatonic River. The citizens submitted information with their comments on the original risk assessment that Asian Americans are catching and consuming fish from the Connecticut portion of the Housatonic River. It is not certain if this consumption is true subsistence, but it is consumption of PCB contaminated fish and this pathway needs to be addressed.

RESPONSE TAG-8:

The question of subsistence fishing and SE Asian or Asian-American populations was also raised by the State of Connecticut with particular reference to the study conducted by Balcom et al. (1999). Please refer to Response CT-10.

Section 10 Integrated Risk Characterization and Major Findings:

This section is new in this version, with the possible exception of any material that was brought in from the Risk Summary of the earlier version of the assessment. This section combining exposure pathways is a positive addition to the risk assessment and EPA is to be commended for making this addition. The examples make the text easier to understand, but uniformed citizens are likely to have problems with this section.

RESPONSE TAG-9:

Section 10 of the HHRA provides guidance and tools for combining multiple exposure pathways, including several examples to illustrate the process. The section was placed near the end of the HHRA document, so that the preceding information regarding individual pathways would provide an uninformed reader with the necessary background in basic concepts of risk assessment to allow the integrated risk characterization to be understood. EPA expects that Section 10 will be viewed in that context, rather than as a stand-alone section.

Table 10-9 This table presents TEQ's from dioxins and furans compared with dioxin-like PCB's. This table demonstrates several points very well, and all need to be indicated in the text. The PCBs dominate the total amount of toxicity from substances that act via the Ah receptor; in addition, dioxins plus furans alone are enough to cause cancer and non-cancer effects and risks at unacceptable levels; finally, the non-cancer effects of dioxins plus furans are not quantified because EPA does not use the RfD that was published in 1984. Added together, the TEQ's for these Housatonic River exposures plus the existing TEQ exposures that the population faces at present from non-HR sources is enormous.

RESPONSE TAG-10:

The purpose of Table 10-9 was to provide information on whether substitution of Housatonic River fish and waterfowl for commercially obtained fish and waterfowl would increase the intake of TEQ for an HRA resident. The discussion in the text focused on this perspective (see page 10-11).

It is correct that, in fish and waterfowl from the Housatonic River, TEQ from dioxin-like PCBs is greater than TEQ from dioxin/furans. The cancer risk calculations for TEQ from dioxin-like PCBs, dioxins, and furans are presented separately in HHRA Volume IV, Appendix C, Tables 5-2 and 5-3 for ingestion of fish from Reaches 5 and 6 (the Primary Study Area [PSA]) and Reach 8 (Rising Pond). The text describing these tables gives the percentage contribution of each source of TEQ to cancer risk.

As noted in the HHRA, noncancer hazards associated with TEQ are not quantified because there is no current RfD, and the science underlying the dose-response assessment of TEQ is under review by the National Academy of Sciences. EPA acknowledged in the HHRA that the lack of quantitation of the potential hazard from noncancer effects results in a potential underestimate of these effects.

Section 10.1 is informative by presenting risks from multiple exposures of the sort that are likely to occur in a realistic situation. The examples are realistic and the explanations are helpful to see how to use the tables.

Section 10.2 considers the consequences of substituting grocery store food with Housatonic River watershed food products. The preceding sections of Volume I have already concluded that estimated cancer and non-cancer health risks from PCB's in the Housatonic River pose unacceptable risks. The entire purpose of this section is unclear, in no small part because the same point is already made in the preceding sections on contamination from specific pathways. I question the purpose of including this section and think the risk assessment may be better without it. The point of the risk assessment is to estimate the human health risks from the contamination on site, and determine if the risks are greater than the regulatory benchmarks, as described in EPA guidelines. The conclusion of section 10.2 is that food products from the Housatonic River watershed, especially aquatic animals, will increase the total health risks from PCBs. This conclusion was already made and is obvious from a comparison of the PCB and dioxin/furan levels in the food items under consideration. The greatest part of the section then is a more detailed demonstration of the point already made- food taken from the Housatonic Ricer is unsafe to eat.

RESPONSE TAG-11:

Section 10.2 is intended to add perspective to the risk calculations presented in other sections of the report, including the impact on food choices made by residents in the HRA.

Section 10.3 Breast Milk

This section is, for the most part, a helpful and useful discussion of the technical aspects of breast milk as an exposure pathway for infants. The text does present the current information and the unknowns, uncertainties and variability of the available data. The bottom line is that breast milk is an important pathway, and one that has been found to raise PCB levels to unacceptable levels. Therefore, the risks from Housatonic River PCB contamination are great for infants and mothers should not eat fish or any meat products from the watershed.

This section has the same problem as other section regarding non-cancer health effects from dioxins and furans – EPA will not use its own RfD or any derivation of this value. In fact, the discussion is weak and thin on the non-cancer health effects presented here.

RESPONSE TAG-12:

The evaluation of the breast milk pathway focused on exposure, specifically the potential for concentrations of PCBs, dioxins, and furans in the breast milk of HRA residents to be elevated above concentrations measured in the general population. The breast milk evaluation did not provide a quantitative evaluation of risk because, as stated in Volume I, Section 10.3, the benefits of breast feeding need to be balanced against the risk of adverse developmental, immunological, and neurological effects that may be associated with exposure to PCBs in breast milk. As pointed out in the HHRA (Volume I, p.10-13), several

studies show the beneficial effects of breast feeding despite the presence of PCBs and other contaminants associated with developmental neurological effects. In addition, there are no relevant toxicity values to serve as benchmarks for noncancer hazards over the short time period associated with breast milk exposures.

There is considerable information that has already been made available to the general public indicating that women and children should not eat fish and other biota from the Housatonic River because of PCB contamination. The Massachusetts Department of Public Health (MDPH) has issued the following consumption advisory for the Housatonic River from Dalton to Sheffield: "The general public should not consume any fish, frogs or turtles from this water body." An additional advisory was issued in 1999 by MDPH: "People should refrain from eating all mallards and wood ducks from the Housatonic River and its impoundments from Pittsfield south to Rising Pond in Great Barrington. In all areas other than the Housatonic River area, to reduce exposure to PCBs, wild waterfowl should be skinned and all fat removed before cooking. Stuffing should be discarded after cooking. Drippings should not be used for gravy. Waterfowl should be eaten in moderation (e.g., no more than two meals per month). Canada geese are not included in this advisory."

The Connecticut Department of Public Health advises pregnant women, those planning a pregnancy within a year and children under 6 not to eat fish from the Housatonic River above Derby Dam with the exception of one meal per month of panfish (yellow perch, sunfish) and one meal per month of bass, white perch and bullhead caught in Lakes Lillinonah, Zoar, Housatonic. The CTDPH advises others to limit their consumption of bass, white perch and bullheads, and not to eat eels, carp, trout and catfish. Section 10.3.2 presents more comparison with the general population. The problem with this type of comparison is that it is not directly relevant to excess risks from the site specific exposures. These exposures exceed any threshold, any benchmark, any consideration of "safe" for the people of Massachusetts and Connecticut. The reader has to wonder what is the point of this section and of comparing PCBs in Housatonic food with the general population. The results show two important factors- the risks from Housatonic River exposures are unacceptably high, and the risks to the general population also exceed most "safe" levels. The combination is alarming.

RESPONSE TAG-13:

The purpose of Section 10.3.2 was to describe the relevant studies of concentrations of PCBs and TEQ in human breast milk to provide a point of comparison for the predicted concentrations of PCBs in breast milk due to consumption of foods in the HRA. A comparison of exposure due to the site with a background or general population exposure was included to provide perspective on the impact of site-related exposure and thus to contribute to characterization of the risk.

Appendix B, Direct Contact:

4.2.2: The document needs to explain that work on Reaches 1-4 has been completed and therefore not included in the assessment.

RESPONSE TAG-14:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

4.2.3.4: The change from residential to commercial in property along Rt 102 does not necessarily mean lower risks. Construction activities could cause exposure to dust, and some commercial uses may actually fall under the recreational use category, and should not be overlooked.

RESPONSE TAG-15:

The purpose of Section 4.2.3 was to generally describe the reasonably foreseeable uses of the floodplain. The information in the section was obtained, for the most part, through interviews with the planning officials for each of the towns along the Housatonic River and review of planning documents. The comment specifically focuses on the residential property along Route 102 south of the Massachusetts Turnpike, which is within the Town of Lee. All of the area, with the exception of one property, was eliminated from further consideration in the Phase 1 Screening Evaluation (see Sections 4.2.2.23 through 4.2.2.25 of Appendix A). The property that was retained for the Phase 2 Direct Contact Risk Assessment was evaluated as EA 79 (see Section 5.5.2.13 of Appendix B). This area was evaluated for recreational exposure using the general recreation scenario. The estimated RME cancer risk is 3E-06, which is at the low end of the EPA risk range. The noncancer hazard index is 0.12. Any future change in land use would result in less intense exposure and lower risks. Based on this, EPA believes that the areas along Route 102 were evaluated appropriately and no additional investigation is required.

4.2.3.6, last line: The assertion that the changes in land use would not result in unacceptable risks needs to be later in the text and sufficient evidence provided to support it.

RESPONSE TAG-16:

This comment refers to the future use of the floodplain in Great Barrington. As described in Section 5 of Appendix A (Phase 1 Direct Contact Screening Risk Assessment), Reach 9, which includes the Town of Great Barrington, was eliminated from further consideration based on concentrations of tPCBs below the conservative screening risk-based concentrations (SRBCs). Only three of 205 floodplain soil samples collected from Reach 9 were greater than the most health-protective screening concentration of 2 mg/kg. This screening level was developed using exposure parameters for residential exposure, which is

considered to be the most conservative land use. Thus, based on the screening level used and the fact that the vast majority of collected samples (>98%) were less than the screening level, EPA believes that it has been adequately demonstrated that unacceptable risks would not occur.

4.2.4: The list of potentially exposed populations should include construction workers operating in the floodplain. Construction is possible according to new information regarding potential future uses. Section 5.5.1.6 indicates that the development of housing is possible in EAs 6, 18, 21, 34, and 86. Construction is also listed as an activity in which adults may be dermally exposed to contaminated soils in section 6.5.1.9.6 Minor construction (ie the construction of a house or other small building) could still pose exposure risks. Risks to construction workers would be similar to those of utility workers and could be easily calculated

RESPONSE TAG-17:

As described in HHRA Section 4.3.6, the construction worker scenario was not considered a complete exposure pathway because flood events and the Massachusetts Wetland Protection Act do not allow for major construction activities, such as residential building and road construction, in the floodplain.

4.3.5: Again, needs to explain the exclusion of reaches 1-4. It is understood why these reaches were not included but should be made clear in the text in the interests of transparency.

RESPONSE TAG-18:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

4.3.6: Construction work should be included in the exposure scenarios for the reasons outlined in the comments on Section 4.2.4.

RESPONSE TAG-19:

As described in Section 4.3.6, the construction worker scenario was not considered a complete exposure pathway because flood events and the Massachusetts Wetland Protection Act do not allow for major construction activities, such as residential building and road construction, in the floodplain.

4.5: Exposure parameters do not include air exposure pathways, specifically the inhalation of contaminated dust. This is a particularly significant pathway, particularly for the recreational and utility worker scenarios where significant amounts of dust may be disturbed during activities. For some activities such as bike riding or the use of other recreational vehicles such as ATVs during dry weather, this may be a more significant pathway than either dermal exposure or ingestion.

RESPONSE TAG-20:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

5.1: Dioxins and Furans should be included wherever there are data. The risks from dioxins are additive to those of PCBs and act along the same or similar mechanisms. Tiny amounts of dioxins can cause significant cancer risks and non-cancer health effects, and should be considered in this section rather than the uncertainties.

RESPONSE TAG-21:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

5.5: The division of the site into Exposure Areas is applauded, and the increased level of detail regarding those Exposure Areas is appreciated.

5.5.1.36: The utility worker scenario should be included in the evaluation of EA 36. The area is owned by utility companies and contains large numbers of transformers and other equipment. It should be expected that maintenance will be required in this area, potentially exposing them to contaminated soils. The EPA should calculate risks to these workers and included them in the Risk Assessment.

RESPONSE TAG-22:

EPA acknowledges that the transformers and other equipment at Exposure Area 36 will need to be periodically maintained by utility workers. However, this equipment is not located within the 1-ppm tPCB isopleth (the approximate extent of the 10-year floodplain); thus, contact with contaminated soil is not expected. There are two utility easements with overhead wires extending from this property. It is possible that utility workers could work on these easements. These easements were evaluated as Exposure Areas 65 and 66. The risks associated with soil exposure while working along the easements are presented in Sections 5.5.1.65 and 5.5.1.66 of Volume IIIA.

6.1.1: This section should also include construction workers to exposed populations for the reasons given above in comments for Section 4.2.4.

RESPONSE TAG-23:

As described in Section 4.3.6, the construction worker scenario was not considered a complete exposure pathway because flood events and the

Massachusetts Wetland Protection Act do not allow for major construction activities, such as residential building and road construction, in the floodplain.

6.5.1: Models should include total body burden of PCBs. PCBs stay in the body for long periods of time, potentially magnifying subsequent exposures.

RESPONSE TAG-24:

The model used in the probabilistic risk characterization is consistent with the model used for all point estimate risk characterizations and the fish and waterfowl probabilistic risk characterization in the June 2003 HHRA. Therefore, the comment does not address new information in the revised HHRA. The model also is consistent with EPA risk assessment guidance (EPA, 1989).

Reference:

EPA (U.S. Environmental Protection Agency). 1989. *Risk Assessment Guidance for Superfund, Volume 1 – Human Health Evaluation Manual, Part A, Interim Final*. Office of Emergency and Remedial Response, Washington, D.C. EPA/540/1-89/002. Publication 9285.7-01A. December 1989. http://www.epa.gov/superfund/programs/risk/ragsa/.

7.2.3.2.2: By not evaluating the non-cancer health effects of dioxins and similar compounds, the Risk Assessment is greatly underestimating risk. There is enough data in both the literature and documents published by the EPA for investigators to evaluate these effects. Though an RfD is not used for dioxins, the reason (which is discussed in this section) is that if one were calculated it would be well below background doses. Therefore, it can be assumed that any additional dose of dioxins would have a detrimental effect and increase the risks to the immune, endocrine, and developmental systems. Considering the sensitivity that these systems have been found to have to dioxins (Mably et al, 1992), it would be difficult to overestimate the risks posed by these compounds.

RESPONSE TAG-25:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

Appendix C, Consumption of Fish and Waterfowl

1.2: This section should include a description of Reaches 1-4 and provide an explanation as to why they were not included in this risk assessment.

RESPONSE TAG-26:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments.

As stated in the introduction to this Responsiveness Summary, EPA solicited public comment only on new information and is responding only to comments that pertain to the new information.

4.2: The addition of breast milk and *in utero* exposure as a potential exposure pathway is welcome and better quantifies risks to particularly vulnerable segments of the population.

4.3.4.1: EPA needs to give more information regarding the meetings between representatives of the Schaghticoke Tribe and EPA. Any formal accounts of the meeting including memos or reports should be cited in the text and included as an attachment. This will insure that the Tribe's views and practices will be heard and part of the public record that is available for review.

RESPONSE TAG-27:

Please refer to Response TAG-1.

6.0: ESC strongly objects to the removal of CSF TEQ risk calculations from the document. This is valuable data that should be included, and at the least the EPA should have an explanation regarding their removal.

RESPONSE TAG-28:

Evaluation of cancer risk based on exposure to TEQ remains in the revised HHRA. For the Agricultural Consumption risk assessment and the Direct Contact risk assessment, these calculations are now provided in the uncertainty section based on comments received during the Peer Review.

Figure 6-92: This figure is not displayed.

RESPONSE TAG-29:

EPA reviewed copies of the February 2005 HHRA in its possession and was able to locate Figure 6-92 in each copy. The figure appears on page 6-87.

7.2.3.2.2: By not evaluating the non-cancer health effects of dioxins and similar compounds, the Risk Assessment is greatly underestimating risk. There are enough data in both the literature and documents published by the EPA for investigators to evaluate these effects. Though an RfD is not used for dioxins, the reason (which is discussed in this section) is that if one were calculated it would be well below background doses. Therefore, it can be assumed that any additional dose of dioxins would have a detrimental effect and increase the risks to the immune, endocrine, and developmental systems. Considering the sensitivity that these systems have been found to have to dioxins (Mably et al, Toxicol Appl Pharmacol 114:108-117. 2930), it would be difficult to overestimate the risks posed by these compounds.

RESPONSE TAG-30:

This comment does not address new information added to the February 2005 revised Human Health Risk Assessment in response to Peer Review comments. As stated in the introduction to this Responsiveness Summary, EPA solicited

public comment only on new information and is responding only to comments that pertain to the new information.

ATTACHMENT 1

MINUTES FROM MEETING WITH SCHAGHTICOKE TRIBAL NATION

May 11, 2004

From: Jim DiLorenzo
Margaret McDonough
Angela Bonarrigo

To: GE HHRA Site File

Subject: GE Site – Minutes from Meeting With Schaghticoke Tribal Nation

Background

EPA is in the process of completing a Human Health Risk Assessment (HHRA) for the Rest of River associated with the GE Housatonic River Site. The June 2003 HHRA report was peer reviewed by a panel of seven scientists, which lead EPA to issue a responsiveness summary for the HHRA in March 2004.

One of the issues raised by three reviewers was with regard to concern over possible subsistence angling by members of the Schaghticoke Tribal Nation who own a 400-acre reservation along the Housatonic River in Kent, CT. Prior attempts to contact the Schaghticoke Tribal Nation members were unsuccessful in determining whether subsistence angling was consistent with actual or desired tribal practices. As required by general response number 3.C of the HHRA responsiveness summary, EPA undertook additional efforts to contact members of the Schaghticoke Tribe.

The Schaghticoke Tribal obtained Federal acknowledgment in January 2004. This allowed members of the EPA GE Case Team to work with members of EPA's Indian Program. Through this joint effort, a meeting was held at the Schaghticoke Tribal Nation's office located in Derby, CT, on April 29, 2004. This memorandum summarizes the meeting and identifies issues of potential relevance with regard to the GE Housatonic River Site.

Attendees

NAME	AFFILIATION	CONTACT
Margaret McDonough	EPA risk assessor	617 918-1276 mcdonough.margaret@epa.gov
Angela Bonarrigo	EPA public affairs	617 918-1034 bonarrigo.angela@epa.gov
Jim DiLorenzo	EPA project manager	617 918-1247 dilorenzo.jim@epa.gov
Jim Sappier	EPA Indian program	617 918-1672
Val Bataille	EPA Indian program	617 918-1674
Joe C. Velky, Jr.	Tribal Council Treasurer and Tribal member	
Charles E. Kilson	Tribal member	
Beth A. Kelly	Tribal descendant	

NAME	AFFILIATION	CONTACT
Frederick N. Parmalee	Tribal member and Housing Authority Chair	203 268-3223 fnpcpa@peoplepc.com
Linda M. Gray	Tribal Clerk/Genealogist	203 736-0782 lindag@schaghticoke.com
Kateri J. Manning	Tribal member	

Meeting Overview

Jim Sappier and Val Bataille congratulated the tribal members on the recent Federal acknowledgement of the Schaghticoke Tribal Nation. There are currently nine federally-recognized tribes in EPA Region 1. They provided the tribal members with information about EPA's Indian Affairs Program and then each explained various assistance and information resources available from EPA Region 1. It was explained that although many resources were dependant upon successful completion of the 180 day appeal process for federal recognition to be final, several resources were immediately available in the form of specific grants and assistance. Val welcome the tribal members to participate on monthly calls coordinated by EPA with the other Federally acknowledged tribes and offered suggested contacts to help complete the recognition process.

It was explained that EPA will assign a coordinator to the Schaghticoke Tribal Nation upon successful completion of the appeals process but that in the interim, members could contact Val or Jim S. with any questions.

The meeting then turned focus to the GE Housatonic River cleanup.

GE Rest of River Overview

Jim DiLorenzo and Margaret McDonough provided an overview of the cleanup and rest of river investigation with emphasis on the human health risk assessment. It was generally explained that the focus of the problem is the PCB-contaminated portion of the river within 10 miles of the former GE facility down to and including Woods Pond. This did not mean that contamination of the river in Connecticut is negligible, but that future investigation and remediation efforts, if any, are unclear. Future monitoring is likely. The importance of determining actual and desired tribal practices with regard to activities, which may occur within the Housatonic River floodplain, were discussed. We clarified that our need is to understand desired future fish and waterfowl consumption and preparation practices, as they may occur in the future in the absence of contamination.

Margaret provided each tribal member in attendance with a copy of a computer disk entitled, "The Human Health Risk Assessment."

Schaghticoke Reservation – Kent, CT

The tribal members explained that the current reservation spans about 400 acres. Efforts are underway which may expand the reservation by more than an additional 2,000 acres. There is

currently a moratorium on building at the reservation that is expected to be lifted in the future. The tribe has a housing authority that plan to construct housing, possibly for elder members, in the future.

Members expressed the following specific concerns with regard to the reservation and PCBs in the river;

1. Sediment contamination – Beth Kelly explained that the tribe had collected 6 sediment core samples from the bank area of the Housatonic for PCB analysis by EPA methods. Results indicate elevated concentrations that are inconsistent with EPA's generally low PCB sediment profile in CT. Beth was not ready to share this data and was looking for resources to first repeat the sampling event. *Jim D. emphasized the importance of sharing this data, with any supporting quality assurance documentation, as EPA currently has no plans for futher sediment sampling in CT. Evidence of elevated levels in the CT floodplain is critical to ensure the final cleanup plans are protective. Angela recommended that Beth contact Tim Gray who manages the existing Technical Assistance Grant and may be able to provide resources for additional sampling.*

2. Warning Signs – Members expressed concern that signs are not posted along the reservation stretch of the river. Signs that are posted at other locations are too confusing for most people. They requested signs for the reservation which contain clear warning for species of fish which are desirable to Schaghticoke members (see below.) *Jim D. stated that EPA is aware of this concern. Angela and Margaret agreed to pursue this issue.*

3. Well Water – Members were concerned that well water on the reservation is contaminated with PCBs. *Jim D. explained that PCB contamination is limited to the river proper and that contamination of the aquifer is not possible since groundwater generally flows to the river.*

4. Direct Contact – Members were concerned whether risks will exist if future infrastructure, homes and/or docks, are added to the reservation. *Jim D. emphasized that the only sediments of potential concern are within the floodplain. Current EPA data suggests direct contact risks with sediment do not exist in CT. Risks could exist if elevated PCBs are present in sediment in areas of possible contact (ie., future dock).*

5. Swimming – A member expressed concern regarding swimming in the river. *Margaret explained that potential risks are from ingestion only. Swimming is safe in the CT portion of the river.*

Tribal Consumption Practices

EPA asked the members about the species of fish desired and consumed from the river. Chuck Kilson acknowledged that members currently catch and release only because of the consumptive warnings. In the absence of such warnings, Chuck listed the following species as desirable; American eel, American bullhead, carp, yellow perch, crayfish and to a lesser extent, chain pickerel. These are in addition to the bass, trout, bullhead and perch already studied in the

HHRA. The preferred method for preparation is pan frying, although a long-desired tribal practice is to prepare the fish by removing the head, wrapping the fish in mud, then foil, and slow-cooking. Chuck also mentioned that helgamites are used as bait for fish such as bass and that there exists some caverns with considerable sediment deposition near the confluence with the Ten-Mile River which is a popular fishing spot.

EPA then asked the members about waterfowl and other species outside the river but which may be located within the floodplain and desired by members. The members stressed that the snapping turtle was widely consumed by the tribe, particularly as a soup. Duck and geese are generally not preferred. Members do currently hunt for deer each November and generally bag 5 or 6 deer between the members. Turkey, grouse, muskrat, frog, rattlesnake, woodcock and rabbit are/were also hunted and consumed. Rattlesnakes were also used for medicinal purposes.

Schaghticoke Survey

The members were very cooperative and recommended that more accurate information could be gained by contacting several tribal members, particularly the remaining Tribal Elders. The members, lead by Linda and Chuck, agreed to conduct a survey to help refine desired tribal practices. *Val offered a standard survey used by other tribes as a good starting point. Jim D. mentioned it is unlikely that results will be gathered in time for use in the final HHRA report but will be included in the Site file.*

HHRA Relevance/Recommendations

1. Subsistence Angler – Members currently use the property for recreational fishing. The tribe intends to expand residential use of the reservation property. EPA will evaluate the subsistence angler pathway in the uncertainties section of the revised HHRA.

2. Future Use – It should be noted in the HHRA that the tribe intends to develop the reservation property for residential purposes.

3. Fish/Other Consumption – EPA will include a qualitative discussion of potential risks to/from species not currently evaluated in the HHRA (i.e., eel and turtle).

4. Sediment Sampling – No additional sampling is recommended for the HHRA, however, EPA will consider future sediment sampling in floodplain portions of the reservation and cavern areas of the river, pending review of any data provided by the tribe.

5. Schaghticoke Survey – EPA will include any survey results in the HHRA **if received by mid-June 2004**. Survey results will become part of the Site file for the rest of river investigation.